6- Degenerative joint disorders

Objectives:

- Not given.

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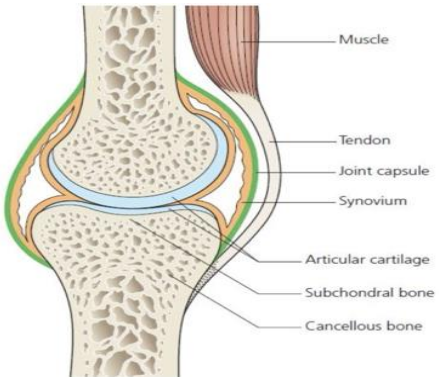
References: 435 team, Doctors' notes, 436 slides, Apley

Introduction

★ Articular cartilage:



- It is avascular and aneural **hyaline cartilage.**
- Viscoelastic material with variable load - bearing properties.
- Decreases joint friction.

★ Cartilage composition:

1- Water (60% to 80% wet weight) most important part

- Pumped in and out of cartilage **depending on load**.
- Contributes to lubrication and Nutrition.

2- Collagen (10% to 20% wet weight)

- Secreted by chondrocytes.
- Mostly type 2 collagen (90%).
- Confers **tensile strength** to cartilage - قوة الشد

3- Proteoglycans (10% to 15% wet weight)

- Secreted by chondrocytes.
- Composed of GAG (glycosaminoglycan) (aggrecan, chondroitin, and keratin sulfate).
- Negatively charged proteins holds water within matrix.
- Provides compressive strength.

4- Chondrocytes (5% wet weight)

- Only cell type in cartilage.

⇒ have little capacity for cell division in vivo[1].

So, when there is a direct damage to the articular surface, cartilage is poorly repaired or repaired only with (poor cartilage) **fibrocartilage** which has inferior biomechanical properties than hyaline cartilage.

⇒ If the collagen network is disrupted, initially the matrix becomes waterlogged and soft.

⇒ Followed by loss of proteoglycans, cellular damage and splitting “fibrillation” of articular cartilage.

⇒ Damaged chondrocytes begin to release matrix-degrading enzymes inside the joint cause further damage.

Basically, when the cartilage is damaged it is replaced with fibrocartilage which is weaker than Hyaline cartilage.

Capsule and ligaments:

- Fibrous structure with tough condensations on its surface (ligaments).
- Together with the overlying muscles, they help to provide stability. which is imp

[1] Studies are those in which the effects of various biological entities are tested on whole, living organisms.

Synovium:

- Thin membrane, richly supplied with blood vessels, lymphatics and nerves.
- It provides a non-adherent covering for the articular surface and **produces synovial fluid.**
- **It is the target of autoimmune reactions in joint infections** (septic arthritis) **and autoimmune disorders such as rheumatoid arthritis** and SLE.

Synovial fluid:

- Have several functions: Nourishes the avascular articular cartilage, plays an important part in reducing friction during movement by type of fluid itself, it also has slight adhesive properties which help in maintaining joint stability.
- The volume remains fairly constant, regardless of movement. Unless the joint got injured or infected it increases resulting in joint effusion = fluid accumulates and we feel it clinically as effusions.
- Role of synovial fluid: lubrication, nutrition, stability.

Degenerative bone disorder

Osteoarthritis video osteoarthritis high yield notes osmosis Toronto noted OA mayo clinic OA

When you get the words Osteoarthritis/arthritis/osteoarthrosis → it meant to be degenerative type unless they specified (infective, inflammatory and so on)

1- Primary (idiopathic) osteoarthritis (OA):

Chronic disorder in which there is:

1. **Progressive softening and disintegration of articular cartilage.**
2. **New growth of cartilage and bone at the joint margins (osteophytes).**
3. **Subchondral bone sclerosis and cyst formation.**
4. **Mild synovitis and capsular fibrosis.**

- Asymmetrically distributed, often localized to only **one** part of a joint for ex: in the knee the medial part is affected usually. هنا الفرق إنه يكون بجزء واحد بس مو كل المفصل متأثر، لأنه غالبًا الويت بيرنق أغلبه بالميديال ساید
- Often associated with abnormal loading.
- **Un**accompanied by any systemic illness.
- **Not primarily an inflammatory disorder** (although there are sometimes local signs of inflammation).
- **Not a purely degenerative** (dynamic phenomenon: it shows features of both destruction and repair). It is a misnomer because they have signs of degeneration and repair / signs of repair like osteophytes.

Keep in mind once degeneration starts it will not stop, we try to slow it down, change the patient's lifestyle to improve quality of life. We mainly depend on quality of life when treating a patient so we depend on it not the x-ray. REMEMBER it about the patients quality of life we don't treat x-rays we treat patients.

2- Secondary OA:

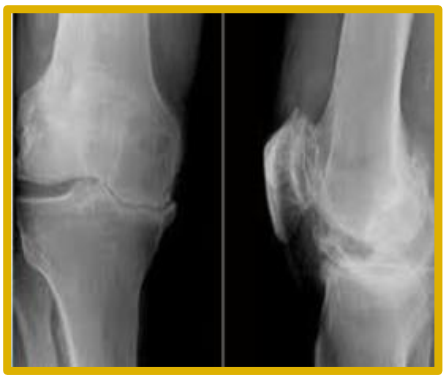
In secondary usually the **whole** joint is affected unlike primary osteoarthritis.

Trauma	Osteochondral, malunion, sport injury.
Infection	
Metabolic	Crystaline deposition disease (gout, CPPD[2]), Paget's disease.
Inflammatory	RA, SLE, Reiter's syndrome[3].
Congenital/developmental	Hip dysplasia, multiple epiphyseal dysplasia.
Necrosis	Perthe's disease[4], osteonecrosis, steroids.
Neuropathic	DM, Tabes dorsalis.
Hematologic	Sickle cell anemia, hemophilia.
Endocrine	DM, Acromegaly.

The classic symptoms of osteoarthritis: Pain, Limitation of movement, swelling.

★ Etiology: OA has no single cause; rather, it is due to a variable combination of several risk factors.

- ↑ Mechanical stress in some part of the articular cartilage.
- OA results from a disparity between the mechanical stress applied to articular cartilage and the ability of the cartilage to withstand that stress. This could be due to one or a combination of **two processes:**
  - **Weakening of the articular cartilage** (due to a genetic defect or enzyme activities).
  - **Increased mechanical stress** in some part of the articular cartilage. This can be caused by overuse or joint instability.
- Varus deformity of the knee. **Medial** side is the most affected.
  For example in the knee, the normal knee is in valgus with the inflammation and degenerative OA it becomes varus



Remember we don't see cartilage in x-ray we see joint space. Usually the patient should be in a weight bearing position (standing) so we can properly interpret the image. (Centigram x-ray)

Other causes (risk factors):

OA is more of a process than a disease.

1. Increases in frequency with age.
2. Obesity (hips and knees take 3-4 times body weight with each step).
3. **Family history** IMP factor

★ Prevalence:

- Osteoarthritis is the **commonest** of all joint diseases.
- Much more common in some joints (knee, hip, spine and the fingers) than in others (the elbow, wrist and ankle).
- **Females** > males.
- Common in our community especially **knees** & presents earlier than in West at about 40's.
- About 90% of those over 40 have asymptomatic degeneration of weight bearing joints.

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[2] Calcium pyrorophosphate dehydrate crystal deposition disease.
[3] Is a form of inflammatory arthritis that develops in response to an infection in another part of the body (cross-reactivity).
[4] Is a rare childhood condition that affects the hip. It occurs when the blood supply to the rounded head of the femur (thighbone) is temporarily disrupted. Without an adequate blood supply, the bone cells die, a process called avascular necrosis.

- Commonest joints are knee, hip, Cervical spine & Lumbar Spine, 1st Carpometacarpal, 1st Metatarsophalangeal and Interphalangeal joints.

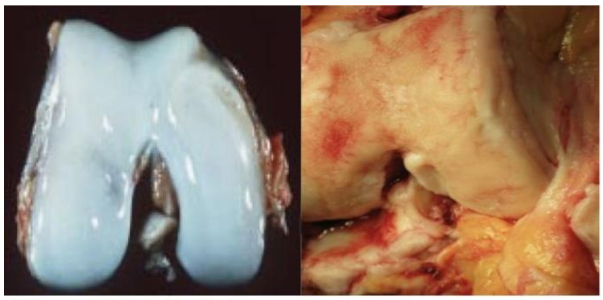
The worst advice you give the patient is telling him/her not to use the affected joint, as a result the patient will develop stiffness, weight gain, muscle loss and the symptoms will be worse!

★ Pathology:

The 5 cardinal (major) features are:

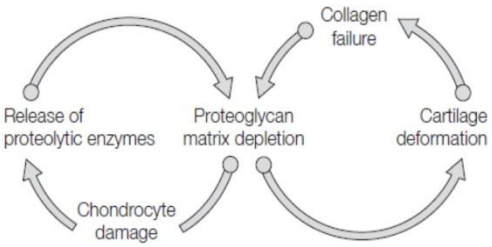
1- Progressive cartilage destruction seen in all X-rays

- **Increase** in water content → Swelling and softening of the cartilage.
- Then later on there will be:
  1. Depletion of proteoglycans.
  2. Chondrocytes damage and synovitis → proteolytic enzymes → collagen disruption splitting of articular cartilage.
  3. Fibrillation on weight bearing surfaces and then complete destruction.



This is normal articular cartilage it is shiny hyaline structure)

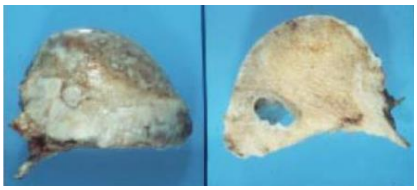
Cracks in articular cartilage

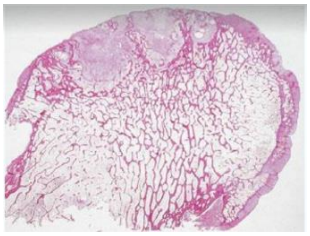


2- Subarticular cyst formation cyst doesn't usually appear in x-rays of knees but in hips is common to see it

Subarticular cyst could arise from: 1 - Local areas of osteonecrosis the bone get necrosed and die **Or**

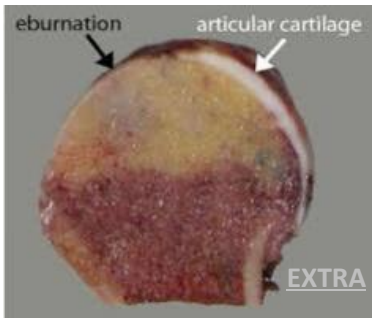
2- From the forceful pumping of synovial fluid through cracks in the subchondral bone plate. From the fibrillation (crack خطوط in the cartilage) → with pressure there will be pumping of fluid which will accumulate under the sub-articular cartilage to form the cyst.

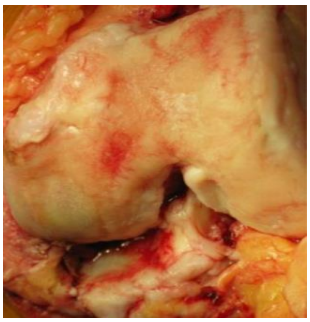


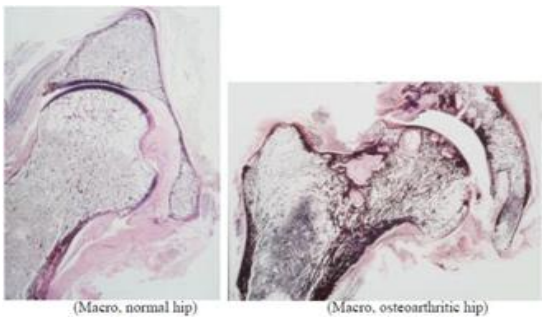


3- Sclerosis of the surrounding bone seen in all X-rays

Bone becomes **exposed** → may be **polished or burnished** to ivory-like smoothness (eburnation). ivory عاج صلب جداً/ in x ray we see it like whitening of the bone.



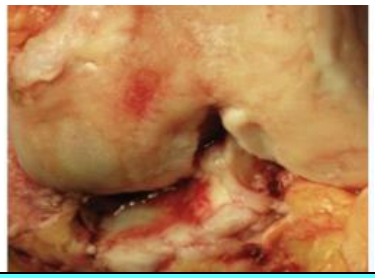




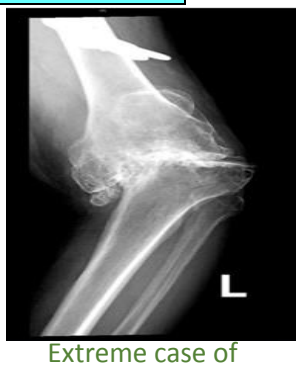
(Macro, normal hip) (Macro, osteoarthritic hip)

4- Osteophyte formation seen in all X-rays

- Proliferation and remodeling of the adjacent cartilage at the edges. followed by
- Enchondral ossification of that cartilage. The joint try to increase the surface area to distribute the stress so it forms a cartilage at the periphery of the joint and this cartilage underwent enchondral ossification and becomes a **bone**.



5- Capsular fibrosis



Extreme case of neglected OA.
- Notice: ligament (LCL) overstretched and (MCL) tighten.
- Varus

- Marked vascularity and venous congestion of the subchondral bone. → Cause of pain (in medial side), Consider it in the management plan.
- The capsule and synovium are often thickened but cellular activity is slight.
- Progressive bone erosion → **BONE COLLAPSE**.
- Fragmented osteophyte → **LOOSE BODIES**.
- Loss of height and ligamentous laxity → **MALALIGNMENT**

Bold are Signs of severe or recurrent OA, if patient presents with night pain or pain at rest (End stage disease)

★ Key pathological features of OA (**EXTRA** FYI from apley):

Pathology	Radiographic correlates
Focal areas of loss of articular cartilage	Joint space narrowing (if loss is extensive)
Bone growth at the joint margins	Osteophytes
Sclerosis of underlying bone	Sclerosis of subchondral bone
Cyst formation in underlying bone	Bone cyst
Loss of bone	Bone attrition
Varying degree of synovial inflammation	Effusions may be apparent
Fibrosis and thickening of the joint capsule	Not visible on radiograph

★ Clinical features:

It has an **intermittent** course, with period of remission sometimes lasting for months, affecting one or two of the weight bearing joints (hip or knee) in our community patients having degenerative **knee** will have degenerative **lumbosacral & cervical** spine, So in the surgery for knees check spine and vice versa.

Pain of OA started in one of two ways either **suddenly** after an event (I did something I'm not used to (Marathon)) or **progressively** (day after day). Pain usually with activity relieved by rest. So? **Mechanical pain!**

Pain not always there + not always the same. يعني بشكل عام الحال متدهور المريض يخفف ألمه بطريقتين يا إما 1- بالمسكنات "طبعًا احتياجه لها بيتغير على مدى السنوات بالبداية مرة كل شهرين مثلًا بعدين صار كل أسبوع!" 2- بتفادي الحركات اللي تسبّب له الألم. طيب، وش الأفضل بالنسبة لنا؟ وش بتنصح مريضك؟ يخفّف ألمه بالمسكنات طبعًا ويحاول يحافظ على حركته لأن لو استسلمنا للألم بنخسر نطاق الحركة الطبيعية للمفصل وبنخسر العضلات بعد. وضعف العضلة نفسه أصلًا يزيد أعراض الخشونة.

Symptoms:

- **Pain:**

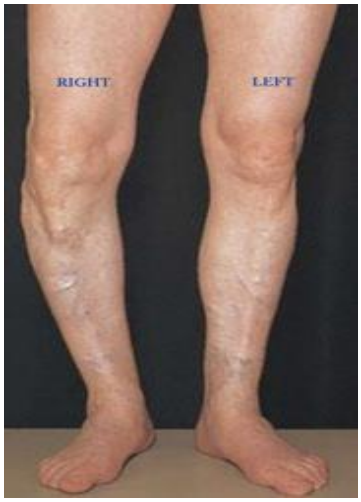
Localized or rarely referred to distant site (e.g: pain in the knee from hip osteoarthritis?)

⇒ Insidious in onset, aggravated by exertion and relieved by rest and in advance stage there will be night pain or pain at rest.

Causes of the pain:

1. Bone pressure due to vascular congestion and intraosseous hypertension **(most important)** osteotomy done in young patient is based on this theory in order to relieve the vascular congestion and intraosseous hypertension.
2. Mild synovial inflammation.
3. Capsular fibrosis with pain on stretching the shrunken tissue.
4. Muscular fatigue

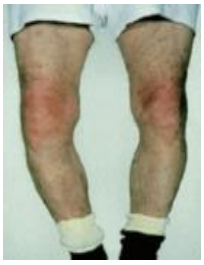
- **Stiffness:** initially after periods of inactivity, but later on it will be constant and progressive
- **Loss of function.**

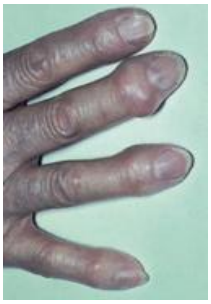


BOTH knees are affected by varus, but right knee is severely affected and left Is moderately affected.
Normal valgus of knees (5-7 degree), 7 in female because they have hip and ligament laxity.

Signs:

- **Swelling**: Intermittent (→effusions), Continuous (→large osteophytes).
- **Deformity**; mal-alignment.
- **Tenderness.**
- **Limited** range of **movement.**
- **Crepitus**[5].
- **Instability** because of Loss of cartilage and bone, asymmetrical capsular contracture and/or muscle weakness.





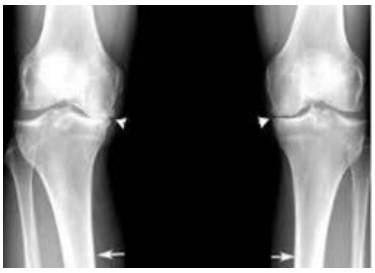
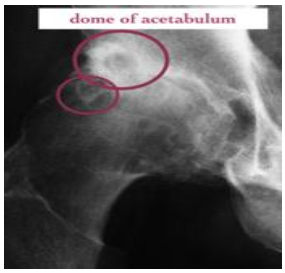
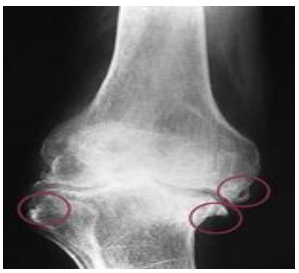
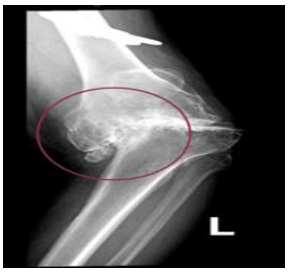
Heberden's nodes

★ Imaging: OA x-ray finding Mnemonic : **LOSS** (Subchondral cyst)

- **A**symmetrical **L**oss of cartilage (narrowing of the 'joint space').
- **O**steophytes **at the margins of the joint.**
- **S**ubchondral bone **S**clerosis.
- **Cysts close to the articular surface** in knee most likely → medial side, hip → dome (superior-lateral)

Late features: Malalignment, Joint subluxation, Bone loss, Loose bodies.

[5] Rubbing of bone against bone, you can both hear and feel.

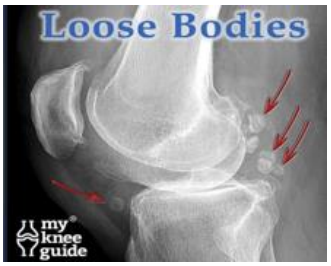
Subchondral bone sclerosis	Subchondral cyst	osteophyte formation	Bone loss
 White sclerotic lesion in the medial side of knee. Notice the narrowing of joint space.	 Cysts at the dome of acetabulum (superior-lateral part)		

Look for signs of other disorder: **Symmetric** narrowing in **inflammatory OA** (such as RA).

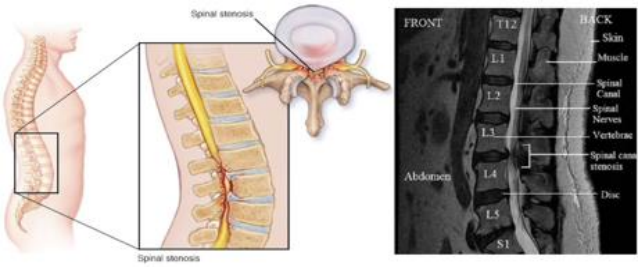
EXTRA: Rheumatoid arthritis x-ray finding Mnemonic: **LESS**
L: loss of joint space (Symmetric), **E:** erosion of joint, **S:** synovial thickening,
S: subluxation and joint deformities.

★ Complication:

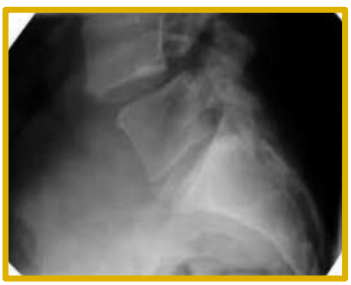
1. Loose bodies.
2. Rotator cuff dysfunction: Acromioclavicular (AC) joint OA. Osteophytes compress the joint and overlying rotator cuff causing impingement of rotator cuff and tendonitis and later on tear
3. Spinal stenosis.
4. Spondylolisthesis.
5. Capsular herniation: Knee OA; marked effusion and herniation of the posterior capsule (Baker's cyst).



Spinal stenosis very dangerous



Spondylolisthesis: Severe segmental instability at L4/ L5.



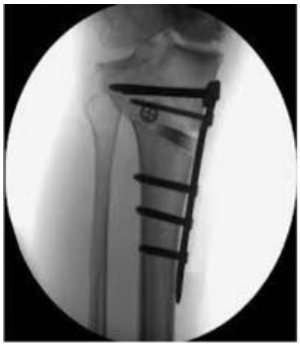
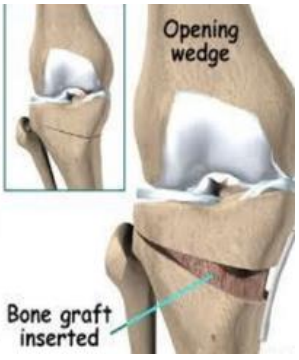
★ Management: Depends on several factors:

- Joint (or joints) involved.
- Stage of the disorder.
- Severity of the symptoms.
- Age of the patient.
- Functional needs.

Early treatment:

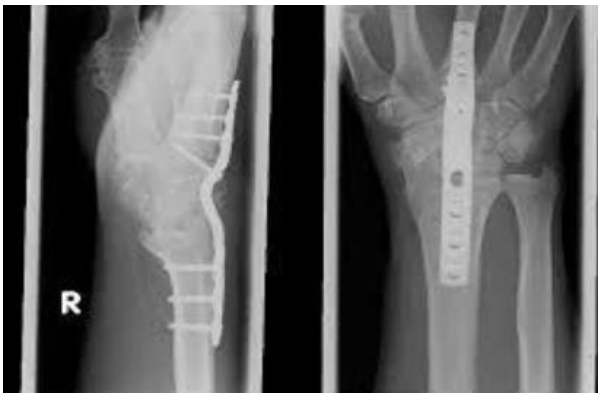
- Maintain movement and muscle strength. Strengthening the muscle & improving the ROM is an important part in extending the life of a joint.
- Protect the joint from overload.
- Relieve pain.
- Modify daily activities.

Conservative treatment:	
Maintain movement and muscle strength: by **physiotherapy** which will help in: 1- Pain relief (either by massage or application of warmth) or by other different types of physiotherapy 2- Prevents contractures. 3- Muscle strengthening. 4- Enhancing the range of motion.	**Protect the joint from overload (Load reduction):** 1- Weight reduction. (if the patient obese) 2- Shock-absorbing shoes. 3- Walking stick. 4- Unloading brace.
Modify activity and sitting habits: Pray in chair instead	**Medication:** ⇒ Oral: paracetamol, NSAIDs, muscle relaxants, narcotics, supplements and herbs. ⇒ Injections: (Local) **not** recommended in general.

Surgical treatment:	
Joint Debridement (Arthroscopy) تنظيف الركبة ○ Removal of: Loose bodies, Meniscal tear or labral tears. For mechanical symptoms. Not common procedure, done under GA, done commonly in private hospital, not indicated and will not correct the disease, used only if there is indication for it.	**Corrective osteotomy** ○ Realign axis and redistributes weight. ○ Knee; hip. ○ Has a role in pain relief by: 1- Vascular decompression of the subchondral bone. 2- Redistribution of loading forces toward less damaged parts of the joint. ○ Usual candidates are: **3** 1. **Young** less than 50, after 50 is better to do total knee replacement. 2. **Active** patients. 3. **Mild OA.**  

Arthrodesis

- Transfer from painful stiff into painless stiff joint.
- Small joints; hand, foot and spine.



Arthroplasty (Joint replacement)

Nowadays; the **procedure of choice** for advanced OA.

#**Total** joint replacement:

- Knee, hip, shoulder, ankle and elbow.
- Painful, deformed stiff joint, old patients.

#**Partial** joint replacement:

- Knee joint.
- Same patient as for osteotomy. (young, active, mild OA)

Excision arthroplasty

⇒ Resection arthroplasty.

⇒ Thumb, AC joint, Hip.

EXTRA: SYMPTOMS AND SIGNS AT DIFFERENT JOINT SITES: (435 team)

#HIP:

- Pain is usually felt in the groin, laterally over the hip and radiates down the anterolateral aspect of the thigh to the knee.

Occasionally the pain can radiate beyond the knee. Referred pain felt only in the knee is not uncommon, and clinicians should always consider hip OA as a cause of isolated knee pain.

- Pain is worse on exercise and walking distance is reduced. Pain at rest and night pain can be particularly troublesome.
- Stiffness is usually experienced first thing in the morning and after having sat still for a while, but it quickly resolves on movement to be replaced by pain. Complex movements, such as getting in and out of a motorcar or putting on socks, which involve deep flexion combined with rotation, are often difficult or impossible to perform. Patients struggle with stairs and in the absence of a banister may only manage stairs on all fours.

EXAMINATION:

- **Antalgic gait,** characterized by an uneven cadence, in which less time is spent in the stance phase of the painful limb.
- There is a **globally reduced range of movement** with internal rotation often restricted early in the disease progression.
- Joint movement is **limited by pain** at the extremes of movement.

Management: Weight reduction + pain management + physiotherapy

#KNEE:

- Knee osteoarthritis occurs most commonly in the **medial tibiofemoral joint** but can occur in all three compartments and is often tricompartmental. Isolated patellofemoral OA is probably due to altered biomechanics of the extensor mechanism.
- Pain is felt globally over the knee and the proximal tibia. In isolated patellofemoral OA the pain is felt anteriorly over the knee and is often worst when ascending or descending stairs as the patella is compressed against the femur.
- As in the hip, the pain is a deep-seated aching sensation related to exercise. Rest pain and night pain develop in the later stages.
- Patients sometimes report **audible crepitus** (crackling or grating sounds) coming from the knee as well as **symptoms of instability** (a feeling that the knee is going to give way).
- They may notice gradual deformity of the knee, in **particular varus deformity**, but less commonly valgus deformity. **Fixed flexion deformity** means that the knees cannot lock in full extension and thus patients cannot stand comfortably for prolonged periods due to muscle fatigue. Loss of flexion beyond 90 degrees makes standing from a sitting position difficult as patients cannot move their centre of gravity anterior to their mid-coronal plane. Swelling and stiffness are common features.

EXAMINATION :

- **Antalgic gait, wasting of quadriceps muscles, joint effusion.**
- **Joint deformity** the joint deformity may be passively correctable. Deformity is towards the compartment most severely affected, usually varus deformity with predominantly medial compartment OA.
- **Crepitus palpable and sometimes audible on movement.**
- **Tenderness along the joint line and palpable osteophytes that can be tender.**

#HAND:

- Most commonly affecting **DIPs and the thumb base** (both the radiocarpal and scaphotrapeziod joints), less commonly, PIPs joints and metacarpophalangeal joints.
- OA of the hand is strongly associated with OA at other joint sites, especially the knee, and with genetic predisposition. It is far more common in **women** starts relatively abruptly around the time of the **menopause** (sometimes called 'menopausal OA') with painful inflammation in DIPs joints over time(years) the inflammation settles and the joint is left with the typical pathological features of OA.
- **Erosions** can occur ('erosive OA'), and cysts containing hyaluronan that protrude at the margins of the joints are not uncommon.
- Distal interphalangeal joint OA is not generally a major problem in terms of function, but thumb base OA can be, as it leads to instability and difficulty with pinch grip.

#Other joints:

Almost any joint can be affected by OA, particularly if it is damaged by severe trauma.

However, there are peculiarities to the phenotype of the condition at different sites. For example, elbow OA is almost always asymptomatic (just causing loss of full extension of the elbow), while shoulder OA is more likely to result in severe bone destruction (a condition sometimes called 'Milwaukee shoulder') than is OA at other joint sites.