



Workplace Safety and Insurance  
**Appeals Tribunal**

**Tribunal d'appel** de la sécurité professionnelle  
et de l'assurance contre les accidents du travail

---

# Osteoarthritis

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

November 2008

Prepared by:

Dr. Marvin Tile

MD, B.SC(Med), FRCSC Professor Emeritus, Department of Surgery,  
University of Toronto Orthopaedic Surgeon, Sunnybrook Health Sci-  
ence Centre

Dr. Marvin Tile graduated from the University of Toronto Medical School in 1957. He did post-graduate training in Orthopaedic Surgery at the University of Toronto from 1958 to 1963, and was awarded the Royal College Fellowship in Surgery (Orthopaedics) in 1963. He was granted the Detweiler Fellowship in 1963 and travelled extensively in Europe, visiting leading orthopaedic centres. He joined the faculty at the University of Toronto in 1966 and holds the rank of Professor (Emeritus) in the Department of Surgery (Orthopaedics).

His clinical and research interests have been in orthopaedic trauma care, and also in the management of arthritis, including hip and knee arthroplasty. He also has a major interest in low back pain.

He has published widely, especially in orthopaedic trauma. He has authored two texts: *Fractures of Pelvis and Acetabulum*, Lippincott, Williams & Wilkins, 3rd Edition, 2003 and *Rationale of Operative Fracture Care* with Dr. Joseph Schatzker, Springer-Verlag, 3rd Edition, 2005, now in 6 languages. Since 1966, he has been on the Active Staff in orthopaedic surgery at Sunnybrook Health Sciences Centre, a University of Toronto, fully affiliated hospital. He was Chief of Orthopaedic Surgery at that institution from 1971 to 1985 and Chief Surgeon from 1985 to 1996. He has been elected to many prestigious positions. He was the founding president of the Ontario Orthopaedic Association (1978-80), Past President of the International Society for the Study of Lumbar Spine (1986-7), Past President of the Canadian Orthopaedic Association (1991- 2), and in 1992-4, Past President of the AO Foundation, Switzerland (devoted to research and education in fracture care, world wide). As well, he was Chair of the Sunnybrook Foundation (1996-2001). An endowed Chair in Orthopaedic surgery has been established in his name at Sunnybrook HSC and the University of Toronto. Dr. Marvin Tile has been a medical counselor in orthopaedics for the Tribunal from 2004.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

## Introduction

### What is arthritis?

The word *arthritis* comes from the Greek; “arthro” meaning joint and “itis” meaning inflammation. With common usage, disorders of synovial joints with little evidence of inflammation (itis) are also included in this broad group of disorders. *Arthrosis* is a more inclusive term, and is used by many physicians to more correctly designate these disorders. Arthritis occurs in a synovial joint (definition below) including small joints such as the finger joints or large joints such as the knee, hip and shoulder.

There are many types of arthritis including but not limited to:

1. those caused by an inflammatory disease of joints of unknown etiology, such as rheumatoid arthritis, or seronegative arthropathies, such as ankylosing spondylitis, psoriatic arthritis and others (see Discussion Paper Trauma and Inflammatory Arthritis)
2. those caused by an inflammatory disease of joints of known etiology, such as a specific bacterial infection in the joint:
  - examples of acute infections include staphylococcus, streptococcus, or gonorrhea
  - others causing more chronic infection in the joint include tuberculosis.
3. Osteoarthritis

### What is osteoarthritis?

*Osteoarthritis* is the most common form of arthritis and indicates damage to the articular surface of a joint. It is not a single disease; rather it is the end result of a variety of joint disorders. Although inflammation is observed within the joint in the chronic course of osteoarthritis, it is not the prime cause of osteoarthritis. The distinction from true inflammatory arthritis is noted above. Although common usage has retained the name osteoarthritis (OA); a more proper name would be *osteoarthrosis* to denote that the primary cause is not inflammatory, but due to other causes. Other names include *Wear and Tear Arthritis* or *Degenerative arthritis*.

There are many causes of OA; the two common types include:

1. *Primary Idiopathic Generalized OA* is of unknown etiology, but is associated with genetic or familial factors, occurring mostly in the older

age group, usually more common in women and involving the small joints of the hands and feet, as well as the large joints.

2. Secondary OA has a known etiology, the degenerative joint disease occurs in response to a recognizable local or systemic factor. For our purpose, those caused by mechanical factors, such as deformity or malalignment in the limb, or injury to a joint are most relevant.

Although there is no generally accepted definition of osteoarthritis most will agree that the joint disorder has the following characteristics, pathologically, radiographically and clinically:

*Pathologically*, OA is a condition of synovial joints characterized by damage to the articular surface of the joint and the accompanying reparative joint response.

*Radiographically*, this leads to a characteristic appearance on plain x-ray, CT or MRI showing cartilage damage and the reparative process. Patients with x-ray changes showing osteoarthritis may or may not have clinical symptoms. *Clinical osteoarthritis* occurs when the involved joint becomes painful, with possible swelling, and/or with altered joint function.

**Table 1 Types of Osteoarthritis**

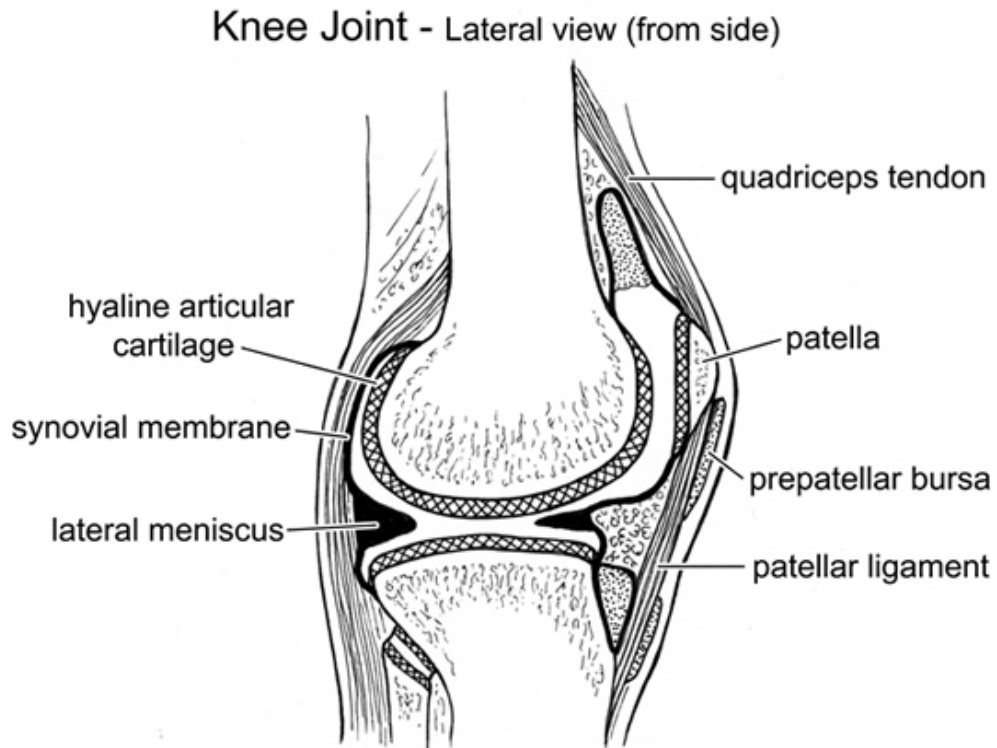
<b>Types of Osteoarthritis</b>	<b>Varieties include:</b>
<b>1. Primary Idiopathic Osteoarthritis</b>	<ol style="list-style-type: none"> <li>a. Nodal or erosive</li> <li>b. Generalized or limited to limited number of joints</li> </ol>
<b>2. Secondary Osteoarthritis</b>	<ol style="list-style-type: none"> <li>a. Injury to a joint (post traumatic OA)</li> <li>b. Biomechanical factors (including congenital dysplasia, limb or joint deformity)</li> <li>c. Sequelae of inflammatory joint disease, including sepsis, rheumatoid arthritis, gout, pseudogout, and seronegative arthropathies</li> <li>d. Avascular necrosis</li> <li>e. Secondary to generalized conditions, such as acromegaly, Ochronosis, Ehlers-Danlos, etc</li> <li>f. others</li> </ol>

## Synovial Joints

**Understanding a synovial joint** is essential to our understanding of osteoarthritis. In a synovial joint two bones meet, each surfaced by hyaline articular cartilage allowing movement within a fibrous capsule. The interior lining of the joint is made up of a synovium which does not cover the cartilage, but serves to nourish the joint with a viscous fluid called synovial fluid.

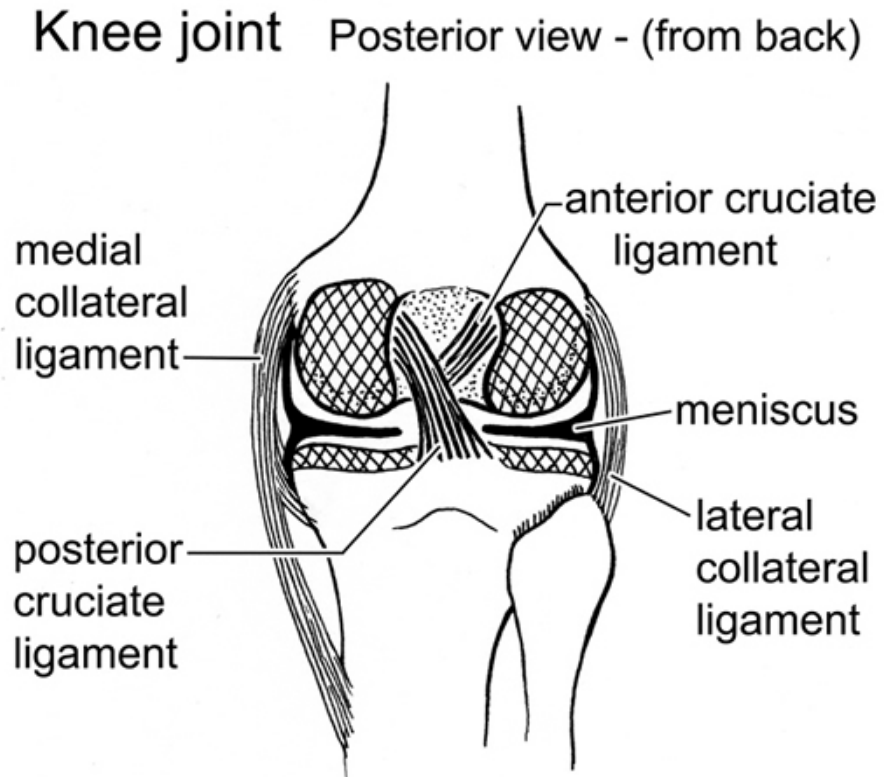
### Figure 1: a. Synovial Joint: Gross appearance Normal Appearance of the Knee Joint From the Side (Lateral) View

Note the smooth articular cartilage surface, the menisci, and the synovial membrane. The ligaments give stability to this joint, the medial and lateral ligaments give side to side stability and the cruciates front to back (anteroposterior).



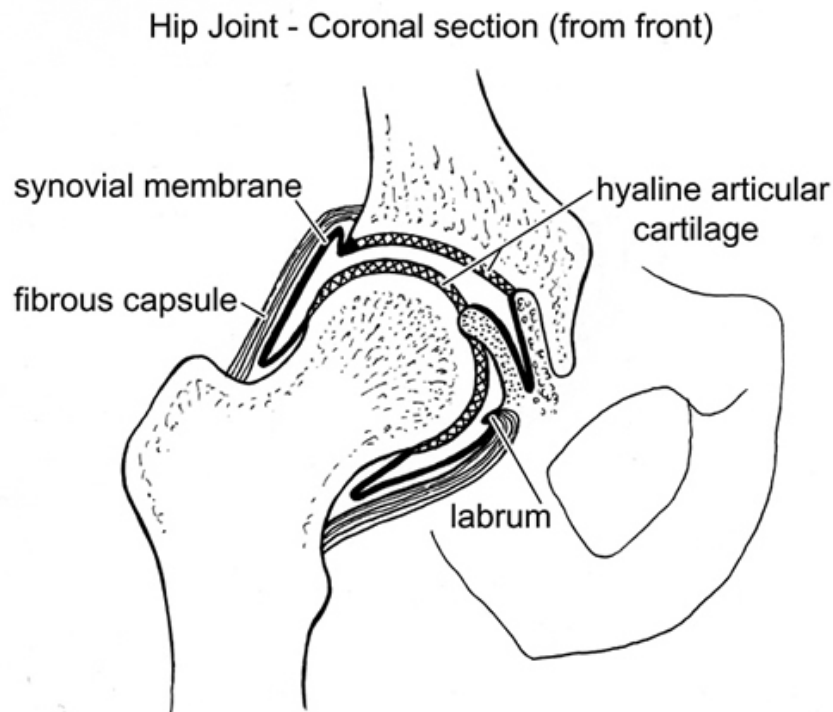
**Figure 1: b. Synovial Joint: Gross appearance. Normal Appearance of the Knee Joint From the Back (Posterior) View**

The ligaments give stability to this joint, the medial and lateral ligaments give side to side stability and the cruciates front to back (anteroposterior).



**Figure 1: c. Synovial Joint: Gross appearance Normal Appearance of the Hip Joint on Cross Section**

Note the similarities to the knee, but in the hip, the meniscus structure is called the labrum as noted. The hip has inherent stability as a ball and joint, so the ligamentous structures are less elaborate.



**Articular cartilage**

The joint surface is made up of articular or hyaline cartilage. This is a translucent extremely smooth surface substance with a low coefficient of friction allowing the two surfaces to glide over each other.

The surfaces of the bones covered by hyaline cartilage can glide smoothly because of the low coefficient of friction, which is less than ice on ice and because of the lubrication from synovial fluid.

This highly specialized articular hyaline cartilage once damaged, cannot repair itself with the same tissue, so that any repair is with a form of fibrocartilage, a less specialized form of cartilage less able to withstand any abnormal stresses on the joint.

## **Synovial Lining**

The synovial lining is a thin membrane which lines the interior surface of the joint but not the articular cartilage. It contains cells that produce synovial fluid which lubricates the articular cartilage, thereby reducing the coefficient of friction and allowing effortless motion. The surface of articular hyaline cartilage does not have its own blood supply, it is an avascular tissue; therefore, the surface cartilage cells are nourished entirely by the absorption of nutrients from the synovial fluid.

## **Fibrous capsule and ligaments**

The joint is held together by a fibrous capsule. In all joints of the body, portions of the capsule are condensed to form ligaments which are structures that help stabilize the joint. Each joint has a specific set of ligaments. These ligaments may be extrinsic<sup>1</sup>, such as the collateral ligaments of the knee, or intrinsic<sup>2</sup> such as is the cruciate ligaments in the knee joint, so essential to maintaining joint stability. These ligaments and the specific local anatomy of the joint is what predetermine the motion in a joint. For example in large joints, the shoulder has motion in many planes as does the hip, whereas other joints such as the knee and elbow have minimal rotational ability and mostly move as a hinge.

## **Menisci**

Some joints have clearly defined menisci that are always attached to fibrous capsule and/or a ligament at the synovial lining. Menisci are made of fibrocartilage and have no specific blood supply except at their attachment to the synovial lining and therefore, if injured, cannot heal except at the periphery. They have many functions but mostly act as a washer, increasing the joint articulating surface. Joints such as the shoulder and hip have a labrum surrounding the periphery of the entire articular surface somewhat as the menisci do in the knee joint.

## **Bone**

In the area immediately adjacent to the articular cartilage called the subchondral area, the bone is extremely dense and is called subchondral bone. Further from the joint surface, the central portion of the bone is porous in nature and is referred to as cancellous or spongy bone containing a rich blood supply and much of the marrow of the body. This bone is different than the cortical bone found in the mid shaft of the long bones.

---

<sup>1</sup> Definition - Originating outside of the part where found or upon which it acts.

<sup>2</sup> Definition - Belonging entirely to a part.



## Types of osteoarthritis

### 1. Primary (Generalized) Idiopathic Osteoarthritis

This condition is common, with high prevalence in society. It affects at least 60% in patients over the age of 60; by 80 it can be found in almost the entire population, in one or more joints. The etiology is generally unknown, but there are several associated risk factors as outlined.

#### ***Risk factors include:***

*Age:* The incidence of OA rises as one ages. The symptoms usually begin at approximately age 50, and are almost inevitable in one or other joint during ones lifetime.

*Gender:* Both sexes are affected, but the incidence is higher in women.

*Obesity:* Obesity is a factor in the progression of symptoms. Recent studies have shown that increased body mass has a greater effect on the knee joint, and little effect on the hip.

*Overuse Factors:* There is extensive literature linking overuse and occupation as risk factors in causing primary osteoarthritis, but none have shown a definite link.

*Genetics, Familial:* There is a clear familial link to primary OA. This may be related to a gene determining longevity of articular cartilage. Research is continuing in that field. In the epidemiology of hip OA, familial factors are important; it has been seen in identical twins.

#### **Several subtypes of Primary (Generalized) Idiopathic Osteoarthritis are seen, including Nodal and Erosive.**

The Nodal type usually involves the hands and is characterized by a multiple Heberden's nodes in the distal interphalangeal joints of the hands and nodes in the proximal interphalangeal joint (Bouchard's nodes). The *Erosive types* usually involve the large joints.

*X-Ray changes of OA*, especially in the hands, but also in other joints may not mirror the symptoms, in fact many patients with such changes may have few or very mild symptoms.

In the large joints, especially in the lower extremity (hip, knee), the symptoms may become progressive and disabling. In any given joint, the symptoms may require medical and possibly surgical intervention (joint replacement). For example, the vast majority of the common surgical procedures of total hip

replacement are done for the primary idiopathic familial type of osteoarthritis; individuals with no mechanical involvement of the joint, from all walks of life, with sedentary and heavy occupations.

## 2. Secondary Osteoarthritis

Mechanical osteoarthritis usually involves a single joint, and has a known cause. Most are caused by a biomechanical abnormality to the joint or limb or a direct injury to the joint. (post-traumatic arthritis)

*Biomechanical factors* causing secondary osteoarthritis include congenital joint disease, such as congenital dysplasia of the hip joint.

Deformity in the limb, especially in the lower extremity, may also cause altered force transmission through the joint leading to osteoarthritis. This is most common in the knee joint where developmental deformity such as bowlegs or knock knees; or post traumatic deformity, may lead to premature osteoarthritis.

*Post-traumatic osteoarthritis* which develops after joint injury is a common well recognized disorder. The joint injury may be acute, following a fracture of the articular surface or a ligamentous injury to the joint, or chronic, (wear and tear).

### Acute Injury

#### *Compression Injury of the Joint Surface (Fracture)*

Based on the type of injury, the articular surface injury caused by compressive forces can be classified into *three main types*. In any type, once a defect occurs in the hyaline cartilage especially by an intra-articular fracture, the ability of hyaline cartilage to regenerate is severely limited.

#### 1. *Cartilage Bruising*

Damage to the joint may leave the overlying articular surface intact on a plain x-ray, but MRI's may show ligament tears and may show bruising and edema of the subchondral bone. It is not certain what this bruising means for the future function of the joint but it is not infrequently seen in major ligamentous injury to joint. In some instances cells which produce matrix for the hyaline cartilage may be damaged leading to secondary type osteoarthritis. At this time, secondary OA from this type of bruising is uncommon, and can only be followed by further MRI. In most cases, the bruised cartilage resolves in time, with no major sequelae.

#### 2. *Cartilage disruption*

Impact injuries disrupting articular cartilage may not extend into the subchondral bone, but are not infrequently seen when surgeons operate to restore joint surfaces in intra articular fractures. In time, these cartilage defects, if large, may leave a permanent articular surface defect that can alter joint mechanical function and increase the risk of joint degeneration.

### *3. Intra-articular fracture*

Fractures in adults often occur at the ends of the bone and frequently involve the articular hyaline cartilage. If these intra-articular fractures occur in the large joints, especially the weight bearing joints of the lower extremity, major disability can ensue if joint anatomy is not restored. Orthopaedic surgeons try to prevent post traumatic osteoarthritis by attempting to restore joint congruity, alignment and stability. If these goals are achieved, the prognosis for restoration of joint function is good, depending on the severity of the injury and the success of the surgery. However, in spite of this optimal treatment, some patients may continue to have pain and disability, because the prevalent force causing these fractures is compression. These compressive forces may also impact the surrounding articular cartilage, causing permanent damage, and eventual post traumatic OA. In these cases, post traumatic arthritis may develop early or late, again depending on many factors, the injury factors (severity of articular disruption, restoration of the joint surface joint congruity and stability, lack of surgical complications) and the patient factors (age, comorbidities, complications, associated injuries)

### **Joint injury to ligaments (soft tissue)**

Joint injury may also occur through the ligamentous stabilizing structures, the ligaments may be stretched or strained or may be completely torn causing joint instability. These injuries are usually caused by shearing forces, which may cause joint instability and ligament injury with less effect on the articular cartilage. Also the menisci, especially in the knee joint, may be injured by these shearing rotational injuries. Meniscal tears which do not heal unless they are in the periphery can cause continuing disability. Recent trends to surgical repair of these peripheral meniscal tears may improve the long term prognosis. Even if surgery is performed, the patient often shows radiographic OA within 10 years, and may also develop clinical OA.

Patients with ligament injury, with or without repair may also develop joint instability. The relationship between joint instability and the development of subsequent OA involvement is not well defined, because it depends on many factors, including the joint involved, the age at the time of injury and the degree and direction of the instability. There are clinical and experimental studies that do show increased risk of OA in unstable joints, particularly the knee.

## Chronic trauma (wear and tear)

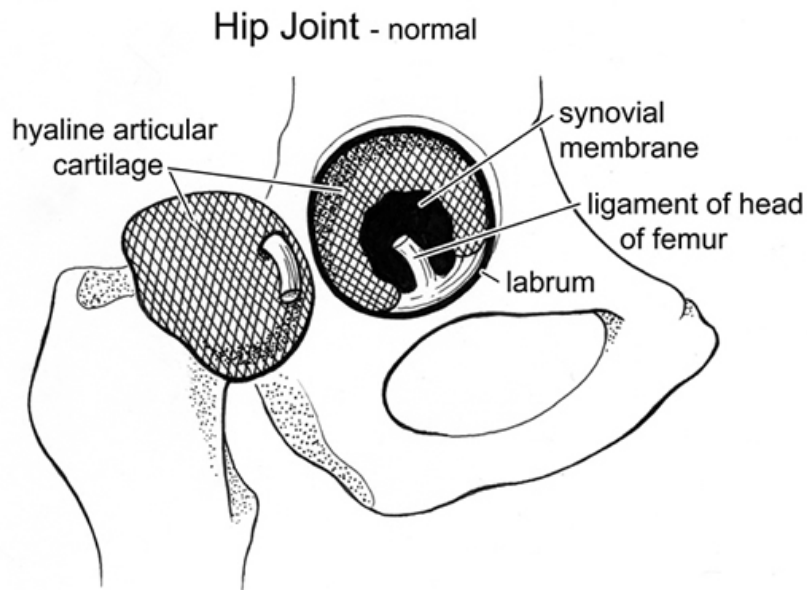
The effect of repetitive trauma on joints is difficult to evaluate, there is extensive literature on the subject, but few studies that stand up to rigid scrutiny. This will be discussed later in this paper

## Limb Deformity

In the lower extremity, residual limb deformity after femur or tibia fracture, may cause uneven loading on the distal joint (knee, ankle) leading to OA long after the event.

### Figure 2: a. Articular Cartilage Hip Joint

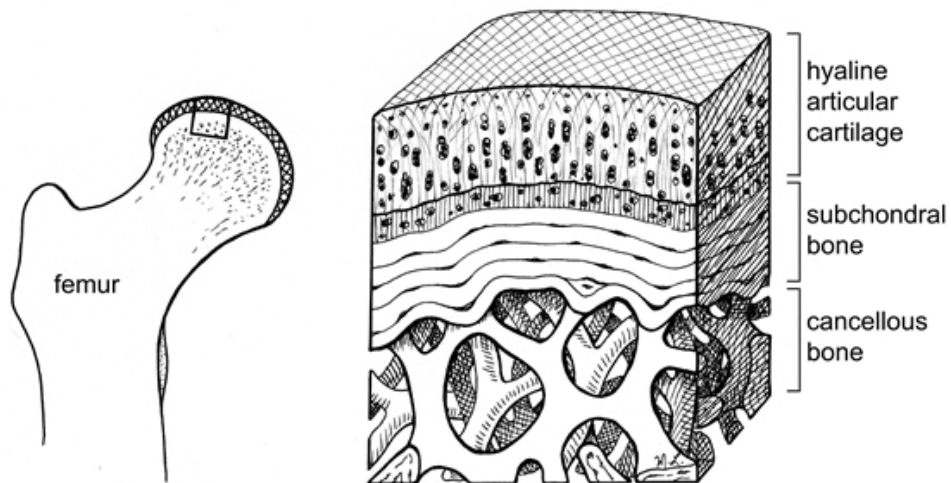
Normal appearance of the open hip joint from the front (anterior). Note the articular smooth surface on the femoral head and the acetabulum, except for the area of the acetabular fossa, at the insertion of the ligament to the head (ligamentum teres)



## Figure 2: b. Articular Cartilage Hip Joint

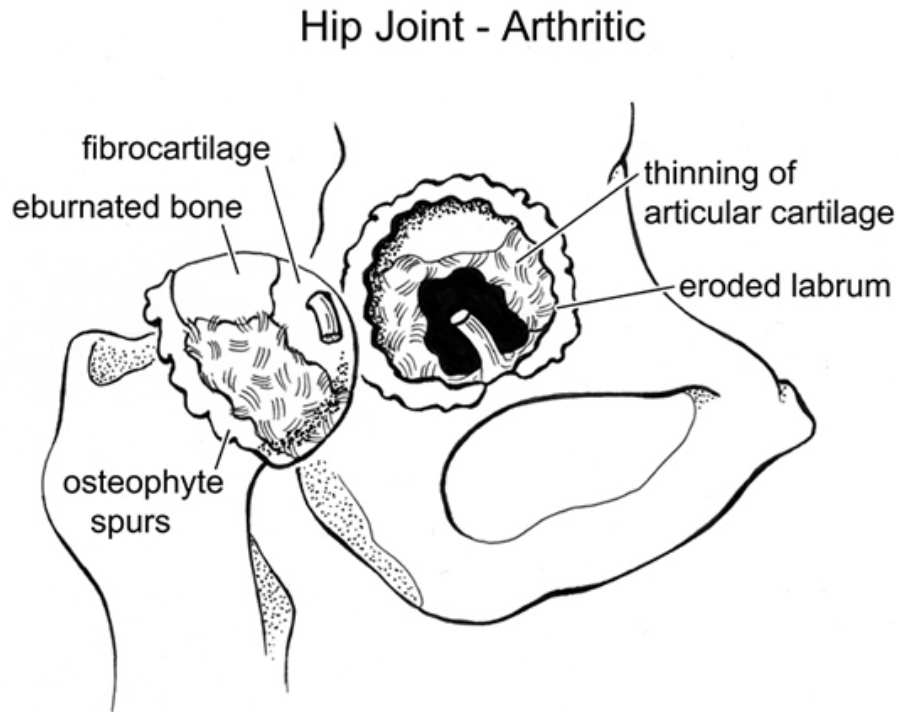
Normal Articular Cartilage of the Hip in cross section. The femoral head is covered by this very smooth surface; the microscopic section shows the layers of normal articular (hyaline) cartilage. Note the smooth surface; the cells on this surface have no blood supply, are nourished by diffusion of synovial fluid, and cannot regenerate, once damaged.

Hyaline articular cartilage & bone - normal



### Figure 2: c. Articular Cartilage Hip Joint

Abnormal appearance of the open hip joint from the front. Note the worn articular surface covered now with eburnated bone on both surfaces, and the large spurs (osteophytes). The worn surface is mainly on the weight bearing aspect of both the femoral head and acetabulum.



## Figure 2: d. Articular Cartilage Hip Joint

Abnormal Osteoarthritic Joint surface (hip) in cross section. The surface is rough, with many defects on the surface. The microscopic section shows the ulcer, the attempt at healing with fibrocartilage. Eventually, the surface is covered with eburnated bone, a poor bearing surface.

### Hyaline articular cartilage & bone - Arthritic

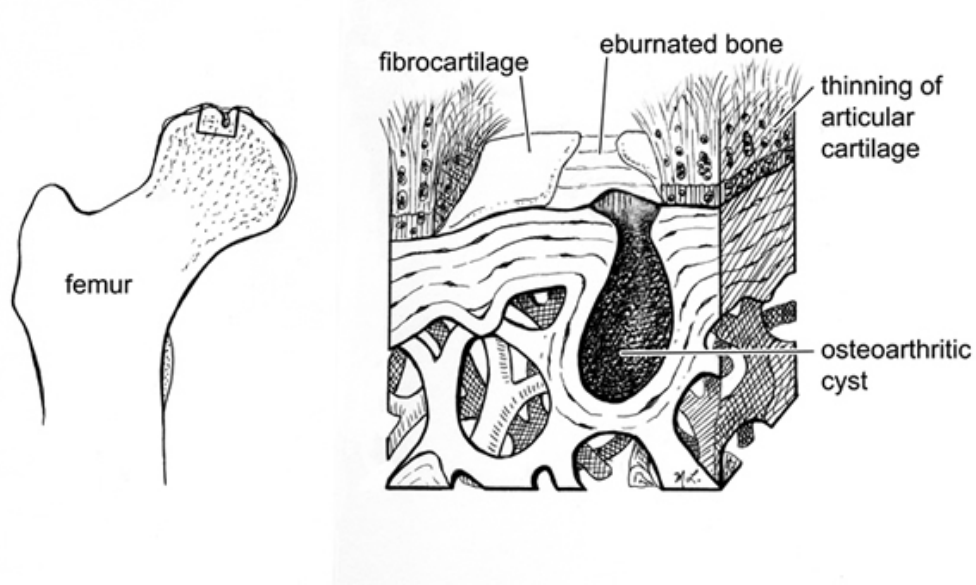


Figure 2.1: X-ray showing severe destruction of the right hip (OA)

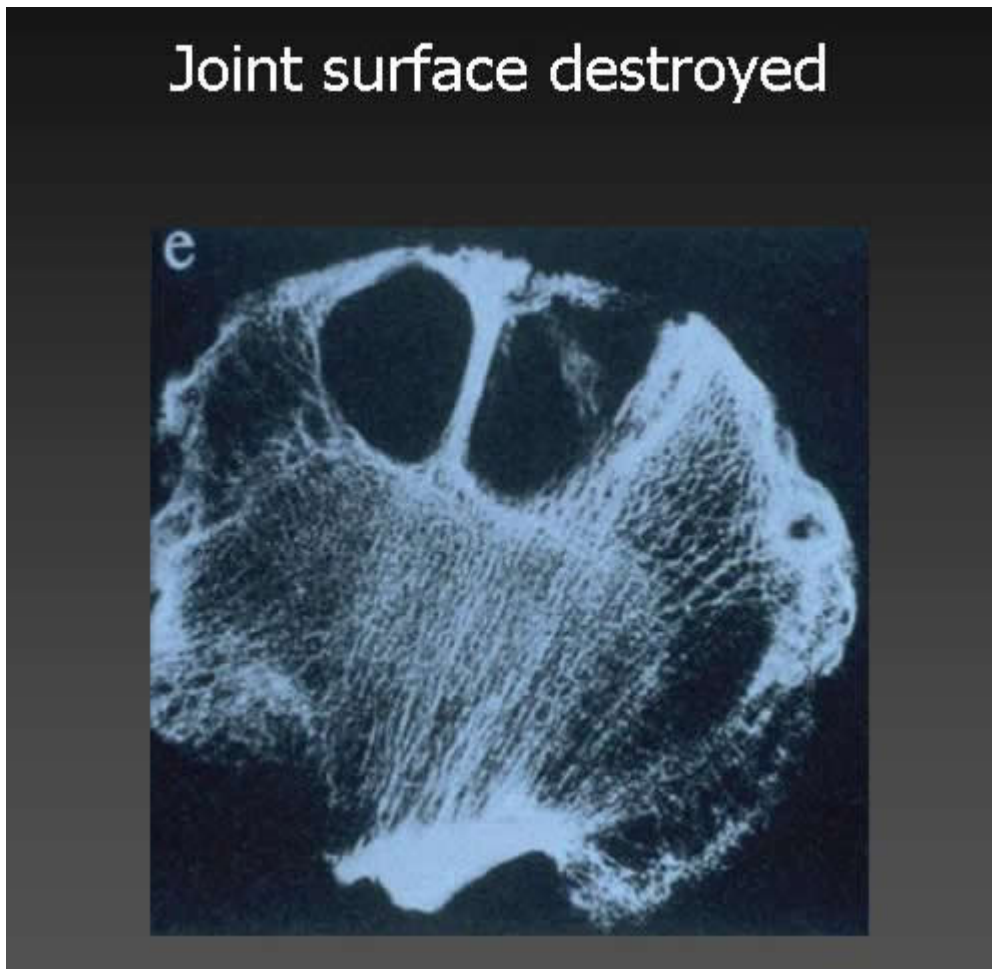




Figure 2.2: The gross appearance of the femoral head at total hip arthroplasty



Figure 2.3: X-ray showing severe destruction of the femoral head

Gross appearance of the femoral head at total hip arthroplasty



## Sequelae of joint injury

### **Pathologic features**

No matter what the cause, be it primary osteoarthritis or secondary, once a defect occurs in the articular surface of the joint, the degenerative process may ensue. Cracks may occur around the defect, the defect may deepen and the articular cartilage lost. The underlying bone then becomes the joint surface, the surface becomes polished or eburnated<sup>3</sup>. This surface is less suitable than articular cartilage for joint movement. The joint and the bone beneath it respond by increasing density in the subchondral bone under the defect and osteophytes or spurs occur along the joint margin. At a later stage, the resulting osteoarthritic joint has large defects in the articular cartilage, thicker bone in the subchondral area, osteophytes (spurs) on the joint margin and in most cases, the joint becomes stiffer. At times during the progressive degenerative process inflammation may ensue, causing acute flare-ups of symptoms.

### **Radiographic features**

Early OA may show no change on plain x-ray, since articular cartilage cannot be seen directly. However, the so called joint space can be seen, and is filled with articular cartilage; therefore one of the early radiographic findings is decrease in the joint space. In the lower extremity this is best seen on weight bearing films comparing one side to the other. As degeneration progresses, one sees a complete loss of the joint space and marked increased density in the subchondral bone and large osteophytes. Eventually, large joint surface cysts and erosions may be seen.

A technetium Bone Scan may be helpful as it may show inflammation in a joint and also increased bone formation. In cases where the radiographs appear normal a technetium bone scan is a good screening tool. For sepsis, a good screening tool is either a gallium or an Indium scan.

MRI has replaced CT as the imaging tool of choice in early OA, as the MRI can effectively show all the anatomic features in a joint. The MRI may show early lesions in articular cartilage, and/or “swelling and bruising” in the subchondral area following acute injury to joints as well as ligament tears.

### **Clinical features**

Pain is the primary symptom, usually with motion but in more severe cases even at rest. The joint may or may not have swelling and bony enlargement from the spurs.

---

<sup>3</sup> Definition - hard and dense like ivory.

On examination, the joint margins may be tender, and motion may be restricted. The appearance of the joint both clinically and radiographically does not always parallel the degree of symptoms. In some patients, symptoms of pain and stiffness will be marked but there is little to see clinically or radiographically. In other circumstances, the x-rays and clinical examination are dramatic but the symptoms are minimal. In joints that are subcutaneous, such as the knee, and hand; exacerbations of pain and stiffness may be seen with inflammatory flare-ups consisting of joint inflammation. In these circumstances, the joints exhibit swelling, and other signs of inflammation, such as heat, redness, and tenderness.

## **Treatment**

Treatment of osteoarthritis may be medical or surgical. Medical treatment consists of drug therapy with the use of anti-inflammatory drugs (aspirin, NSAIDs), local injections of the joint with steroid in the inflammatory phase, and if the joint becomes stiff, an active physiotherapy rehabilitation process.

Surgery should always be a last resort. At this time, in larger joints such as the knee, hip and shoulder, joint replacement (arthroplasty) is the treatment of choice in individuals over the age of 50.

Other procedures, such as arthroscopic debridement have been questioned, but are useful in select cases.

Osteotomy meaning realignment by cutting the affected bone and repositioning it, should be considered in younger individuals with a good joint surface but with a deformity in the limb, usually in the lower extremity.

Joint arthrodesis is rarely used now in large joints but may be useful in the region of foot, hand and wrist.

## **Questions commonly asked about osteoarthritis**

1. *What is the Incidence of Primary osteoarthritis?* What is the usual incidence of this condition in the general population? Is gender related is it more frequently found in some joints than others?

Eighty percent of the population over the age of 50 has x-ray evidence of osteoarthritis in one or more joints. There is a greater prevalence in females especially in the small joints of the hand and feet. The hip and knee joints are the commonest large joints involved, but the shoulder, elbow and ankle are not spared. The condition is often bilateral, although the opposite joint OA may not appear for several years. The carpal and tarsal joints are frequently involved with a definite familial pattern well documented.

2. *What is the Age of onset?* The symptoms usually become prevalent in the 50's and 60's, in primary OA, but at any time in secondary.

3. *Does the presence of osteoarthritis in the x-ray of an asymptomatic patient suggest that he or she will likely become symptomatic?*

This disease is often, but not always, slowly progressive on x-ray; and in those cases, the patient may become aware of pain and stiffness in the involved joint. The individual clinical response to the presence of OA on x-ray and the degree of disability varies markedly.

4. *What factors might precipitate the onset of symptoms of in osteoarthritis?*

Episodes of inflammation in the joint may precipitate the onset of symptoms. Trauma to the joint may also initiate symptoms

5. *When an individual exhibits symptoms of osteoarthritis do they correlate with the x-ray findings?*

There is no direct correlation between the x-ray findings and symptoms of osteoarthritis. In the early stages the x-ray may be normal, but the patient may be symptomatic from inflammation. This can often be seen on an MRI.

In the late stages, patients with very marked x-ray changes, especially in the large weight bearing joints such as the knee and hip, usually have symptoms, but as noted above, the degree of disability, and the treatment required, varies markedly.

6. *Is osteoarthritis one joint or multiple joint?*

*Primary osteoarthritis* is usually a multiple joint disease. In the *nodal type*, the same joints of the hands are involved bilaterally; also, *erosive primary OA* involving the large joints is commonly bilateral.

In *secondary arthritis*, the joints involved are related to the specific cause, and usually involve a single joint.

### **Questions commonly asked about Post-traumatic Arthritis**

1. *Specific injuries may lead to osteoarthritis of the joint and if so would it be only in that joint or in other joints opposite or the same extremity?*

This has been discussed this in the body of the report above but will respond as follows.

*An injury to a joint but not involving damage to the joint surface or cartilage.*

Usually rotation or shearing type injuries and may cause ligament strain or ligament rupture. The joint surface looks normal on plain radiograph and even on MRI or arthroscopic intervention. In those circumstances, it is unlikely that a lesion will develop in articular cartilage and osteoarthritis would therefore be rare.

In some injuries, bruising and edema has been noted on MRI. There have been some linear studies; in most cases, the bruising and edema on MRI disappear with no major clinical sequelae. There are rare cases where the cartilage impact has caused cell death leading to late osteoarthritis, that situation would be rare.

*An injury involving damage to cartilage, meniscus or joint surface.*

*Intra-articular fractures*, by definition going through subchondral bone and articular cartilage, cause permanent damage to the joint. If left in an unreduced state especially in the lower extremity in younger individuals, osteoarthritis will ensue quickly. If surgical intervention restores the joint surface to normal anatomy, then the onset of posttraumatic OA of the large joints may be delayed for many years. However if a young individual (e.g. age 20) sustained an intra-articular fracture to the tibial plateau in the knee and came back at age 50 with an osteoarthritic knee, almost certainly, there is a direct causal relationship.

*Cartilage damage.* Articular cartilage may be impacted without an early radiographic abnormality noted. This may occur in high impact ligamentous disruption especially in the knee; and by major compression injury in the hip. This can be seen on early MRI if available or, should the joint be operated upon either arthroscopically or by open technique. These injuries are not infrequent. These injuries may eventually lead to radiographic or clinical OA; whether it becomes clinically important will depend on many other factors as discussed.

*Meniscal injuries* are mainly noted in the knee joint, but if one considers the glenoid and acetabular labrum, may also include the shoulder and hip. These cartilaginous structures have no blood supply except at their peripheral attachment, therefore in substantive tears, the meniscus cannot heal. Peripheral tears along the synovial marginal attachment may heal if reattached surgically. Removal of a meniscus causes late secondary arthritis even in cases of incomplete removal such as a flap tear. The radiographic appearance of the joint is altered in almost every case at 10-20 years and clinical symptoms may ensue.

## *2. Does instability of a joint lead to osteoarthritis?*

Patients with ligament injury, with or without repair may also develop joint instability. The relationship between joint instability and the development of subsequent OA involvement is not well defined, because it depends on many factors, including the joint involved, the age at the time of injury and the degree and direction of the instability. There are clinical and experimental studies that

do show increased risk of OA in unstable joints, particularly the knee. 3. *Does employment in heavy work for many years cause osteoarthritis?*

Clearly, a specific traumatic work related injury can lead to post traumatic OA, either by direct or indirect means. Indirect factors may be limb deformity in femoral or tibial fracture causing altered biomechanics, and late OA.

Also, if the joints were immobilized for long periods of time in abnormal positions such as a cast (e.g. an ankle in plantarflexion >90), joint stiffness and late OA may ensue.

Although there is extensive literature on this subject, in my opinion, there is no compelling evidence linking occupations requiring heavy work and arthritis.

Work related OA from repetitive stress is recorded, but the studies are not credible with respect to cause and effect. It is well accepted that radiographic changes of OA occur in some joints in 80% of individuals over age 55, therefore, it is difficult to attribute these changes to occupation, when they are so common in the general population.

Epidemiologic studies do not support a causal relationship to heavy work. Hip and knee arthroplasty, the two commonest forms of intervention for primary OA, show no such direct relationship, in fact, there is a slight preponderance of women, not engaged in heavy work; in men, there are just as many done in individuals with sedentary jobs, (professionals: judges, lawyers, doctors, office workers, etc) than in workers engaged in heavy work (construction, trades, etc), following the incidence of these jobs in the general population.

Individuals that do heavy work are no more likely to develop OA than those that do sedentary work; however, the heavy work may render the joint more symptomatic, creating the impression that osteoarthritis is more common in these workers.

### **Questions commonly asked about Specific activities or injuries aggravating and/or accelerating an underlying primary osteoarthritis**

#### *1. Acute Injury*

Patients with primary OA can be affected by any of the types of acute injury noted above in any specific joint, which may aggravate the symptoms.

Many scenarios are possible: The OA may have been severe, and in some instances the worker may have already a booking for a total joint replacement. In this scenario, the injury, unless severe, would play only a minor role in exacerbating the symptoms, as they were already severe, requiring Joint Arthroplasty.

In others, the OA may be mild and the injury minor; whereas in others, the injury may be major; therefore the aggravating factor is multifactorial, and the cases must be reviewed individually. With mild injury, recovery should proceed to preinjury state quickly, whereas, in more major injury, the return to preinjury state may be more delayed. The doctors can be guided by the objective findings in each individual case, including the imaging studies.

## 2. *Chronic Strain*

In more chronic types of repetitive trauma, such as having to walk long distances daily in one's work, the symptoms of OA may be increased, but the objective findings on clinical and x-ray exam might not. There is no evidence that the arthritic process is accelerated by repetitive stress, but the joint may be more symptomatic.

For example an individual with bilateral knee osteoarthritis who was required to walk all day might become more symptomatic than an individual sitting at a desk who would have pain mainly when he or she got up from the desk and took the first few steps. The arthritic process wouldn't change but the clinical symptoms would.

## 3. *Prolonged static position and normal alignment*

Many patients with pre-existing osteoarthritis have difficulty with sitting for long periods or standing for long periods. Many are helped when they walk. Symptom complexes are extremely variable but a prolonged static position either standing or sitting would not accelerate the pathological process.

## Bibliography

### **Epidemiology and Risk Factors**

1. John Klippel (ed), *Primer on the Rheumatic Diseases*, Arthritic Foundation; Atlanta, Georgia, USA, 2001 Pg. 285-297.
2. Kenneth Brandt, Michael Doherty et al., *Osteoarthritis*, Oxford University Press, 1998, Pg 13-22.
3. Murphy L, Schwarz, TA et al; Lifetime risk of symptomatic knee osteoarthritis, *Arthritis and Rheumatism* 59(9): 1207-13, 2008.
4. Roux, C.H., Saraux, A., et al; Screening for Hip and Knee Osteoarthritis in the General Population: predictive value of a questionnaire and prevalence estimates; *Annals of Rheumatic Diseases*: 67(10), 1406-11.



5. Kopec JA, Rahman, MM et al, Trends in physician- diagnosed osteoarthritis in an administrative database in British Columbia, Canada, 1996-7 through 2003-4, *Arthritis and Rheumatism*, 59(7), 2008.
6. Brouwer GM, van Tol AW et al, Association between valgus and varus alignment and the development and progression of radiographic osteoarthritis of the knee, *Arthritis and Rheumatism*, 56(4) : 1204-11.
7. Ding C, Martel-Pelletier J, et al; Meniscal tear as an osteoarthritis risk factor in a largely non – osteoarthritic cohort; a cross-sectional study, *Journal of Rheumatology*, 34(4). 776-784.
8. Jordan JM, Helmick CG, et al, Prevalence of knee symptoms and radiographic and symptomatic knee osteoarthritis in African Americans and Caucasians: the Johnstone County osteoarthritis Project, *Journal of Rheumatology*, 34(1); 172-180.
9. Zeng QY, Zang C, et al; Associated risk factors of knee osteoarthritis: a population survey in Taiyuan, China, *Chinese Medical Journal*, 119(18):1522-7.
10. Rossignol M, Leclerc A, et al; Primary osteoarthritis of the hip, knee and hand in relation to occupational exposure; *Occupational & Environmental Medicine*, 62(11):772-7.
11. Margreth Grotle, Kare B Hagan et al; Obesity and Osteoarthritis in knee, hip, and/or hand. An epidemiological study in the general population with 18 year follow –up, BMC; Musculoskeletal disorders.

## Injury

1. Buckwalter j and Brown T, *Joint Injury, Repair and Remodeling: Roles in al; – traumatic Osteoarthritis*, CORR: Jun. 2004, pg 7-16, Symposium on Articular fractures.
2. Vrahas M, Mithoefer K et al, The Long term effects of articular impaction: CORR (423), 40-43, Jan. 2004.