

We are IntechOpen, the world's leading publisher of Open Access books Built by scientists, for scientists

4,800

Open access books available

122,000

International authors and editors

135M

Downloads

Our authors are among the

154

Countries delivered to

TOP 1%

most cited scientists

12.2%

Contributors from top 500 universities



WEB OF SCIENCE™

Selection of our books indexed in the Book Citation Index
in Web of Science™ Core Collection (BKCI)

Interested in publishing with us?
Contact book.department@intechopen.com

Numbers displayed above are based on latest data collected.

For more information visit www.intechopen.com



Osteoarthritis of the Hip Joint

Gerard A. Sheridan and James P. Cashman

Abstract

The incidence of osteoarthritis of the hip is increasing internationally. With the population becoming older and the rates of obesity increasing on a global scale, we are seeing more traumatic and idiopathic degeneration of the native hip joint. The pathological processes occurring in the hip have been described at a macroscopic and microscopic level. The inability of surface hyaline cartilage to heal is one of the major contributors to the irreversible nature of degeneration once it begins. Many classification systems have been described to characterise the extent of disease. History and examination play a pivotal role in the management algorithm. The goals of treatment are to improve pain, function and quality of life. Numerous non-operative treatments exist as do many operative interventions. Total hip arthroplasty is arguably the most successful operation developed in orthopaedic surgery to date. We discuss the condition of osteoarthritis as it pertains to the hip and we consider the patients' course from onset of symptoms through their investigation up to their definitive management.

Keywords: hip, osteoarthritis, osteotomy, total hip arthroplasty, revision total hip arthroplasty

1. Introduction

The hip joint is a ball and socket-type joint which is commonly affected by degenerative changes leading to osteoarthritis. Osteoarthritis of the hip is a non-inflammatory arthrosis caused by progressive loss of cartilage on the surface of the femoral head and the acetabulum. These two surfaces articulate normally with smooth lubricated motion. This allows painless weight-bearing through the normal hip joint and efficient mobilisation. When cartilaginous changes take place on the joint surface, degeneration occurs. This in turn leads to pain, restricted range of motion and limited function for those affected by the condition. These are the main clinical hallmarks found in osteoarthritis of the hip.

In the United States of America, the incidence of osteoarthritis is reported as 8 per 100,000 patients. Osteoarthritis of the hip is the main surgical indication for total hip arthroplasty [1]. Studies from the UK have demonstrated that osteoarthritis has an incidence of 9 in 1000 at-risk adults every year [2]. Yu et al. state that these figures are consistent across the international community. It is reasonable to take these figures as representative of the incidence of osteoarthritis in the developed world. Developing countries may demonstrate different incidences of osteoarthritis however. Unfortunately, robust data is not easily available for all countries in this area.

2. Aetiology

There are two commonly accepted aetiologies for osteoarthritis of the hip. These fall loosely under the headings of genetic causes and environmental causes.

2.1 Genetics

The genetic elements contributing to the condition have not yet been fully characterised. Pollard et al. assessed the risk of developing hip osteoarthritis in a population with a genetic predisposition. It was found that even when controlling for confounding variables, having a relative with hip osteoarthritis was associated with a significantly higher risk of developing the condition when compared to a population without any genetic predisposition [3]. Identification of a causative gene has yet to be confirmed. Defects in the *Col2 gene* (which codes for typ. 2 collagen, the main collagen type found in articular cartilage) may play a role in the development of hip osteoarthritis from a genetic perspective [4]. In 2015, Prof. A.J. Carr of Oxford was the senior author on the work entitled 'Osteoarthritis' which was published in The Lancet journal that year. The group references the arcOGEN consortium which had identified 11 genetic loci at the time associated with Osteoarthritis [5]. Carr also references the role of single nucleotide polymorphisms and how they may explain the genetic role in osteoarthritis by coding for BMI, bone mineral density and hip morphologies in the affected populations [5]. One can appreciate the significant role that genetics seems to play in this condition.

2.2 Environment

Environmental factors contributing to osteoarthritis of the hip are much better understood. The hip joint is a mechanical entity that relies on a number of key concepts for its functioning.

2.2.1 Lubrication

There are many types of lubrication described which will be discussed in detail later in this chapter. The hip is a synovial joint and fluid-film lubrication predominates in this type of joint [6]. The main purpose of lubrication is to reduce friction between two opposing surfaces in motion. Friction is described by the ***following equation [6]:

$$F = \mu_f \times W$$

In this equation, F = frictional force, μ_f is the coefficient of friction for a given material and W is the applied load. It follows that the lower the coefficient of friction for a surface bearing is, the less frictional load and wear that surface will undergo. When lubrication of a joint is insufficient to prevent friction, wear and degeneration occur. This is the mechanism by which obesity and heavy manual labour contribute to osteoarthritis in the hip.

2.2.2 Congruency

A congruent joint is one that has a uniform surface in contact with another uniform surface. Wear is defined as the progressive loss of a bearing substance (i.e. cartilage) as a result of chemical or mechanical action [6]. In the case of an incongruent joint, mechanical wear occurs at a much higher rate because the loss of

uniform surfaces in contact means that focal stresses are much higher and lubrication is much less effective. An example of this would be a femoral head fracture. This is a rare traumatic injury but rates of post-traumatic arthritis have been reported as high as 20% [7]. Acetabular fractures have been known to lead to contribute to post-traumatic arthritis of the hip. Magala et al. described the importance of joint congruency as it relates to the hip. It was found that patients with undisplaced fractures of the acetabulum had significantly better functional outcomes than those who sustained a displaced fracture of the acetabulum. This illustrates the importance of joint surface congruity, and how disturbance of this can lead to accelerated degeneration and poor functional performance of the hip joint [8]. Paediatric conditions such as Perthes disease, regular use of steroids and fractures to the neck of femur may all eventually result in avascular necrosis of the femoral head causing incongruity of the joint with resulting osteoarthritis of the hip as described. Avascular necrosis is the process of cell death secondary to vascular insufficiency. Bones with tenuous blood supplies are more predisposed to developing this condition. Any bone can sustain this injury but the commonest bones with a classically poor blood supply include the scaphoid, the talus and of course the femoral head. Subchondral bone loses its integrity leading to collapse, articular incongruity and rapid degenerative changes in the joint leading to significant functional limitations and pain in many cases.

2.2.3 Contact surface area

The hip joint is composed of a spherical head that rotates within a socket (acetabulum). The acetabulum covers the femoral head superiorly allowing forces to be transmitted up from the lower limbs to the pelvis and up through the spine during gait. The amount of force being transmitted depends on the mass of the body and the surface area of the hip joint.

'Stress' is defined as the 'force per unit area applied' and it is measured in N/m^2 [6]. For a given force (body mass) acting across the hip joint, the stress level at the joint surface will vary depending on the amount of acetabular and femoral head surface in contact. When the acetabulum covers a large amount of the femoral head, two large surface areas are in contact. This leads to lower contact stresses at the joint surface



Figure 1.
Centre-edge angle demonstrated on a paediatric pelvis.

with reduced wear and enhanced joint preservation. When the acetabular surface area is small (e.g. in developmental dysplasia of the hip), the contact stresses across the hip joint are very high leading to accelerated degeneration and wear rates. In developmental dysplasia of the hip (DDH), the hip joint does not form normally. This leads to a spectrum of disease from poor femoral head coverage to dislocation in utero. The centre-edge angle (CEA) is a measurement used to quantify the amount of coverage provided to the femoral head by the acetabulum. Hips with larger CEAs have higher surface areas in contact leading to less wear and degeneration. Terjesen looked at the effect of CEA on the development of hip osteoarthritis in a population with DDH. It was found that patients with a normal CEA (20° or above) had only a 5% risk of developing hip osteoarthritis. Patients with an abnormal CEA ($<20^\circ$) had a 22% chance of developing hip osteoarthritis. This demonstrates the significance of the contact surface area in the native hip and its role in the aetiology of hip degeneration (Figure 1).

3. Pathology

3.1 Cartilage constituents

Before describing the pathological processes that occur in the process or osteoarthritis we will first consider the normal constitution of articular cartilage. As with most connective tissues, articular cartilage consists of cells (chondrocytes) contained within an extracellular matrix. This extracellular matrix contains many elements as described by Ramachandran [6]:

- Fibres (collagen, elastin)
- Water (75% of wet weight)
- Proteoglycans
- Glycosaminoglycans
- Glycoproteins
- Matrix metalloproteinases
- Extracellular ions

The two major matrix components are the collagen fibres and the proteoglycans. Collagen mostly present in cartilage is type II. Three α -chains are arranged in a triple helix formation. This forms a collagen molecule which is then arranged in a quarter-staggered array. Collagen is essential for the integrity of the extracellular matrix of cartilage. Proteoglycans are molecules consisting of glycosaminoglycans (GAGs). Glycosaminoglycans such as keratan-sulphate and chondroitin-sulphate have a negative charge. This negative charge attracts cations and water which contributes to the osmotic pressure within cartilage and therefore the compressive strength of cartilage aggregate is bound by a sugar bond to a hyaluronic acid backbone to form the proteoglycan aggregate. These proteoglycan aggregates entwine with collagen fibres and chondrocytes to form the majority of the microstructure of articular cartilage [6].

Glycoproteins are macromolecules such as laminin and lubricin that are dispersed throughout the synovial fluid to act as a lubricant for the articulating joint surface. The role of lubricin was investigated by Galicia et al. [9]. It was postulated that pro-inflammatory markers were increased in an osteoarthritis population requiring total joint arthroplasty. When the arthroplasty group were compared to a control group, it was found that levels of IL-6, IL-8, VEGF, IL-1 β , MCP-1, EGF, and TNF- α were significantly increased [9]. These proinflammatory markers were raised in the arthroplasty group both preoperatively and postoperatively. Of note, compared to the controls, lubricin levels were decreased. The implication of these findings is that a traumatic event may induce a cascade of increased pro-inflammatory markers in osteoarthritic patients. This cascade seems to reduce the levels of lubricin circulating in the synovial fluid of the joint. This may explain one of the mechanisms responsible for post-traumatic arthritis development.

There are various enzymes present in the hip joint which can either destroy or preserve cartilage. Once these enzymes are balanced with a certain homeostasis, normal cartilage integrity will be preserved. If however the destructive enzymes are more prominent than the protective enzymes, there will be a net loss of cartilage tissue. There are two main enzymes responsible for cartilage degradation: aggrecanase and collagenase [4]. These two enzymes are known as matrix metalloproteinases (MMPs). Other MMPs include gelatinases, stromelysins, matrilysins and membrane-type MMPs [10]. Aggrecanase is responsible for the degradation of proteoglycans (e.g. aggrecan), an example being ADAMT. Another type of MMP is collagenase. Collagenase degrades collagen found in the substance of the articular cartilage of the hip. An example of this MMP would be MMP-13. IL-1 β is a substance found in the synovium which has a role in the activation of MMPs. It also activates nuclear factor $\kappa\beta$ (NF- $\kappa\beta$). If it were possible to reduce the activation of these MMPs by inhibiting IL-1 β , the downstream effect would be cartilaginous preservation in the hip joint. A study published in February 2018 by Zhang et al. investigated this effect [11]. This study was analysing the *in vitro* effect of isoliquiritigenin on primary cultured chondrocytes. By analysing mRNA and protein expression levels, inhibition of MMP expression by isoliquiritigenin was assessed. *In vitro* studies were also performed on mice articular cartilage. Final results confirmed a reduction in the expression of MMPs and a reduced activation of NF- $\kappa\beta$. They concluded that this pathway may be targeted in future to treat osteoarthritis of articular joints.

There are two enzymes responsible for inhibition of the MMPs. These are known as tissue inhibitors of matrix metalloproteinases (TIMPs) and plasminogen activator inhibitor-1 (PAI-1). The ratio of MMP to TIMP has a role in determining the net MMP activity, ECM turnover and tissue remodelling [10]. This is an antibiotic traditionally used to treat infections such as chlamydia. This has a broad systemic mechanism of action and therefore as many unwanted side effects. Liu et al. report that newer, highly targeted TIMP therapy may reduce the generic musculoskeletal side effects traditionally associated with TIMP therapy which may allow a more widespread uptake of the medication to treat osteoarthritis in the population [10].

3.2 Cartilage structure

Articular cartilage is arranged in a series of layers which all play a role in the diverse functions of the cartilage at different levels. The most superficial layer is the lamina splendens which contains long collagen fibres (mainly type II collagen) orientated parallel to the joint surface. This layer has flat chondrocytes, high concentrations of water and low concentrations of proteoglycans. This layer also has the

greatest tensile stiffness [4]. Collagen fibres and chondrocytes are oriented parallel to the surface to resist shear forces. Shear is the type of force generated when two opposing surfaces in contact move in opposite directions.

Longitudinal fibres at a perpendicular orientation to the joint surface would be poorly adapted to cope with shear stresses on the surface cartilage. As the depth of the articular cartilage increases, fibres and cells are oriented in a diagonal fashion. This essentially acts as a 'transition zone' to allow smooth progression from the superficial lamina splendens described above and the deeper layers of articular cartilage. In the deep radial zone, fibres are aligned to withstand compressive forces. This deep layer has a low water content and high proteoglycan content compared to the more superficial layers. Fibres and cells run perpendicular to the joint surface. This gives a mechanical advantage to the deep cartilage in compression. The arrangement of deep fibres in the radial zone allows the cartilage to withstand this compression.

Deep to the radial layer is the tidemark. The tidemark is composed of type X collagen. It demarcates the boundary between the flexible superficial cartilage and the deep calcified cartilage in the calcified zone [4]. Deep to the tidemark is the calcified zone of cartilage which then blends with subchondral bone completing the transition between cartilage superficially to bone in the deep layers.

Understanding the structure of cartilage is imperative to understanding how and why osteoarthritis develops the way it does. The lamina splendens as described is essential for the frictionless motion of one joint surface in contact with another. With loss of this layer, surface irregularities begin to manifest and loading across the joint surface becomes less uniform leading to focal areas of high loading with increased wear rates for the joint overall. Ramachandran describes the process of structural change in cartilage with the progression of osteoarthritis [6]. Firstly, collagen is disrupted, either through direct trauma or else via the MMP mechanism already described. Interference of this meshwork then allows proteoglycans to attract more water. This has an effect on the 'Young's modulus of elasticity' of the articular cartilage. This modulus (depicted by the symbol ' ϵ ') is a measure of the materials behaviour when a certain level of stress (or load) is applied to that material. For materials with a high Young's modulus, a high level of stress will cause a relatively low amount of strain (material displacement/movement) compared to materials with a lower modulus. This applies to the hip joint in the following way: with osteoarthritis, collagen is degraded, and water content increases due to more proteoglycan exposure. This increased amount of water lowers the Young's modulus meaning that when a load is placed through the joint, a higher level of strain or displacement will occur in the substance of the cartilage. Essentially the cartilage is now less stiff and so is more likely to deform and become damaged through the normal weight-bearing process. In this way, the macroscopic degeneration of cartilage is a synergistic process of degradation where one flaw in structural integrity increases the likelihood of developing a further flaw in the structure.

Based on the above discussion, Ramachandran proposes three main reasons for the observed effect of cartilage deformation [6]:

1. Collagen-proteoglycan matrix disruption
2. Large interstitial fluid movements causing loss of proteoglycan and decreased stiffness
3. Rapid repeated high loading leaving no time for stress relaxation and repair of collagen-proteoglycan matrix

Again, the key to understanding this vast limitation in this tissue can be found in the structure of the tissue. The layered configuration of cartilage has many implications in relation to its healing. In 1980, a classic study published by Mitchell et al. observed the essential role of the tidemark in cartilage healing. Using micron electroscopy and various staining techniques in rabbit models, they found that cartilaginous defects tended to heal due to the proliferation of a cell population emanating from the tide mark [12]. The tide mark marks the boundary between flexible superficial cartilage and deep stiff calcified cartilage. In 1993, Shapiro et al. described the morphological composition of tissue that fills the void of cartilaginous defects in full-thickness defects of articular cartilage [13]. Defects were made in the cartilage of New-Zealand white rabbits down to the tidemark. It was found that the tissue type to replace the deficient area was a type of cartilage known as fibrocartilage. If the cartilage sustains an injury more superficial than the tide mark, the defect will simply remain without any healing or regeneration. This is due in part to the relatively avascular nature of this part of the tissue. If then the femoral head of the hip were to sustain an injury of its cartilage that were deep enough to violate the tidemark, Shapiro has shown that regenerative tissue will form. It is very important to note however that this 'new cartilage' is fibrocartilage and not hyaline cartilage. Fibrocartilage has some differing features when compared with hyaline cartilage. This consists mostly of typ. 1 collagen, unlike hyaline cartilage which consists of typ. 2 collagen. Importantly, fibrocartilage is not designed for weight bearing like hyaline cartilage is since it has a higher coefficient of friction when compared to hyaline cartilage. In this way, articular cartilage does have the capacity to regenerate and heal defects that fulfil a certain set of criteria. This regeneration however is not optimal for the function intended in articular joints, and so once the articular cartilage is injured, it is fair to say that it will never be normal again.

In the 1970s, Maroudas and Venn published their work describing the physiology of cartilage as we know it today. The classic picture of increased water content and reduced glycosaminoglycans was detailed in this seminal work [14].

4. History

When considering the symptoms associated with osteoarthritis of the hip, pain and function are the two biggest contributors to the natural history. A precise description of the pain is essential to obtain in the history. Usually, patients will describe an aching type of pain in the groin. There may be contributing areas such as the greater trochanter and buttock but the groin for the most part is the site of complaint. This pain usually has an insidious onset. The traditional description of osteoarthritic pain is one that is less severe in the morning and with rest. The traditional teaching is that exercise and progression through the day towards evening time will be associated with deterioration of pain and symptoms throughout the day. It is well established that significant weight gain may be associated with significant deterioration in the patients reported symptoms. In early stages of osteoarthritis, reversal of this effect through weight loss can be seen in a number of cases. The reason for this association is simply related to the load passing through the hip joint, as the overall body mass increases, the force per unit area (N/m^2) or 'stress' passing through the hip joint is significantly increased. Subjective pain is also significantly affected by the patients' general psychological status. It is well described that patients suffering from depression and other disorders are poorly adapted to cope with pain and may experience subjectively higher levels of pain

when compared to a patient that does not have depression but does have the same level of osteoarthritis on radiographic examination.

It is important to consider a number of factors in relation to pain. In 1891, in his series of lectures relating to “rest and pain”, Hilton described a very important fundamental principle in orthopaedics [15]. Hilton’s law states that “the nerve supplying a joint also supplies the muscles that traverse the joint and the skin sensation over that joint”. It follows that when a nerve traverses more than one joint, pain actually originating in the knee for example may manifest as pain in the hip and vice versa. In this way, hip osteoarthritis may actually present as pain in the knee or the lumbar spine. Therefore, a good rule of thumb in orthopaedic practice is to always examine the joint above and below the area of complaint.

The effect of pain on the patient’s life is essential to characterise. If the pain is deteriorating, it is important to confirm over what time frame and whether there are any specific exacerbating activity. Often, avoidance of the activity leading to the pain is enough to reduce symptoms to a level acceptable to the patient. In this way, lifestyle modification and activity limitation can play a role in the early conservative management of early osteoarthritis. It is important for patients to stay active however. Muscle deconditioning around the hip and weight gain in general are associated with poorer hip function and deterioration in the symptom profile of the patient. Jeanmaire et al. described the effect of low lean mass on the quality of life and function of patients with osteoarthritis of the hip [16]. They concluded that having less lean mass (i.e. a deconditioned hip with poor strength) is associated with poorer quality of life and poorer function in this cohort. This emphasises the importance of strengthening exercises and remaining active in this cohort. This can be a very difficult cohort of patients to treat, especially because of the known association with high BMI rates and infection of implanted total hip replacements. At a mean follow-up of 3 years, Pulos et al. described a higher rate of total hip replacement revision for infection. This was seen if the patients BMI was over 35 [17]. This illustrates the complex relationship between pain profiles, patient BMI and surgical infection as experienced by many orthopaedic hip surgeons.

Other factors to consider in the history are past medical and past surgical history. Rondon et al. assessed the complications of performing total hip and knee replacements in patients with Parkinson’s disease. It was shown that the risk of periprosthetic fracture and dislocation were significantly higher in this patient cohort when compared to patients without Parkinson’s disease [18]. In the patient with neurological dysfunction, proprioceptive awareness and motor control are commonly lacking. In the initial postoperative phase, stringent limitations are placed on the patient regarding acceptable positions of the hip to prevent the risk of dislocation in both the acute and chronic settings. If the patient is unable to adhere to these instructions, they are at much higher risk of dislocation and chronic instability. Instability episodes may also lead to falls and fractures which are very significant injuries in this frail cohort of patient.

5. Examination

Examination is an essential part in the management algorithm of patients with hip osteoarthritis. Patients should be examined under the following headings.

5.1 Inspection

Much information can be ascertained through inspection alone. Scars, swellings, muscle wasting (particularly in the gluteal and quadriceps region), asymmetry and deformity are essential to comment on in the examination of the hip.

Sagittal alignment should reveal the presence of any fixed flexion deformity in the hip. Patients will often compensate for this malalignment by flexing the knee and plantarflexing the ankle to maintain foot contact with the floor. A hyperlordosis of the lumbar spine may mask the severity of a fixed flexion contracture. This can be objectively evaluated using Thomas' test. This will be discussed in turn.

5.2 Gait

Gait analysis will often show an antalgic gait. This is when the patient demonstrates a shortened stance phase on the side of the affected hip due to pain on weight bearing. Other abnormal patterns of gait include Trendelenburg gait. This occurs due to weakness or painful inhibition of the hip abductors during gait. During single leg stance on the affected side, the planted foot acts as a fixed support for the body. To clear the contralateral leg during its swing phase, the abductors contract thereby lifting the contralateral pelvis and allowing foot clearance.

Another type of gait that occurs is the fused hip gait. Typically, these patients have no terminal stance and they may present with an exaggerated lumbar lordosis also.

Limb length discrepancy manifests either through circumduction of the long leg, ankle plantarflexion of the short leg or hip vaulting of the long leg through hip flexion. Patients may become very good at compensating for a leg length discrepancy, so the clinical suspicion should be high for this abnormality in the preoperative setting. Coleman blocks should be used to evaluate the degree of clinical discrepancy as perceived by the patient.

5.3 Limb length discrepancy (LLD)

It has frequently been described after total hip replacement that limb length discrepancy remains a very significant complication. It has often been quoted as the main reason for patient dissatisfaction and is also noted to be the commonest reason for litigation against orthopaedic surgeons in the postoperative period [19]. For this reason, it is imperative to identify the presence of any preoperative limb length discrepancy. This can be allowed for in the surgical technique utilised by the surgeon. Regardless of the surgical technique, the most important point is to notice it in the preoperative setting.

There are many ways of assessing the clinical limb length discrepancy. The use of Coleman blocks has already been described and these are very useful tools to have available in the out-patient setting. Firstly, the true and apparent limb lengths should be attained. The true limb length is measured as the distance from the anterior superior iliac spine (ASIS) on the pelvis down to the medial malleolus. The apparent limb length discrepancy includes the adaptive mechanisms that the patient has developed and gives an indication to the LLD that the patient feels subjectively. The apparent LLD does not give a measure of the true length discrepancy in the lower limbs. If it is confirmed that there is indeed a true LLD, the next step is to characterise where exactly in the lower limb this is coming from: the tibia, the femur or the hip.

The Galeazzi test is used to identify where the discrepancy may be originating from. To perform the test, the patient is laid supine, hips are flexed to 45° and the knees are flexed to 90°. The ankles are brought together at the level of the medial malleoli and the knees are then observed. On lateral inspection, if the right patella is lower and more distal to the left, it is likely that the shortening is coming from the right tibia. If the right patella is lower and more proximal to the left, it is likely that the right femur is shorter than the left. If the femur is the suspected source of shortening, one must proceed to perform the digital Bryant's test. Again, the patient

is still supine. A line is drawn directly vertical down from both ASIS's. The tip of the GT is palpated bilaterally, and a line is drawn perpendicular to the line drawn from the ASIS. This horizontal line passes from the tip of the GT on both sides and ends once it intersects with the vertical line passing vertically down from the ASIS. If there is a discrepancy in the length of these two lines, one can assume that the source of femoral shortening is supratrochanteric. If these two lines are equal bilaterally, one can assume that the source of femoral shortening is below the level of the trochanters.

5.4 Palpation and motion

Finally, then, one should ask the patient to identify the source of pain. Classically the patient will point to the groin region. Assure the patient that you will try to avoid causing them pain during the examination, but this is not always possible. Begin palpating away from the source of pain initially and then migrate towards the site of pain then.

After joint palpation, motion should then be assessed. Firstly, the presence of contractures should be documented. Thomas' test has been described to eliminate any compensatory lumbar lordosis developed by the patient [20]. In a patient with a fixed flexion deformity (FFD) of the hip, hyperlordosis of the lumbar spine often occurs to improve the overall hip extension and to allow improved gait patterns. To perform the test, place your flattened hand behind the patient's lumbar spine and ask them to flatten their back. This eliminated the lumbar curvature. Then place your other hand behind the patient's ipsilateral knee, ask them to extend the knee and compress the popliteal fossa against your hand. Patients with an FFD will be unable to perform this and the angle subtended by the bed and the posterior aspect of the flexed femur is the fixed flexion angle of the hip joint.

Before assessing active and passive ranges of motion in the hip joint, the hip must be squared to expose any coronal contractures of the hips and allow a more accurate comparison of the ranges in both hips. Firstly, ask the patient to show their active range of motion (ROM). An initial straight leg raise will show the strength of the hip and potentially reveal a contributing spinal aetiology to the pain if Lasègue's test is positive. Document the hip flexion, abduction and adduction with the pelvis squared. With the hip and knee flexed to 90°, document the range of internal and external rotation of the hip. A very common finding is impingement indicated by pain at the ends of the rotational range of motion. Stinchfield's test may be performed. The hip is flexed to 30° with the knee in extension. The patient is asked to flex the femur up against resistance. Pain induced by this examination implies an intraarticular source to the pain.

Place the patient in the lateral position then and assess abductor strength. Hip extension is also easily assessed in this position. Ober's test is used to identify ITB tightness as described above. To perform Ober's test, position the patient laterally and flex the lower hip to eliminate the lumbar lordosis. Then flex the upper knee to 90° while abducting and extending the hip. In patients with a tight ITB, the hip will remain passively abducted and will not adduct as the lateral structures of the thigh are either too painful or too tight to allow passive adduction [20]. Piriformis test can be performed in this position by flexing the knee to 90° and the hip to 60°. Downward pressure on the painful leg reproduces pain.

Next, place the patient prone. Assess gluteal bulk and hip extension again in this position. The lumbar spine and sacroiliac joints are easily palpated in this position. Prone is the best position to assess the version of the femur. Craig's test is used to assess the proximal femur version [20]. Flex the knee to 90° and hold the ankle in one hand. Internally and externally rotate the hip joint whilst palpating the greater

trochanter (GT). When the trochanter feels most prominent, this is when the femoral neck is parallel to the floor. If the GT is most prominent with 15° of internal rotation of the hip, this means that the femoral neck has an anteversion angle of about 15°. If the GT is most prominent in 23° of external rotation of the hip, this means that the femoral neck is 23° retroverted.

Finally, one must never forget to examine the knee and lumbar spine when performing the hip examination.

6. Investigations

6.1 Radiography

Investigations used in hip osteoarthritis are dominated by the simple plain radiograph. An adequate X-ray of the pelvis will allow characterisation of the disease extent and even detailed preoperative planning in the vast majority of cases. There are 4 radiographic findings classically described when describing osteoarthritis of the hip joint:

1. Loss of joint space
2. Osteophyte formation
3. Subchondral sclerosis
4. Subchondral cysts

This can be represented simply by the mnemonic 'LOSS'. Loss of cartilage through the pathological mechanisms already discussed leads to an approximation of the acetabular and femoral bone on plain radiograph. With disease progression, the bony ends appear to be in direct contact due to the complete destruction of all articular cartilage. Cartilage is not ossified in the normal hip and so it is radiolucent giving the appearance of an apparent 'joint space'. Loss of cartilage therefore gives rise to a loss of this joint space.

Osteophytes are the metaplastic osseous and cartilaginous tissues found at the rim of articular surfaces of joints that experience subtle instability. They may play a number of roles including protection of articular cartilage and redistribution of stresses borne by the hip joint [21]. Interestingly, Tsurumoto et al. described the relationship between the severity of stress experienced by an osteoarthritic joint and the size of osteophyte. It was demonstrated that joints subjected to higher stresses were likely to develop larger osteophytes [22]. In this way, osteophytes may act as a surrogate marker for the severity of degeneration in the hip. Due to microscopic and macroscopic changes in the structural integrity of cartilage, areas of weakening develop. These are known as subchondral cysts. Areas of reactive sclerosis develop as a generic response to injury. This accounts for the common radiographic finding of subchondral cysts and subchondral sclerosis observed in severe cases of hip osteoarthritis (**Figure 2**).

Many classification systems have been developed to try and create an accurate way of describing radiographic findings. There are numerous classifications used to describe osteoarthritis throughout the years. In 1963, Kellgren described 4 grades of osteoarthritis based on the progressive observation of osteophytes, sclerosis, joint space narrowing, femoral head deformity and cyst formation [23]. In 1990, Croft et al. also described a classification system based on the progressive appearance of

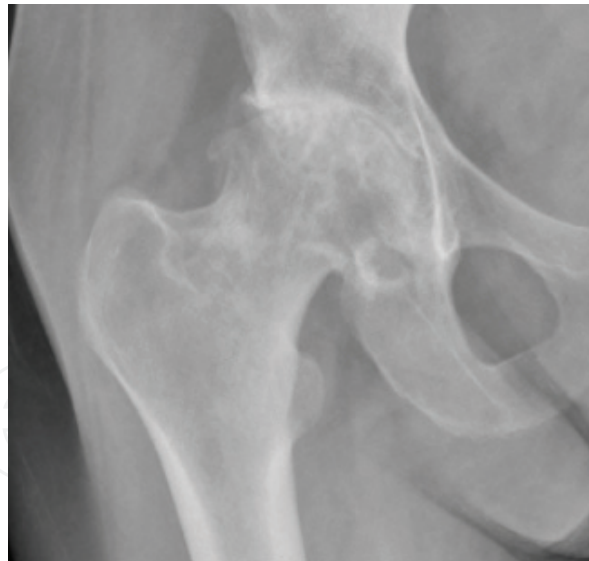


Figure 2.

Hip radiograph illustrating loss of joint space, femoral and acetabular osteophytes, femoral head subchondral sclerosis and acetabular subchondral cysts.

similar factors described by Kellgren. In 2005, Jacobsen et al. described a third classification system for osteoarthritis of the hip. This system was different to the preceding two in that it included some level of accurate measurement. The Kellgren and Croft systems are clearly open to extensive inter-observer variability given the use of vague, non-specific terms to describe the stages of a condition. Jacobsen et al. looked at a specific measurement defined as the joint space width (JSW). Three measurements are taken between the weight-bearing surface of the femoral head and the surface of the acetabulum. If any of these three measurements are below 2 mm, this is defined as osteoarthritis. In 2012, Terjesen et al. performed an evaluation of the above three classification systems. They found that the JSW <2 mm system gave the highest rate of interobserver reliability. It was also the simplest system and so was felt to be the most useful classification system for assessment of osteoarthritis of the hip.

Magnetic resonance imaging (MRI) can play a role in the evaluation of osteoarthritis of the hip. It is mostly used to assess soft tissue pathology in and around the hip joint. There is more of a role for MRI in research. Delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) can characterise the very subtle features of early osteoarthritis and so it is often used in the clinical research setting. Again, this is not a commonplace modality in the standard investigation of osteoarthritis.

7. Non-operative management

The aim of all treatment for osteoarthritis of the hip is to relieve pain and to improve function. Characterising and simplifying the patients presenting complaint will guide the decision and make it clear as to what management path to take. Red flags to be mindful of in the history include night time pain that wakes the patient from their sleep, progressively reducing walking distance due to pain and functional limitation to a level that is not acceptable to the patient. A very comprehensive examination will help to rule out contributing factors from sources besides the hip as described already. We will discuss the full spectrum of hip osteoarthritis management and outline the indications and concerns associated with each management path.

7.1 Non-pharmacological

Non-operative intervention is either pharmacological or non-pharmacological. Non-pharmacological methods include quadriceps strengthening, hip ROM exercises, manual therapy, gait assistance and gait aids. A walking stick will often help when held in the hand opposite to the symptomatic side of hip osteoarthritis. In terms of free-body diagrams, the stick reduces the moment of the body weight acting around the painful hip joint by providing a counter-moment in the opposite direction. The abductors need to work less, and the overall joint force reaction is reduced. Carrying a heavy item in the ipsilateral hand can have the same effect on the free-body diagram by assisting the abductor force and thereby reducing the work performed by the abductor mechanism.

Sharma recently analysed the effect of non-pharmacological and non-operative intervention in hip osteoarthritis [24]. It was found that pharmacological treatment for osteoarthritis is lacking and needs to be optimised as soon as possible in the clinical setting. With time to total hip replacement (THR) as the outcome measure, Svege et al. reported their findings of a long-term randomised trial. Patients were randomised either to education about their condition with appropriate non-operative interventions or education supplemented with exercise therapy. Exercise therapy and education were associated with a longer time to THR implying the beneficial role of exercise in treating this condition [25]. Anecdote would seem to suggest however that once a hip is significantly painful with degeneration confirmed on radiograph, then non-operative measures are unlikely to ever really succeed. Bennell et al. performed a well designed prospective double-blinded randomised control trial to assess the role for physical therapy in the management of hip osteoarthritis [26]. 102 patients with significantly painful osteoarthritis of the hip were included in the study. Forty-nine patients were in the active group. They underwent education, manual therapy, home exercise and gait aid as appropriate. The remaining 53 patients underwent a sham intervention consisting of a self-applied gel three times a week. The intervention lasted for 12 weeks in total and pain and functional scores were assessed for both groups. The use of physical therapy did not show any significant improvement in the pain and functional outcomes of that patient cohort. In conclusion, the non-pharmacological modalities are highly effective in treating osteoarthritis currently. Sharma mentions the need however, to reconcile acceptable physical activity levels with osteoarthritis progression in the future for better understanding of the condition.

7.2 Pharmacological

Pharmacological analgesic control of hip osteoarthritis is important as it often improves painful symptoms to a baseline that is tolerable to the patient thereby allowing them to function. This may achieve the two aims of management in hip osteoarthritis: namely pain control and restoration of function. The 'World Health Organisation' introduced a document entitled 'cancer pain relief' in 1986 [27]. This was a document aimed at introducing a graded system for the controlled introduction of opioids into a patient's analgesic regimen. This was specifically targeted at cancer patients originally but has been adapted as a good approach to managing pain in the majority of painful conditions. The ladder has three steps as follows (**Figure 3**):

Step 1		Non-opioid	Adjuvant
Step 2	Weak opioid	Non-opioid	Adjuvant
Step 3	Strong opioid	Non-opioid	Adjuvant

Figure 3.
WHO analgesic ladder.

Non-opioids consist of medications such as paracetamol and aspirin. These are drugs with low side effect profiles which is why they are the first step on the ladder. Once the non-opioid medications have been exhausted, adjuvant non-opioid medications should be added in step 1. COX-2 inhibitors include medications such as celecoxib. The advantage of selective COX-2 inhibition is the reduction in the unwanted gastric side effects. Gastric inhibition of COX results in reduced PGE₂ and PGI₂. Reduction of these prostaglandins in the stomach reduces blood flow, increases acid production and results in dyspepsia, nausea and gastritis [6]. With the use of agents like celecoxib, constant usage instead of intermittent usage has been associated with significantly less episodes of painful flares [28]. Celecoxib appears to be the commonest disease-modifying analgesic prescribed in this cohort with function primarily being through PG and cytokine levels in the joint [29]. Topical NSAIDs may have some role also. The British National Institute for Clinical Excellence (NICE guidelines) released in 2014, recommend the use of topical NSAIDs before the use of oral NSAIDs.

After the pain has exceeded the control of step 1. Step 2 in the ladder should be commenced. Step 2 sees the introduction of weak opioids such as codeine, dihydrocodeine and tramadol. Opioid analgesics act on mu (μ) receptors in the spinal cord and brain. Receptors are located mostly in the dorsal horn of the spinal cord and the thalamus. Strong opioids found on the third step of the ladder include the likes of morphine, fentanyl and oxycodone. Again, these agents have a more significant inhibitory effect on pain but the risk of side effects may outweigh treatment in many cases.

Once all oral options have been exhausted, intraarticular injections of corticosteroids should be considered. Most of the national and international guidelines available for hip osteoarthritis will recommend intraarticular steroid injection as a good option before surgery to temporise the operation and in some cases avoid operation. McCabe et al. reviewed five studies assessing the effect of intraarticular steroid injections for the hip [30]. They found a significant reduction in pain levels at 8 weeks post-injection. In 2017, Chambers et al. published their work assessing the effect of intraarticular steroid injections of the hip. The study included 456 patients in total. 106 patients received 2 or more injections and then underwent total hip replacement. A matched cohort of 350 patients received only 1 injection and then underwent total hip replacement. Postoperative prosthetic infection rates were reported in both groups. The 'single injection' group had a significantly lower infection rate at 2%. Those receiving 2 or more injections had an infection rate of 6.6% [31]. Perhaps a reasonable approach to this issue would be to offer multiple steroid injections to patients who will likely never have an operation-either due to comorbidity or volition. If one suspects that a patient will likely undergo a total hip replacement in the future, then it is reasonable to offer a single injection only and then consider operation.

8. Operative management

8.1 Non-arthroplasty techniques

We have discussed the role of hip pathology in the young adult and how both intraarticular and extraarticular deformities may contribute to early onset osteoarthritis of the hip. Hip arthroscopy is a practice that is becoming increasingly used to treat predisposing conditions for arthritis and indeed treat arthritic patients also. We know that the presence of labral tears leads to

chondral damage and therefore the development of hip osteoarthritis [32]. Hip arthroscopy has a role in the treatment of labral tears, focal chondral lesions and even ligamentum teres tears. Byrd et al. described a beneficial role of arthroscopy in patients with the above findings in the setting of DDH and mechanical abnormality. Questions are often asked posed about the role hip arthroscopy plays in patients with established hip osteoarthritis. Kemp et al. performed a systematic review assessing 22 studies [33]. They looked at pain and functional improvements in patients undergoing hip arthroscopy. Patients were divided into two groups: those with osteoarthritis and those without. Findings suggested that hip arthroscopy does improve function and pain in patients with pre-existing osteoarthritis. Their improvement was not as marked as the non-degenerative patients. Predictors of conversion to THR included patient age and the severity of chondral damage.

Other non-arthroplasty techniques include proximal femoral and acetabular osteotomies. We will consider the commonest osteotomy used for the treatment of dysplasia in the young adult—the Bernese (or Ganz) periacetabular osteotomy. In 1988, Ganz described his original Bernese periacetabular osteotomy [34]. The goal of the surgery was to realign the acetabular orientation to improve joint congruency, increase joint surface contact area, reduce high focal stresses and ultimately preserve the hip joint in the young adult for as long as possible. The technique describes an anterior (Smith-Petersen) approach to the hip joint. Three cuts are made in the pelvis as follows: superior pubic ramus cut (complete), supraacetabular cut (complete and extraarticular), ischial cut (incomplete). Nine parameters were described by Clohisy et al. that should be checked in the operating room before finishing the operation [35]:

1. Surface (weight-bearing acetabulum) should be more horizontal with an inclination of 0–10°
2. Lateral femoral head coverage should be improved with an angle of 25 to 35°
3. Medial aspect of the femoral head should be within 5 to 10 mm of the ilioischial line (this may require medialisation of the femoral head depending on the position of the individual case)
4. Acetabular version should be correct (one can assess a retroverted acetabulum by observing the anterior and posterior acetabular wall lines. If retroversion has occurred, the classic “crossover sign” will be evident on imaging)
5. Anterior femoral head coverage should be improved to 20–25° on the false-profile view of the proximal femur (a false profile view of the femur is a lateral view with roughly 25° internal rotation of the whole body on that side. This will give a true lateral view of the femoral head as it is situated in the acetabulum. Only on this intraoperative view can the anterior femoral head coverage be commented on)
6. The correction produces a congruent joint
7. Adequate head–neck offset is present or has been produced with osteochondroplasty
8. Adequate internal fixation has been achieved with acceptable screw position

9. Hip flexion of at least 90° and hip abduction of at least 30° can be achieved on table before the end of the operation

This is a significant operation for the patient to undergo and it is not without complication. Patients undergoing PAO (periacetabular osteotomy) tend to be young with relatively few comorbidities. For this reason, the operation is usually very well tolerated, and patients return to function soon after the procedure.

8.2 Total hip arthroplasty

According to the American Academy of Orthopaedic Surgeons (AAOS), total hip arthroplasty (THA) is one of the most successful procedures in all of medicine [36]. Over 300,000 THAs are performed yearly in the U.S. Hip osteoarthritis and total hip arthroplasty play a very prominent role in the burden of orthopaedic procedures performed each year worldwide. With the population of the planet ageing at the rate it is, this demand will only increase. Kurtz et al. projected this increased demand in their 2007 paper. It was estimated that by 2030, the demand for primary total hip arthroplasty will rise by 174% to 572,000. Demand for revision THA is expected to double by the year 2026 [37]. These figures confirm that total hip arthroplasty is an essential operation and will only be increasing in the future.

The NICE guidelines published in 2014 suggest appropriate referral requirements for potential hip replacement candidates. Taken directly from the document entitled 'Osteoarthritis: Care and Management', we consider a few recommendations from the section entitled 'Referral for consideration of joint surgery' [38]:

- 1.6.3 Consider referral for joint surgery for people with osteoarthritis who experience joint symptoms (pain, stiffness and reduced function) that have a substantial impact on their quality of life and are refractory to non-surgical treatment. [2008, amended 2014]
- 1.6.4 Refer for consideration of joint surgery before there is prolonged and established functional limitation and severe pain. [2008, amended 2014]
- 1.6.5 Patient-specific factors (including age, sex, smoking, obesity and comorbidities) should not be barriers to referral for joint surgery. [2008, amended 2014]

In 1979, Sir John Charnley, a British Orthopaedic surgeon published his seminal work "Low friction arthroplasty of the hip". In his writings he explained the technique of the total hip arthroplasty. At the time, Charnley was aware of the concepts of friction and how it was important to reduce wear in the implants. He designed a component known as the Charnley stem. This was a monoblock device, meaning it had no modularity or changeable parts. The head size was 22.225 mm in diameter and the bearing surface used for the acetabular replacement was Teflon (polytetrafluoroethylene). Both the femoral and acetabular components were fixed with cement that secured the prostheses in bone. Unfortunately, and understandably with the development of a new technology, there were some issues with the original design of this implant. In the following 40 years, the total hip arthroplasty has evolved significantly in several areas that we will discuss here.

8.2.1 Approach

There are many surgical approaches to hip joint were described. Traditionally, the most common were the anterolateral approach and the posterior approach.

Disadvantages of the anterolateral approach included compromise of the abductor mechanism. Gluteus medius and minimus are traversed in this approach. Although they are repaired afterward, there is often a notable limp or 'Trendelenburg gait' due to abductor weakness. The advantage of this approach is the relative stability it ensures. Historically, the anterolateral approach was associated with a lower risk of dislocation when compared to the posterior approach to the hip. In 1982, the postoperative dislocation rate of the posterior approach was reported as significantly higher when compared to the anterolateral approach. The dislocation rate was reported as 2.3% through the anterolateral approach and 5.8% through the posterior approach [39]. Techniques have advanced since then and the posterior approach has been optimised. Historically, the posterior elements were not always repaired with meticulous technique. In an attempt to reduce the dislocation rate through the posterior approach, posterior soft tissue repair of the capsule and short external rotators has improved the postoperative stability levels to such a degree that surgical approach no longer plays a role in postoperative surgical dislocation rates [40].

8.2.2 Fixation methods

There are two methods of securing the femoral and acetabular components. They may be fixed with cement or with an uncemented technique. Discussion continues regarding the ideal combination of cement and uncemented techniques on both the acetabula side and femoral side of the THA. Options now include fully cemented, fully uncemented, hybrid (cemented stem and uncemented cup) and reverse hybrid (uncemented stem and cemented cup).

Bone cement consists of polymethylmethacrylate. This is a polymer that comes as a liquid (containing the monomer N,N-dimethyltoluidine and hydroquinone) and a powder (consisting of PMMA copolymer, barium dioxide for radiopacification and benzoyl peroxide for polymerisation initiation). These 2 substances are mixed, and an exothermic chemical reaction ensues. Cement is inserted at around 2–4 minutes and is completely hard at 10–12 minutes. This allows some finesse of the implant position up to a certain point, but beyond that if the final position is suboptimal, all the cement must be removed, which is a significant undertaking in a primary THA.

There are some concerns with the use of cement however. Bone cement implantation syndrome is characterised by hypotension, hypoxemia, cardiac arrhythmias and cardiac arrest or a combination of any of these [41]. In their study, Ereth et al. assessed this phenomenon prospectively in 35 patients undergoing cemented and uncemented THA with transoesophageal echocardiography and invasive haemodynamic monitoring. Findings confirmed that the use of cement in THA increased the risk of embolisation, reduced cardiac output, increased pulmonary artery pressure and increased pulmonary vascular resistance [41]. This syndrome has also been associated with sudden intraoperative death. The pathology behind this serious complication involves dissemination of bone marrow debris and amorphous cement particles into the circulation which eventually locate in the pulmonary vasculature causing the above described effect [42]. Cemented procedures take a few minutes longer while waiting for the cement to set. Uncemented stems also work on a principle of preserving bone stock whereas cemented stems often remove more cancellous bone stock than their uncemented counter parts. This is important in revision surgery where inadequate bone stock may dictate the usage of a more complex implant and procedure to attain adequate fixation.

Uncemented femoral stems and acetabular components function through a completely different mechanism. By reaming the acetabulum to a certain diameter or broaching the femur to a certain size and then inserting a cup or femoral



Figure 4.
Uncemented femoral prosthesis.

component that is slightly larger in diameter or size, one can achieve a “press-fit” (**Figure 4**). This provides immediate mechanical stability until biological fixation occurs. Uncemented prosthesis has a porous coating which allows either ingrowth or ongrowth of the native bone. Hydroxyapatite coatings allow growth of bone into metal which provides the fixation in the long term. There is a vogue for using these stems in the younger population as it is necessary to have a reasonable bone stock. There are many reports conferring improved survival of uncemented stems in the younger populations [43]. The risks of bone loss are reportedly higher in the cemented stems and aseptic loosening has also been reported as higher in the cemented stems [44, 45]. There is an increased rate of usage of uncemented stems in modern day practice [46]. Uncemented stems are not without complication however. Many studies have shown that intraoperative periprosthetic fracture rate is higher with the uncemented stem prostheses [34, 47]. Added to this, registry data from around the world has often reported an improved all-cause revision rate in cemented stems over uncemented stems [48, 49]. For this reason, there is no consensus on which stem type is better. The likelihood is that there is a role for both stem types, uncemented in a younger cohort with good bone stock and cemented for a more elderly population with poor bone quality.

8.2.3 Bearing surfaces

The traditional bearing surfaces consisted of a metal femoral head (usually cobalt chrome) and a polyethylene acetabular cup (ultra-high molecular weight polyethylene—UHMWPE). This is a very commonly used combination today. Other surface bearings include ceramic on polyethylene, ceramic on ceramic and metal on metal. Metal on metal bearings have been associated with high failure rates [50]. They have been associated with high levels of adverse reactions to metal debris (ARMD). These local reactions lead to the formation of painful pseudotumours and pain with a difficult revision procedure to correct. High systemic levels of circulating cobalt and chromium may pose a serious health risk to patients. For this reason these implants have fallen out of favour. Ceramic on ceramic bearings have the

lowest coefficient of friction and produce the least amount of wear particles which is desirable to reduce the incidence of aseptic loosening. Unfortunately, because these bearings are very rigid, cases of femoral head fracture and squeak have been reported [51].

Ceramic on polyethylene bearings appear to be the most favourable when considering revision rates controlled for bearing surfaces. The New Zealand registry data from 2017 supports this claim also [52]. It is reasonable to conclude that either a ceramic on polyethylene or a metal on polyethylene bearing should be used in modern day total hip arthroplasty. Metal femoral heads are much cheaper than the ceramic options and so arguments for their usage are still valid.

8.3 Revision total hip arthroplasty

The demand for total hip arthroplasty revision will increase significantly in the near future [37]. Large collections of data known as registries now exist and allow analysis on a large scale of the reasons for failure of THA. The National Joint Registry (NJR) is the UK which has the largest collection of THA data in the world every year. According to their 2017 figures, the commonest reasons for revision of 85,199 total hip replacement, in order of decreasing frequency are as follows [53]:

1. Aseptic loosening (41,077)
2. Pain (17,231)
3. Lysis (13,194)
4. Implant wear (11,808)
5. Dislocation/subluxation (11,172)
6. Periprosthetic fracture (8079)
7. Infection (7832)
8. Adverse reaction to metal debris (7095)
9. Malalignment (4448)
10. Implant fracture (2862)
11. Head-socket size mismatch (628)
12. Other (6399)

In order to deal with the above complications, we must improve our technology continually. Developments in the polyethylene have produced new highly-crosslinked polyethylene (XLPE) and vitamin-E treated polyethylene. XLPE has been associated with lower revision rates for aseptic loosening [54]. Vitamin E is an antioxidant which has been shown to reduce wear rates also in the polyethylene [55]. Dislocation rates may be improved through the use of larger femoral heads, restoring length and offset and meticulous surgical repair of the anatomical exposure, regardless of the approach [56]. Periprosthetic fractures are going to rise in the

future populations also. General bone health in the elderly population and safe mobilisation will reduce the rates seen. Regarding infection, Parvizi has carried out extensive research in the field. The first definition of prosthetic joint infection (2011) was only described in 2011 [57]. Currently, the 2014 modified accepted definition of PJI is as follows [58]:

- A. *There is a sinus tract communicating with the prosthesis, OR*
- B. *A phenotypically identical pathogen is isolated by culture from 2 or more separate tissue or fluid samples obtained from the affected prosthetic joint, OR*
- C. *When three of the following five criteria exist:*
 - i. *Elevated serum erythrocyte sedimentation rate AND serum C-reactive protein concentration*
 - ii. *Elevated synovial white blood cell count, OR ++ change on leukocyte esterase test strip*
 - iii. *Elevated synovial polymorphonuclear percentage*
 - iv. *Positive histological analysis of periprosthetic tissue*
 - v. *A single positive culture*

The gold standard treatment for PJI is a 2-stage revision procedure. This involves removal of all infected tissue and insertion of an antibiotic-impregnated spacer (**Figure 5**). This remains in place until the infection has completely cleared. Usually at around 3 months, the second stage procedure is performed. Recurrence rates with this 2-stage approach are much lower when compared with a single stage revision for infection [59]. Clearly, there are many improvements that must be made to reduce the rate of revision THA surgery. This will be an ongoing effort in the future.



Figure 5.
Antibiotic impregnated spacer with antibiotic beads in the soft tissues.

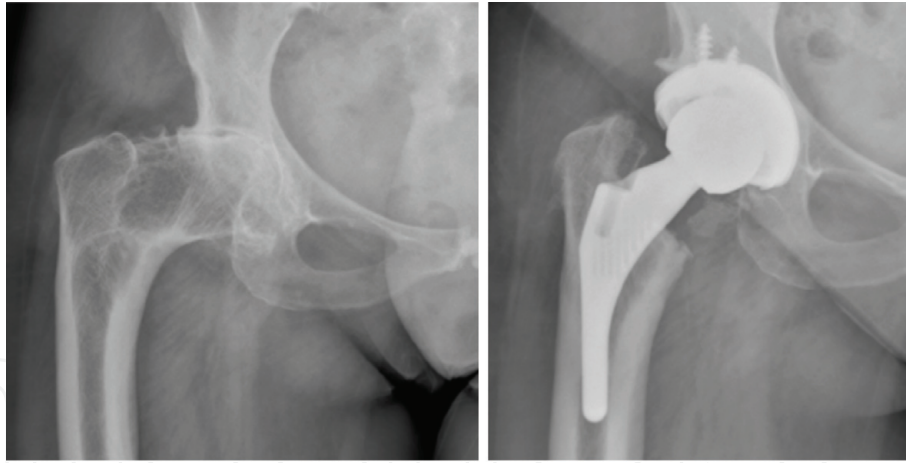


Figure 6.
CAD CAM prosthesis illustrating good fixation in the presence of a highly deformed proximal femur.

8.4 Salvage

Historically, hip arthrodesis and excision hip arthroplasty with complete excision of the femoral head were used to treat end-stage hip osteoarthritis. Hip arthrodesis is rarely indicated anymore due to the success of modern implants. Excision arthroplasty however, does have a role. It may be particularly useful as a salvage procedure in patients with intractable infection. Mobility and pain may be significantly improved through this procedure. Specialised custom-made prostheses which are computer-assisted design and computer assisted manufacture (CAD CAM) have a very niche role in patients with very abnormal hip morphology that cannot be accounted for by standard prostheses (**Figure 6**).

9. Conclusion


Osteoarthritis of the hip is a highly prevalent condition that will be more common in future generations due to the relative increase in the population. As always, history and examination supplemented by good radiographic techniques will guide further management. Total hip arthroplasty is one of the great medical success stories throughout history. There is still room to refine our techniques and this will be the focus of technological advance in the future.

Author details

Gerard A. Sheridan* and James P. Cashman
Cappagh National Orthopaedic Hospital, Dublin, Ireland

*Address all correspondence to: sheridga@tcd.ie

IntechOpen

© 2018 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] Varacallo M, Varacallo M, Johanson NA. Hip, Replacement. Treasure Island (FL): Stat Pearls; 2018
- [2] Yu D, Peat G, Bedson J, Jordan KP. Annual consultation incidence of osteoarthritis estimated from population-based health care data in England. *Rheumatology (Oxford, England)*. 2015;**54**(11):2051-2060
- [3] Pollard TC, Batra RN, Judge A, Watkins B, McNally EG, Gill HS, et al. Genetic predisposition to the presence and 5-year clinical progression of hip osteoarthritis. *Osteoarthritis and Cartilage*. 2012;**20**(5):368-375
- [4] Miller MD, Thompson. Miller's Review of Orthopaedics. 7th ed. Amsterdam: Elsevier; 2016
- [5] Glyn-Jones S, Palmer AJ, Agricola R, Price AJ, Vincent TL, Weinans H, et al. Osteoarthritis. *Lancet*. 2015;**386**(9991):376-387
- [6] Ramachandran M. Basic Orthopaedic Sciences: The Stanmore Guide. Boca Raton: CRC Press; 2006
- [7] Giannoudis PV, Kontakis G, Christoforakis Z, Akula M, Tosounidis T, Koutras C. Management, complications and clinical results of femoral head fractures. *Injury*. 2009;**40**(12):1245-1251
- [8] Magala M, Popelka V, Bozik M, Heger T, Zamborsky V, Simko P. Conservative treatment of acetabular fractures: Epidemiology and medium-term clinical and radiological results. *Acta Chirurgiae Orthopaedicae et Traumatologiae Cechoslovaca*. 2015;**82**(1):51-60
- [9] Galicia K, Thorson C, Banos A, Rondina M, Hopkinson W, Hoppensteadt D, et al. Inflammatory biomarker profiling in Total joint arthroplasty and its relevance to circulating levels of Lubricin, a novel proteoglycan. *Clinical and Applied Thrombosis/Hemostasis*. 2018;**24**(6):950-959. DOI: 10.1177/1076029618765090. Epub 2018 Apr 22
- [10] Liu J, Khalil RA. Matrix metalloproteinase inhibitors as investigational and therapeutic tools in unrestrained tissue remodeling and pathological disorders. *Progress in Molecular Biology and Translational Science*. 2017;**148**:355-420
- [11] Zhang L, Ma S, Su H, Cheng J. Isoliquiritigenin inhibits IL-1beta-induced production of matrix metalloproteinase in articular chondrocytes. *Molecular Therapy—Methods & Clinical Development*. 2018;**9**:153-159
- [12] Mitchell N, Shepard N. Healing of articular cartilage in intra-articular fractures in rabbits. *The Journal of Bone and Joint Surgery American Volume*. 1980;**62**(4):628-634
- [13] Shapiro F, Koide S, Glimcher MJ. Cell origin and differentiation in the repair of full-thickness defects of articular cartilage. *The Journal of Bone and Joint Surgery American Volume*. 1993;**75**(4):532-553
- [14] Venn M, Maroudas A. Chemical composition and swelling of normal and osteoarthrotic femoral head cartilage. I. Chemical composition. *Annals of the Rheumatic Diseases*. 1977;**36**(2):121-129
- [15] Hilton J. Rest and Pain: A Course of Lectures on the Influence of Mechanical and Physiological Rest in the Treatment of Accidents and Surgical Diseases, and the Diagnostic Value of Pain. London: Bell and Daldy; 1891
- [16] Jeanmaire C, Mazieres B, Verrouil E, Bernard L, Guillemin F, Rat AC. Body

composition and clinical symptoms in patients with hip or knee osteoarthritis: Results from the KHOALA cohort. *Seminars in Arthritis and Rheumatism*. 2018;**47**(6):797-804

[17] Pulos NMM, Courtney PM, Lee GC. Revision THA in obese patients is associated with high re-operation rates at short-term follow-up. *The Journal of Arthroplasty*. 2014;**29**(9):209-213

[18] Rondon AJ, Tan TL, Schlitt PK, Greenky MR, Phillips JL, Purtill JJ. Total joint arthroplasty in patients with Parkinson's disease: Survivorship, outcomes, and reasons for failure. *The Journal of Arthroplasty*. 2018;**33**(4): 1028-1032

[19] El Bitar YF, Stone JC, Jackson TJ, Lindner D, Stake CE, Domb BG. Leg-length discrepancy after total hip arthroplasty: Comparison of robot-assisted posterior, fluoroscopy-guided anterior, and conventional posterior approaches. *American Journal of Orthopedics (Belle Mead, N.J.)*. 2015; **44**(6):265-269

[20] Sharma H. FRCS (Tr&Orth) Part II Examination: How to Get Slick in the Clinicals: FRCSOrthExam Education; 2008.

[21] Mao Y, Yu D, Xu C, Liu F, Li H, Zhu Z. The fate of osteophytes in the superolateral region of the acetabulum after total hip arthroplasty. *The Journal of Arthroplasty*. 2014;**29**(12):2262-2266

[22] Tsurumoto TSK, Okamoto K, Imamura T, Maeda J, Manabe Y, Wakebe T. Periarticular osteophytes as an appendicular joint stress marker (JSM): Analysis in a contemporary Japanese skeletal collection. *PLoS One*. 2013;**8**(2):e57049

[23] Terjesen T, Gunderson RB. Radiographic evaluation of osteoarthritis of the hip: An inter-observer study of 61 hips treated

for late-detected developmental hip dislocation. *Acta Orthopaedica*. 2012; **83**(2):185-189

[24] Sharma L. Osteoarthritis year in review 2015: Clinical. *Osteoarthritis and Cartilage*. 2016;**24**(1):36-48

[25] Svege I, Nordsletten L, Fernandes L, Risberg MA. Exercise therapy may postpone total hip replacement surgery in patients with hip osteoarthritis: A long-term follow-up of a randomised trial. *Annals of the Rheumatic Diseases*. 2015;**74**:164-169

[26] Bennell KL, Egerton T, Martin J, Abbott JH, Metcalf B, McManus F, et al. Effect of physical therapy on pain and function in patients with hip osteoarthritis: A randomized clinical trial. *Journal of the American Medical Association*. 2014;**311**(19):1987-1997

[27] Organization WH. Cancer pain relief: With a guide to opioid availability. 1986

[28] Strand V, Simon LS, Dougados M, Sands GH, Bhadra P, Breazna A, et al. Treatment of osteoarthritis with continuous versus intermittent celecoxib. *The Journal of Rheumatology*. 2011;**38**(12):2625-2634

[29] Nakata K, Hanai T, Take Y, Osada T, Tsuchiya T, Shima D, et al. Disease-modifying effects of COX-2 selective inhibitors and non-selective NSAIDs in osteoarthritis: A systematic review. *Osteoarthritis and Cartilage*. 2018;**26**(10):1263-1273. DOI: 10.1016/j.joca.2018.05.021. Epub 2018 Jun 8

[30] McCabe PS, Maricar N, Parkes MJ, Felson DT, O'Neill TW. The efficacy of intra-articular steroids in hip osteoarthritis: A systematic review. *Osteoarthritis and Cartilage*. 2016; **24**(9):1509-1517

[31] Chambers AW, Lacy KW, Liow MHL, Manalo JPM, Freiberg AA, Kwon

- YM. Multiple hip intra-articular steroid injections increase risk of periprosthetic joint infection compared with single injections. *The Journal of Arthroplasty*. 2017;**32**(6):1980-1983
- [32] Keeney JA, Peelle MW, Jackson J, Rubin D, Maloney WJ, Clohisy JC. Magnetic resonance arthrography versus arthroscopy in the evaluation of articular hip pathology. *Clinical Orthopaedics and Related Research*. 2004;**429**:163-169
- [33] Kemp JL, MacDonald D, Collins NJ, Hatton AL, Crossley KM. Hip arthroscopy in the setting of hip osteoarthritis: Systematic review of outcomes and progression to hip arthroplasty. *Clinical Orthopaedics and Related Research*. 2015;**473**(3):1055-1073
- [34] Aaboud M, Aad G, Abbott B, Abdinov O, Abeloos B, Abidi SH, et al. Search for dark matter produced in association with a Higgs boson decaying to $b\bar{b}$ using 36 fb^{-1} of pp collisions at $\sqrt{s}=13 \text{ TeV}$ with the ATLAS detector. *Physical Review Letters*. 2017;**119**(18):181804
- [35] Clohisy JC, Beaulé PE, O'Malley A, Safran MR, Schoenecker P. AOA symposium. Hip disease in the young adult: Current concepts of etiology and surgical treatment. *The Journal of Bone and Joint Surgery American Volume*. 2008;**90**(10):2267-2281
- [36] Surgeons AAoO. 2015. Available from: <https://orthoinfo.aaos.org/en/treatment/total-hip-replacement/>
- [37] Kurtz S, Ong K, Lau E, Mowat F, Halpern M. Projections of primary and revision hip and knee arthroplasty in the United States from 2005 to 2030. *The Journal of Bone and Joint Surgery American Volume*. 2007;**89**(4):780-785
- [38] nice.org.uk. Osteoarthritis: Care and management clinical guideline [CG177] February 2014
- [39] Woo RY, Morrey BF. Dislocations after total hip arthroplasty. *The Journal of Bone and Joint Surgery American Volume*. 1982;**64**(9):1295-1306
- [40] Jameson SS, Baker PN, Mason J, Gregg PJ, Brewster N, Deehan DJ, et al. The design of the acetabular component and size of the femoral head influence the risk of revision following 34 721 single-brand cemented hip replacements: A retrospective cohort study of medium-term data from a National Joint Registry. *The Journal of Bone and Joint Surgery British Volume*. 2012;**94**(12):1611-1617
- [41] Ereth MHW, Abel MD, Lennon RL, Lewallen DG, Ilstrup DM, Rehder K. Cemented versus noncemented total hip arthroplasty—embolism, hemodynamics, and intrapulmonary shunting. *Mayo Clinic Proceedings*. 1992;**67**(11):1066-1074
- [42] Razuin R, Effat O, Shahidan MN, Shama DV, Miswan MF. Bone cement implantation syndrome. *The Malaysian Journal of Pathology*. 2013;**35**(1):87-90
- [43] Wechter J, Comfort TK, Tatman P, Mehle S, Gioe TJ. Improved survival of uncemented versus cemented femoral stems in patients aged < 70 years in a community total joint registry. *Clinical Orthopaedics and Related Research*. 2013;**471**(11):3588-3595
- [44] Dan D, Germann D, Burki H, Hausner P, Kappeler U, Meyer RP, et al. Bone loss after total hip arthroplasty. *Rheumatology International*. 2006;**26**(9):792-798
- [45] Eskelinen A, Remes V, Helenius I, Pulkkinen P, Nevalainen J, Paavolainen P. Total hip arthroplasty for primary osteoarthritis in younger patients in the Finnish arthroplasty register. 4,661 primary replacements followed for 0-22 years. *Acta Orthopaedica*. 2005;**76**(1):28-41

- [46] Lehil MS, Bozic KJ. Trends in total hip arthroplasty implant utilization in the United States. *The Journal of Arthroplasty*. 2014;**29**(10):1915-1918
- [47] Lindberg-Larsen M, Jorgensen CC, Solgaard S, Kjersgaard AG, Kehlet H, Lundbeck Foundation Centre for Fast-Track H, et al. Increased risk of intraoperative and early postoperative periprosthetic femoral fracture with uncemented stems. *Acta Orthopaedica*. 2017;**88**(4):390-394
- [48] Hughes RE, Batra A, Hallstrom BR. Arthroplasty registries around the world: Valuable sources of hip implant revision risk data. *Current Reviews in Musculoskeletal Medicine*. 2017;**10**(2): 240-252
- [49] Hailer NP, Garellick G, Karrholm J. Uncemented and cemented primary total hip arthroplasty in the Swedish hip arthroplasty register. *Acta Orthopaedica*. 2010;**81**(1):34-41
- [50] Maurer-Ertl W, Friesenbichler J, Holzer LA, Leitner L, Ogris K, Maier M, et al. Recall of the ASR XL head and hip resurfacing systems. *Orthopedics*. 2017;**40**(2):e340-e3e7
- [51] Pomeroy E, Rowan F, Masterson E. Atraumatic fracture of a BIOLOX Delta ceramic femoral head articulating with a polyethylene liner: A case report. *JBJS Case Connector*. 2015;**5**(4):e112
- [52] Sharplin PWM, Rothwell A, Frampton C, Hooper G. Which is the best bearing surface for primary total hip replacement? A New Zealand joint registry study. *Hip International*. 2018;**28**(4):352-362. DOI: 10.5301/hipint.5000585. Epub 2017 Jan 12
- [53] Reito A, Lehtovirta L, Lainiala O, Makela K, Eskelinen A. Lack of evidence-the anti-stepwise introduction of metal-on-metal hip replacements. *Acta Orthopaedica*. 2017;**88**(5):478-483
- [54] Callary SA, Solomon LB, Holubowycz OT, Campbell DG, Munn Z, Howie DW. Wear of highly crosslinked polyethylene acetabular components. *Acta Orthopaedica*. 2015;**86**(2):159-168
- [55] van der Veen HC, van den Akker-Scheek I, Bulstra SK, van Raay JJ. Wear, bone density, functional outcome and survival in vitamin E-incorporated polyethylene cups in reversed hybrid total hip arthroplasty: Design of a randomized controlled trial. *BMC Musculoskeletal Disorders*. 2012;**13**:178
- [56] Rowan FE, Benjamin B, Pietrak JR, Haddad FS. Prevention of dislocation after total hip arthroplasty. *The Journal of Arthroplasty*. 2018;**33**(5):1316-1324. DOI: 10.1016/j.arth.2018.01.047. Epub 2018 Mar 7
- [57] Parvizi J, Zmistowski B, Berbari EF, Bauer TW, Springer BD, Della Valle CJ, et al. New definition for periprosthetic joint infection: From the workgroup of the musculoskeletal infection society. *Clinical Orthopaedics and Related Research*. 2011;**469**(11):2992-2994
- [58] Proceedings of the international consensus meeting on periprosthetic joint infection. Foreword. *Journal of Orthopaedic Research*. 2014;**32** (Suppl. 1):S2-S3
- [59] Aggarwal VK, Rasouli MR, Parvizi J. Periprosthetic joint infection: Current concept. *Indian Journal of Orthopaedics*. 2013;**47**(1):10-17