# Osteoarthritis

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Musculoskeletal pain caused by arthritis is one of the most common reasons why people seek medical attention.[1] Arthritis commonly manifests as musculoskeletal pain and inflammation, and patients with arthritis can have either monoarticular or polyarticular disease. The spectrum of arthritis includes more than 100 conditions. Osteoarthritis (OA) is the most common form of arthritis in the United States, affecting more than 20 million Americans. OA commonly affects the knees, hips, spine, and fingers. Other joints less frequently affected include the wrists, shoulders, and ankles; OA in these joints is usually a result of trauma or repetitive stress to the joint that exceeds the loading of the articular cartilage.[2] OA is associated with age, with the majority of persons showing radiographic evidence of OA by age 65 years, although most are asymptomatic.[1] With the aging of the population, a large number of people are approaching the age range in which OA is more prevalent. These patients often expect to maintain a high level of function and an active lifestyle. The physician is challenged to keep patients with arthritis active by utilizing both nonsurgical and surgical options. This article briefly reviews the pathogenesis and diagnosis of OA and provides a more specific review of the treatment modalities available for patients with OA.

## PATHOGENESIS

Arthritis is the end result of a variety of disorders that lead to the structural or functional failure of one or more joints. Diarthroidal joints consist of bone, cartilage, and connective tissue. The subchondral bone within the diarthroidal joint is covered by hyaline cartilage, which contains type II collagen, chondrocytes, and proteoglycans. The arrangement of type II collagen along the joint surface provides tensile strength to the subchondral bone, while proteoglycans within the joint's matrix surface assist with water retention. Together type II collagen and proteoglycan serve as a low-friction surface and act as a shock absorber for the joint surface. Normal joint fluid contributes to the health of the surface cartilage by providing nourishment and decreasing joint loading via its viscoelastic properties. Joint fluid volume often increases in an arthritic joint; however, the composition of the fluid is abnormal, with higher levels of prostaglandins, collagenases, tumor necrosis factor (TNF), and interleukin-1 (IL-1) and relatively low levels of hyaluronic acid.

OA is primarily a disease of the load-bearing cartilage, in which repetitive microtrauma is believed to cause subtle biochemical and biomechanical alterations of the cartilage matrix that lead to breakdown of both cartilage and subchondral bone, initiating an inflammatory response. The degradation of cartilage in OA occurs in several phases.[3] At the biochemical level, there is an increase in the water content within the type II cartilage and initially an increase in proteoglycan synthesis as a mechanism for the joint to repair itself. Ultimately, the repair process begins to slow, and a cycle characterized by decreased proteoglycan synthesis and subsequent decreased cartilaginous load-bearing capacity ensues. Chondrocytes within the cartilage matrix gradually become overwhelmed while attempting to repair the cartilage damage. In response to the increased stress, chondrocytes release metalloproteinases (collagenase, gelatinase, stromelysin, and lysosomal proteases), causing further destruction of the collagen matrix and further thinning of the cartilage matrix. As the cartilage matrix thins, more subchondral bone is exposed. Load-bearing is then increased across the subchondral bone, precipitating a compensatory increase in subchondral sclerosis, which is demonstrated in late-phase radiographic studies of the OA joint by the presence of osteophytes.

Throughout the inflammatory process, the cartilage attempts to repair itself, the bone remodels, the underlying subchondral bone hardens, and bony cysts form within the joint. This in turn leads to further impaired joint mechanics and symptoms of pain and stiffness.[3]

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Evidence suggests that some patients with OA may have a point mutation in genes controlling the synthesis of minor collagen types in cartilage, such as in type IX or X.[4] However, it is accepted that the majority of the osteoarthritic process begins as a result of excessive or repetitive loading of normal joint tissues.[4]

## DIAGNOSIS

Diagnosing end-stage osteoarthritic disease in an elderly patient is usually straightforward with appropriate radiographs and physical examination. However, diagnosing arthritic disease in a patient with minimal or no radiographic changes may be more difficult because many of the signs and symptoms of OA are common to many different diseases (eg, inflammatory arthritides, autoimmune disorders, myofascial syndromes). The diagnostic evaluation should include a review of the patient's medical history, physical examination findings, current medications, gait, laboratory test results, and appropriate radiographs or other imaging tests.[2,4]

### Signs and Symptoms

Arthritis pain is usually described as a dull ache that is occasionally sharp with sudden movements. Mild to severe swelling is associated with this pain, and some patients may experience stiffness. Pain associated with knee OA is usually located over the joint line and in the parapatellar area. The medial compartment of the knee is most susceptible to age-related wear and tear and therefore is an area where knee OA is commonly seen.[5] Patellar compartment arthrosis usually presents with pain that is worse with prolonged sitting in a chair or car or with climbing inclines or stairs. Arthritic pain is usually relieved with rest, ice, compression wrapping, and oral or intra-articular anti-inflammatory medications.

### Radiographic Evaluation

An appropriate radiographic evaluation is needed to determine the extent and severity of OA in a joint. Hyaline cartilage of the joint is not seen on routine radiographs; therefore, weight-bearing knee radiographs are needed to assess the amount of joint space narrowing under physiologic loads. At least 2 views (eg, anteroposterior, lateral) are needed to fully appreciate the area of interest, as abnormalities can potentially be hidden by superposition.[6] Osteophyte formation and the presence of subchondral cysts are often seen as the disease becomes more advanced. Computed tomography scanning can also help in select instances when radiographic films are difficult to evaluate or are inconclusive because of retained hardware, congenital abnormalities, or severe malalignment.[7]

### Laboratory Evaluation

Laboratory evaluation is critical for the diagnosis and differentiation of the many types of arthritis; laboratory testing commonly includes measurement of the erythrocyte sedimentation rate and rheumatoid factor titer, a leukocyte count, and evaluation of synovial fluid aspirate. An erythrocyte sedimentation rate less than 40 mm/h, rheumatoid factor titer less than 1:40, and a clear, viscous synovial fluid with a leukocyte count less than $20 \times 10^3/mm^3$ suggests a diagnosis of noninflammatory arthritis. Elevations in the erythrocyte sedimentation rate and C-reactive protein may indicate inflammatory or infectious etiologies for joint pain and swelling and can often exclude OA.

### Diagnostic Criteria

A method for diagnosing OA of the knee originally proposed by Altman et al[5] is based on the presence of knee pain plus at least 3 of the following clinical features: (1) age 50 years or older, (2) more than 30 minutes of morning stiffness, (3) joint crepitus, (4) joint tenderness, (5) bony enlargement, and (6) no palpable joint warmth.[5,8] The sensitivity and specificity for OA of these criteria alone are 95% and 69%, respectively. However, when results of laboratory findings are applied, the sensitivity and specificity for OA changes to 92% and 75%, respectively (**Table**).

Similar findings and complaints as described above may be seen during examination of other joints. Although not part of an accepted system, pain with activity, stiffness, mild warmth, and crepitus are some cardinal examination signs for arthritis in any joint.

## NONPHARMACOLOGIC MANAGEMENT

Physicians should be familiar with the various treatment options for OA that are safe, effective, and noninvasive. Continual damage to the joint's cartilage surface along with the joint's impaired ability to repair this damage can cause significant disability in patients with OA. The key to nonoperative management of arthritis is based on reducing damage to joint surfaces. There are multiple modalities that physicians can recommend to their patients to help relieve joint pain and improve joint mechanics. Primarily, patients need encouragement and self-management tools to help them achieve healthier joints.

The first step that patients can take is to reduce their weight. However, weight reduction is often a difficult task for older patients who have other medical conditions that impair their ability to exercise. Referrals to nutritionists and dietitians are underutilized and can provide much needed help to those with lifelong

poor eating habits. Reducing body weight by as little as 20 lb may relieve many patients of their arthritic complaints.[9]

Patients also should be encouraged to maintain an active lifestyle with exercises specific for arthritic patients. Consultation with a physiotherapist may help the patient learn simple, reproducible exercises in a controlled setting. Low-impact exercise (eg, swimming, walking on flat surfaces) is beneficial to keep joint fluid healthy and can help reduce joint pain associated with arthritis. Proper footwear also can reduce reactive forces to both the knee and hips while walking and performing activities of daily living. It is recommended that shoes have an adequate shock-absorbing heel and sole and a stabilizing heel cup to prevent excessive foot motion.

If the above modifications have not provided satisfactory results for patients with knee OA, a knee brace can provide stability to reduce joint motion as well as increase the warmth of the joint to help with pain relief.[10] A brace may shift the weight away from the diseased compartment of the joint to the unaffected compartment. An elastic support around the knee may enhance stabilization, especially for unicompartmental medial or lateral OA. Valgus bracing (also known as an off-loader brace) is often used for varus knees (bow legged), in which OA primarily involves the medial compartment. Less frequently, varus bracing may be indicated for lateral compartment OA, in which the knees are primarily in valgus (knock-knee) alignment.[11]

## PHARMACOLOGIC MANAGEMENT

Pain relief is the primary indication for the use of pharmacologic agents in patients with OA who do not respond to nonpharmacologic interventions (eg, weight loss, exercise). Medications can be applied directly to the joint itself, taken orally (over-the-counter or prescription strength), or administered as an injection.

### Acetaminophen and Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

Over-the-counter oral pain medications, such as regular strength acetaminophen and ibuprofen, are also effective for relief of minor OA pain. Acetaminophen (up to 4 g/d) is the drug of choice for pain relief in mild to moderate OA. Adverse reactions associated with acetaminophen include hepatotoxicity and renal damage, which is primarily seen in patients who concurrently consume excessive amounts of acetaminophen (> 4 g/d) in addition to large amounts of alcohol.

An NSAID may be indicated in patients with moderate OA who fail to respond to acetaminophen. NSAIDs are thought to decrease the amount of prostaglandin $E_2$ in human OA cartilage, thereby affecting joint inflammation. Studies suggest that prostaglandin $E_2$ may contribute to local inflammation within the joint space; therefore, decreasing the amount of prostaglandin $E_2$ may lead to less cartilage damage and pain. To achieve an anti-inflammatory response, an ibuprofen dose of 2400 mg/d is recommended; to achieve an analgesic effect, the recommended dose is 1200 mg/d. A randomized, double-blind trial of 184 patients with knee OA compared the efficacy of ibuprofen 2400 mg/d with acetaminophen 4000 mg/d after 4 weeks of treatment and demonstrated equivalent improvement in all major outcome variables, including pain and disability scales.[3] The disability scales used included the Stanford Health Assessment Questionnaire, which assessed time needed to walk 50 ft and the physician's global assessment of the patient's arthritis.[3]

**Table.** Methods for Diagnosing Osteoarthritis

| |
|---|
| **Clinical criteria*** |
| Age 50 years or older |
| More than 30 minutes of morning stiffness |
| Joint crepitus |
| Joint tenderness |
| Bony enlargement within the joint |
| No palpable joint warmth |
| **Laboratory values†** |
| Erythrocyte sedimentation rate less than 40 mm/h |
| Rheumatoid factor negative |
| Leukocyte count less than $20 \times 10^3/mm^3$ |
| Clear synovial fluid |

Adapted from Altman R, Asch E, Bloch D, et al. Development of criteria for the classification and reporting of osteoarthritis. Classification of osteoarthritis of the knee. Diagnostic and Therapeutic Criteria Committee of the American Rheumatism Association. Arthritis Rheum 1986;29:1039–49. Reprinted with permission of Wiley, Liss, Inc., a subsidiary of John Wiley & Sons, Inc.

*Sensitivity and specificity for diagnosing osteoarthritis are 95% and 69%, respectively, when applying clinical criteria only.

†Sensitivity and specificity for diagnosing osteoarthritis are 92% and 72%, respectively, when applying clinical criteria plus laboratory values.

### Cyclooxygenase-2 (COX-2) Inhibitors

Prescription anti-inflammatory drugs known as COX-2 inhibitors have been shown to benefit patients affected by moderate to severe OA with concomitant gastric disorders.[12] Selective COX-2 inhibitors have received approval from the US Food and Drug Administration (FDA). Recently, however, the FDA has

questioned the safety of these drugs, citing their potential for increasing the risk of cardiac events. Although celecoxib is still considered safe, rofecoxib and valdecoxib have been removed from the market by their manufacturers. These drugs have at least a 200- to 300-fold selectivity for inhibition of COX-2 over COX-1. Clinical data demonstrate that selected COX-2 inhibitors produced comparable analgesia and anti-inflammatory effects to nonselective NSAIDs but with fewer gastric and duodenal ulcers detected by serial endoscopy.[12] However, recent evidence has shown increased cardiac adverse effects associated with COX-2 inhibitors.[13,14]

### Oral Supplementation

Chondroprotective agents, such as oral glucosaminoglycan polysulfate and chondroitin sulfate, have also been shown to protect joint cartilage from further degradation and to stimulate cartilage production.[15] The proposed mechanism of action of oral glycosaminoglycan polysulfate is inhibition of the degradative process within the cartilaginous matrix, which in turn, may slow cartilage breakdown.[6,15,16] A controlled human study showed a modest decrease in pain symptoms due to OA and a decrease in joint space narrowing over time in patients administered 1500 mg of glucosamine daily compared with placebo.[16] However, a 2003 meta-analysis of long-term, double-blind, randomized trials evaluating the safety and efficacy of glucosamine and chondroitin in patients with knee OA concluded that further studies are needed to determine the ideal time, dose, patient baseline characteristics, and structural efficacy for both glucosamine and chondroitin.[17]

Antioxidants and minerals, including vitamins C, D, and E, have been shown to be beneficial in the proper formation of cartilage. One study comparing patients with OA taking vitamin C 152 mg/d found a threefold reduction in risk of OA progression compared with patients not taking vitamin C.[18] Data are still pending for comparison results with vitamin E (400 IU/d), vitamin D, boron, and selenium.

### Topical Agents

Topical medications to relieve OA pain can be used after a careful review of the patient's medical history and allergies. Some recommended over-the-counter medications for treatment of minor OA are Aspercreme and capsaicin. Aspercreme, a 10% trolamine salicylate cream, can be massaged into the affected areas 3 to 4 times a day. The cream is absorbed percutaneously and is hydrolyzed to salicylic acid. In a pH-dependent process, the salicylic acid is absorbed into the synovial fluid and slowly converted to salicyluric acid and other metabolites; it is excreted in the urine. Application of topical capsaicin to the joint affects the A delta and C fibers within the joint and secondarily depletes them of substance P and blocks intraneuronal axonal transport and synthesis of substance P, which leads to a decreased perception of pain.[8,19]

### Intra-articular Injections

Injectable forms of anti-inflammatory medication combine a corticosteroid plus a local anesthetic. Injections of methylprednisolone and triamcinolone acetonide cause less local postinjection flare and soft tissue atrophy and reduce the chance of tendon rupture compared with other long-acting oral agents.[20] Standard doses of triamcinolone acetonide at 40 mg (1 mL) can be used for large joints such as knees and hips. The corticosteroid is often mixed with an equivalent volume of 1% lidocaine to reduce postinjection flare caused by crystallization of the intra-articular steroid and to provide more immediate relief of pain from the injection.[20] The number of injections that can be administered ranges from 4 injections per lifetime in joints with primary OA to 1 injection per month in joints severely affected by rheumatoid arthritis.[2] Systemic side effects from local injections may include allergic reactions to the steroid or local anesthetic or elevated blood glucose levels in diabetics. Frequent steroid injections over the long term carry the risk of local fat atrophy and cartilage softening due to decreased collagen formation.[21]

### Viscosupplementation

Injectable chondroprotective agents, known as viscosupplementation agents, are also available for patients with symptomatic knee OA. Viscosupplementation involves a series of intra-articular injections of hyaluronic acid, a natural chemical in synovial fluid that decreases friction between joint surfaces. Hyaluronic acid injections should be considered in patients with symptomatic OA who have not responded adequately to standard nonpharmacologic and pharmacologic treatments or are intolerant of these therapies secondary to gastrointestinal disorders.[22] One proposed mechanism of action of hyaluronate is that it protects against further proteoglycan depletion caused by free radicals by acting as a free radical scavenger.[12] On the other hand, some data show that viscosupplementation is no more efficacious that cortisone injections.[23]

There are 2 viscosupplementation agents currenly available: natural hyaluronan (Hyalgan, Sanofi-Synthelabo, NY) and synthetic hylan G-F 20 (Synvisc,

Genzyme, Cambridge, MA). The difference between these agents is based on the molecular weight of the cross-linked hyaluronic acids. Synthetic hylan G-F 20 is more efficacious than hyaluronic acid because it has a higher molecular weight and enhanced elastoviscous properties and resides longer in the joint space. A 12-week, randomized trial comparing hylan G-F 20 with hyaluronan in 70 patients demonstrated that patients who received hylan G-F 20 had better results on all outcome measures compared with hyaluronan. Based on this trial, hylan G-F 20 may have better clinical efficacy, but studies are ongoing.[24]

Clinical experience and studies of natural hyaluronan and synthetic hylan G-F 20 are inconclusive but seem to indicate beneficial effects with minimal adverse reactions in a significant number of patients. Several studies have reported that both hyaluronate derivatives have been found to be superior to placebo and corticosteroid injections in pain relief of osteoarthritic knees, but the evidence is limited due to short follow-up time and high dropout rate.[25,26] Studies have also shown that injectable hyaluronic acid is approximately equivalent in efficacy to an oral NSAID; however, the injections tend to produce an analgesic effect that may last months.[27,28]

The recommended injection schedule is 1 injection per week for 5 weeks for hyaluronan and 1 injection per week for 3 weeks for hylan. There are no specific postinjection instructions for either drug (ie, activity as tolerated). Repeat courses of viscosupplementation can be performed after 6 months. Complications of viscosupplementation include iatrogenic joint infection, postinjection inflammation, and allergic reactions with the second hyaluronate injection. The cost of hyaluronic acid is significant: the average wholesale price for 5 vials of Hyalgan is US $661 ($132.20 per vial), and a package of 3 prefilled syringes of Synvisc costs US $620 on average. Third-party reimbursement is variable, but Medicare and most insurance companies now cover viscosupplementation.

### Other Agents

A number of experimental oral medications and injectable agents for the treatment of OA may become available in the future. Antibiotic medications (eg, tetracycline) have been shown to have a variety of anti-inflammatory effects by inactivation of matrix metalloproteinases. This group of proteolytic enzymes, which includes collagenases, stromelysins, and gelatinases, degrades all components of articular extracellular matrix and can cause destruction of articular cartilage.[29] Two proteins that inhibit the effects of metalloproteinases have been identified; these tissue inhibitors of metalloproteinase (TIMP-1 and TIMP-2) block the proteolytic activity of matrix metalloproteinases.[30] Other injectable agents currently being researched range from vectors that express specific genes to delivery systems for recombinant protein drugs. Interests have focused on genes that lead to the local production of biologic molecules that block IL-1.[31]

## SURGICAL MANAGEMENT

Although a subgroup of patients is likely to respond to nonoperative management, others will ultimately require operative management for pain relief and to improve functional activity or quality of life. Standardized treatment algorithms built on evidence-based methods have not been developed. Current available operative management options for patients with OA include arthroscopic débridement, drilling of exposed subchondral bone to encourage fibrocartilaginous repair, realignment osteotomy, unicompartmental arthroplasty, and total joint arthroplasty.[32]

### Arthroscopy

Arthroscopic débridement is a first-line procedure for patients with acute- or subacute-onset arthritic type joint pain of the knee. Mechanical symptoms caused by unstable articular cartilage flap tears, meniscal tears, or loose bodies are common indications for arthroscopy and débridement. Arthroscopic débridement of the arthritic knee is a good option for patients who have not responded well to conservative medical management. Additionally, some orthopaedic surgeons are performing arthroscopic microfracture at the time of initial débridement by using an awl to crack exposed or eburnated condylar bone, creating subchondral bleeding in hopes of stimulating fibrocartilage development.[33] Arthroscopic débridement is an outpatient procedure with less risk of serious complications than other surgical treatments for OA. The procedure may not exclude the need for further surgery, but it may provide temporary symptomatic relief.[34]

A study published in 2002 that followed 180 patients with osteoarthritic knees treated with arthroscopic débridement reported that there was no difference in pain or gain in functional outcome compared with placebo.[35] However, a follow-up study published by Dervin et al[34] investigated health-related quality-of-life scores of osteoarthritic knees following arthroscopic débridement in symptomatic patients. In this study, 126 patients aged 40 to 75 years in whom conservative medical management and supervised physical therapy

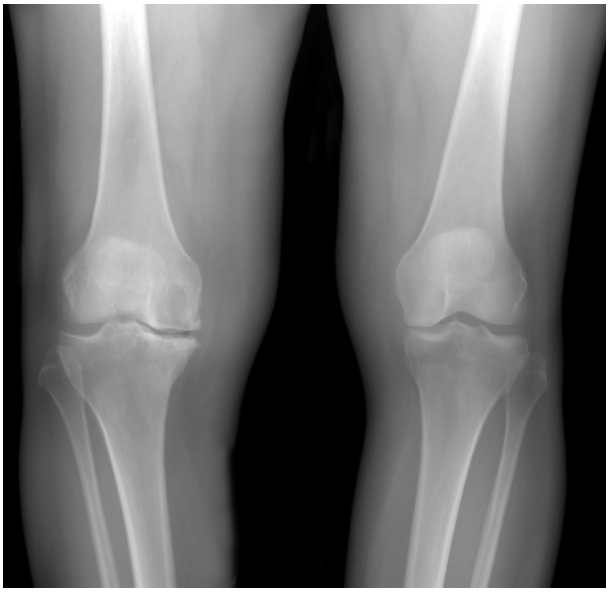

**Figure 1.** Radiograph showing preoperative end-stage left varus gonarthrosis.

failed underwent arthroscopic débridement, which included resection of unstable chondral flaps and meniscal tears. The Western Ontario and McMaster University Osteoarthritis Index scores were used to assess improvements in pain, stiffness, and physical function following arthroscopic débridement. Improvement in function occurred in 44% of patients, while the remaining patients were classified as failures and underwent either a repeat arthroscopic débridement, knee osteotomy, or total knee arthroplasty. Predictive success was associated with the presence of medial jointline tenderness, pain with internal and external rotation of the tibia with the knee at 90 degrees (Steiman test), and the presence of an unstable meniscal tear at the time of arthroscopy.[34]

Another available option for patients to arthroscopically alleviate arthritis is drilling of exposed subchondral bone to encourage fibrocartilaginous repair. The procedure includes débridement of osteophytes, loose bodies, and meniscal fragments. The success rate for the procedure has been similar to that of other fibrocartilaginous stimulation techniques. In a study by Petersen et al,[36] 46 of 60 patients were satisfied with the result of exposed subchondral bone drilling and considered it successful; 5 patients believed that their arthritic symptoms had worsened.

### Osteotomy

High tibial valgus osteotomy (HTO) or distal femoral varus osteotomy can be performed in young patients (ie, aged < 50 years) who have isolated unicompartmental arthritis of the knee with associated malalignment of the joint surface. Radiographic findings of unicompartmental arthritis knee include joint space narrowing confined to either the lateral or medial side of the joint surface with associated squaring of the involved femoral condyle and varus or valgus malalignment of the affected limb. The objective of the HTO procedure is to transfer weight-bearing forces from the arthritic portion of the knee to the healthier location of the joint. Holden et al[33] followed 45 young patients treated with osteotomy and found that 70% of patients had significant improvement in pain and their ability to participate in running and jumping activities—activities that they would be unable to do if they were managed with a total joint arthroplasty. Another study of medial opening wedge osteotomies of the proximal tibia using porous hydroxyapatite to treat medial compartment knee OA was successful in a series of 21 knees in 18 patients (average follow-up, 78.6 months).[37] All patients had pain relief and improvement in walking ability, and no patients required conversion to total knee arthroplasty or had graft collapse. Of note, external fixation of the proximal tibia is required with either pin fixation or external frame. Complications of HTO include pin track infection, nonunion, and deep venous thrombosis. In addition, an external frame is required for an average of 7 weeks.[16]

### Arthroplasty

If the patient has unicompartmental disease (**Figure 1** and **Figure 2**) without malalignment, studies have shown that a unicompartmental knee replacement is beneficial for providing symptomatic relief and improvement in function.[38] The indications for the procedure are (1) OA associated with full-thickness loss of cartilage that is limited to 1 tibiofemoral compartment diagnosed on standing and stress radiographs and (2) the absence of a systemic, inflammatory type of arthritic condition.[20] According to the follow-up study by Argenson et al,[38] of 147 patients who underwent unicompartmental arthroplasty, 88% reported significant improvement in pain and mobility, and 5% reported continued moderate to severe pain (based on the Hospital of Special Surgery Knee Score).

Patients with significant arthritic involvement of the joint space or those who have failed to achieve symptomatic relief from osteotomy or unicompartmental knee replacement may benefit from a total knee or total hip replacement (**Figure 3** and **Figure 4**). Total hip and total knee arthroplasties are reliable and suitable surgical procedures to improve joint function. A systematic review of published studies using outcome

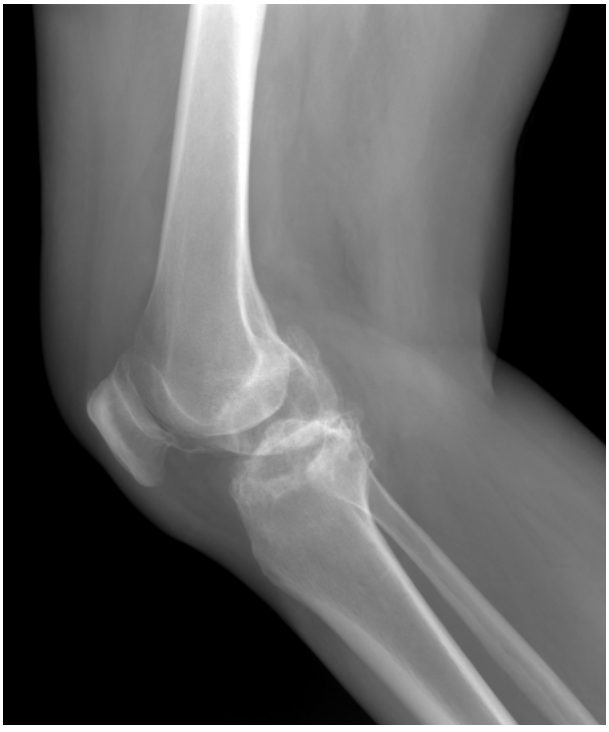

**Figure 2.** Left knee preoperative lateral radiograph demonstrating end-stage varus gonarthrosis.

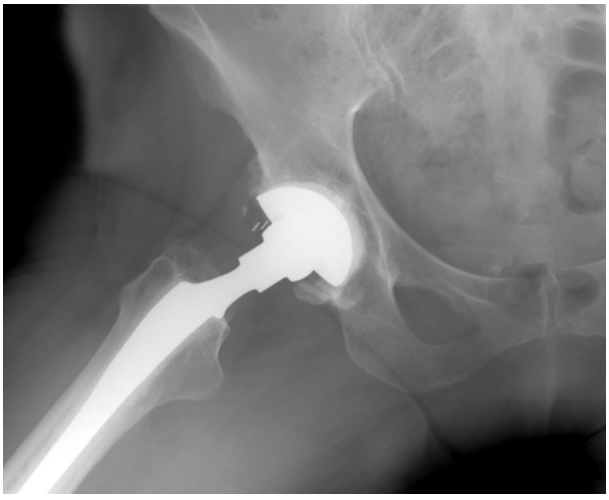

**Figure 3.** Postoperative frog-leg view of right cemented total hip arthroplasty.

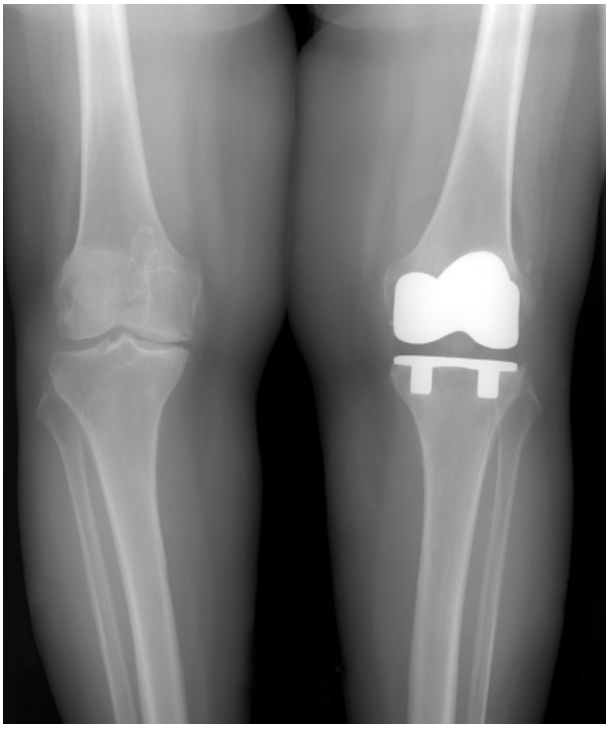

**Figure 4.** Postoperative radiograph of a left total knee arthroplasty.

scores of patients with severe OA treated with total hip and total knee arthroplasty using Short Form (SF)-36 and McMaster University Osteoarthritis Index found that both procedures improve health-related quality of life overall.[20] Fitzgerald et al[39] reported that 6 months after total hip arthroplasty patients' scores on all SF-36 subscales (pain, physical and social functioning, energy, and activity limitations) were the same or better than the age- and gender-adjusted population-based SF-36 scores. In a study of patients who had a total knee arthroplasty, Van Essen et al[40] reported that at 3 months postsurgery, there was significant improvement in pain and function. Potential complications of the procedure must be evaluated before making the decision to undergo total hip or total knee arthroplasty. The incidence of fracture, nerve injury, and vascular injury during total hip and total knee arthroplasty ranges from 0.1 to 1.0.[20]

### Other Surgical Options

Interest in developing disease-modifying therapies for diffuse OA of the knee and hip is expanding, and these therapies are currently under development. Current investigations include chondrocyte transplantation, gene therapy, small-molecule therapy, and marrow stimulation. Other new modalities available, such as allograft meniscal transplantation, can be performed after subtotal or total meniscectomy when arthritis is unicompartmental and not severe. However, the benefit versus risk of meniscal transplantation has not yet been determined.

## CONCLUSION

Arthritis is a leading cause of disability in the adult population. OA affects over 20 million Americans, and its prevalence has to the potential to dramatically rise with the aging population. Despite lack of complete understanding of OA, modern pharmacologic, biologic, and surgical advances are being made. More than ever, the restoration of a normal, active lifestyle can be achieved despite the diagnosis of OA. Science continues to investigate new areas of prevention and treatment, while improved surgical techniques and materials are providing less painful, more durable surgical options for OA patients. **HP**

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