

Mechanical Disorders of the Knee

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A mechanical disorder of the knee is defined as any condition interfering with normal joint motion or mobility. Such disorders include any intra-articular process or *internal derangement* blocking or limiting motion such as a torn meniscus, articular osteochondral fragment, or loose body. Conditions external to the joint leading to articular surface damage through altered joint kinematics are included also. Ligamentous disruption, patellar malalignment, and quadriceps or hamstring insufficiency are the most important of this group. Finally, knee osteoarthritis (OA), which ultimately results secondary to these problems, becomes an irreversible factor limiting normal joint function.

The pathomechanical basis of these disorders is due to abnormally increased stress at the cartilage articular surface accumulated over a long period. Whether this is a mechanical block, acting like a grain of sand in a fine bearing, or abnormal wear analogous to that occurring in an improperly balanced tire, the results are the same. The pathologic changes of OA with joint space narrowing, spurs, subchondral sclerosis, and joint deformity represent the final common pathway. This discussion will be limited to the common acquired conditions that are seen after maturity and are usually traumatic or degenerative in nature.

MENISCAL INJURY

Approximately two thirds of all derangements of the knee joint are due to lesions of the menisci.¹⁻⁶ These structures are triangular-shaped rings of fibrocartilage filling the space between the convex condyles of the femur and the flat posterior sloping tibial plateaus (Fig. 90-1). The collagen fibers are oriented circumferentially, allowing the meniscus to withstand tensile loading. This arrangement allows the translation of vertical

compressive loads into circumferential or hoop stresses. The menisci enhance knee stability, improve joint congruency, and transmit between 50 and 70% of the load applied across the knee during axial loading.⁷ Their nutrition comes from the joint fluid and from the blood vessels supplying their periphery. In the newborn, 50% of the periphery of the menisci is vascularized, but in adulthood only 10 to 30%, or 2 to 3 mm, has a blood supply.^{8,9}

Meniscal injuries are commonly associated with traumatic ligamentous disruptions resulting from athletic injuries and falls during which abnormal excursion of the articular surfaces under load entraps the margin of the meniscus. The ligament damage typically occurs with exaggerated valgus and rotation causing anterior cruciate stretching or tearing as the tibia moves forward under the femoral condyle. A vertical longitudinal tear of the middle portion of the meniscus results as the femur and tibia contact in this abnormal position (Fig. 90-2). With a displaced bucket handle tear, the torn inner portion displaces toward the intercondylar notch as the knee reduces.¹⁰⁻¹⁷

Acute hemarthrosis accompanies over 80% of these traumatic injuries and is associated with pain and limited range of motion. Displaceable tears cause the sensation of "catching" and "popping," and the knee may be locked in a flexed position. The symptom of "buckling" or "giving way" is due to instability from the commonly associated anterior cruciate ligament (ACL) injury. Joint line tenderness is the hallmark of meniscal injury and results from chronic pinching of the meniscus in the joint, which tugs on the peripheral joint capsule, which has a rich nerve supply.¹⁸⁻²⁰

Signs of meniscal entrapment include pain on forced flexion, the presence of a block to extension, and positive McMurray and Apley grind tests. The McMurray test is done with the patient in the supine position with

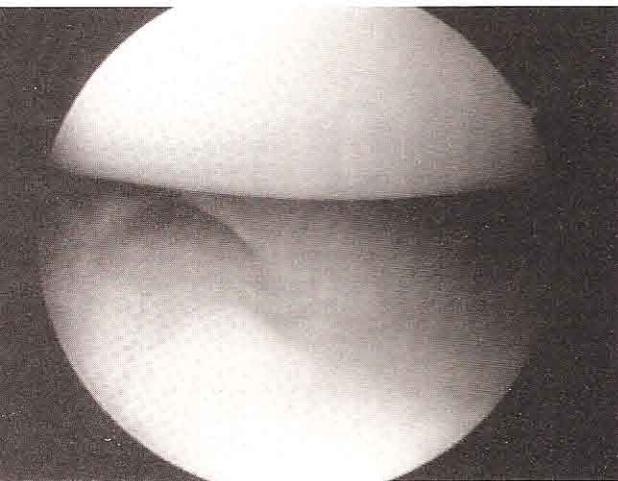


FIGURE 90-1. Arthroscopic view of normal lateral meniscus.

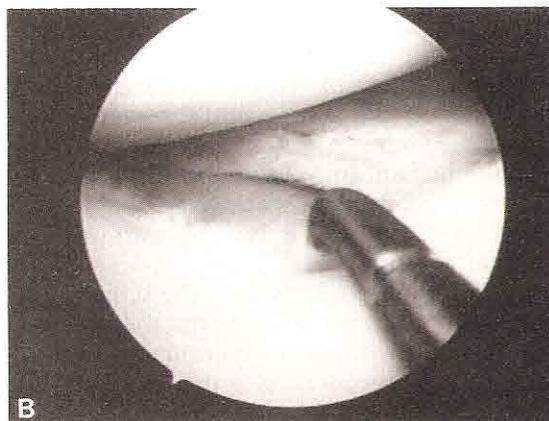
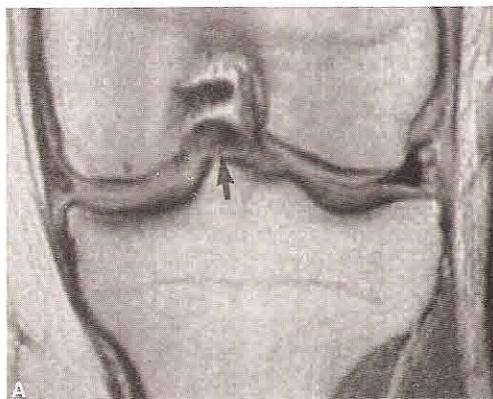


FIGURE 90-2. A, Magnetic resonance image of a displaced bucket handle tear (arrow) and absent meniscus sign. B, Arthroscopic view of verticle longitudinal medial meniscus tear with displaced bucket handle tear under probe.

the affected knee maximally flexed until the heel almost touches the buttock. The knee is then forcibly internally or externally rotated to impinge the lateral or medial meniscus. A palpable or audible snap in the joint constitutes a positive test. Pain may not be present in older tears. The Apley grind test is done with the patient prone. The tibia is flexed 90° and manually compressed on the knee joint. Rotation of the knee internally and externally will produce a “snap” consistent with a torn meniscus. A combination of joint line tenderness with one or both of signs correlates very well with subsequent diagnostic tests.²¹

Degenerative meniscal tears cause a horizontal cleavage. They usually occur in the posterior third of the meniscus and are frequently associated with OA (Fig. 90-3). Flap tears are common, most frequently occurring on the superior surface of the meniscus. Symptoms differ from verticle tears in that pain and injury are more subtle. An episode of squatting and twisting or a sudden misstep may be the only history. Effusion may be slight, but clinical findings of joint line tenderness and meniscal entrapment are usually present.

Dandy studied 1000 cases of symptomatic meniscal lesions with arthroscopy; 81% of the patients were men, most commonly with medial meniscus lesions of the right knee (56.5%). Of the medial meniscus tears, 75% were vertical, which were frequently locked; 23% were horizontal. Of the flap tears, superior flaps were six times more common than inferior flaps. Vertical tears occurred most frequently in the fourth decade, while horizontal tears were seen in the fifth. The common pattern seen in the lateral compartment was a vertical tear involving half the length and width of the meniscus. Myxoid degeneration consisting of a softening of the meniscal substance was seen in a small portion of the lateral tears. The patient experiences a deep-seated aching pain in the knee at night with localized swelling. In distinction from meniscal “cysts” or ganglions, there is significant destruction of the meniscus, which usually requires meniscectomy.²²

Clinical findings are accurate in making a proper diagnosis in over 75% of cases. The diagnosis of meniscal pathology can be confirmed by arthrogram, magnetic resonance imaging (MRI), or diagnostic arthroscopy. In experienced hands, arthroscopy is nearly 100% accurate in identifying the morphology of derangement and is considered the gold standard. Double-contrast arthrography has been used for many years and has an accuracy of 60 to 90%, depending on the experience of the person interpreting the study. Most recently, MRI has been used with a diagnostic accuracy of 70 to 95%. It is noninvasive, offers excellent soft tissue contrast, and has multiplanar capabilities. It has certain advantages over arthroscopy in that lesions involving the posterior horn or inferior surface of the meniscus may be identified. Other lesions may be differentiated such as osteochondral fractures, osteochondritis dissecans, ligamentous injuries, and pigmented villonodular synovitis. The greatest value of MRI lies in its high negative predictive value; in few cases, use of MRI failed to result in

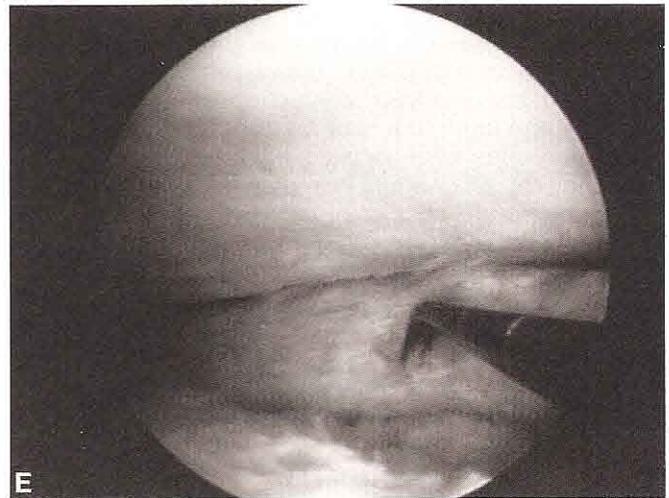
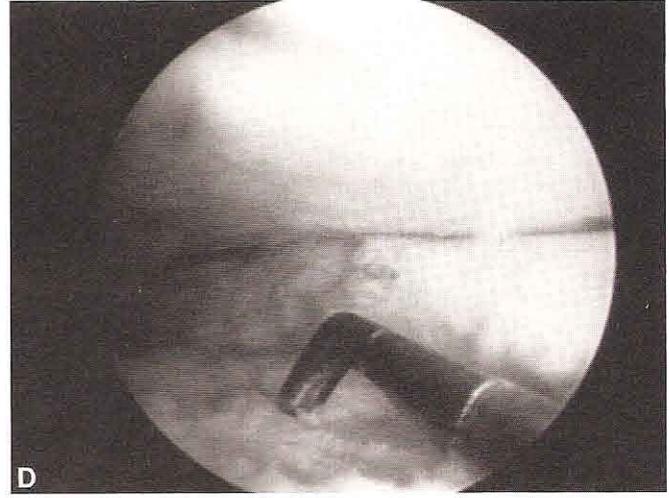
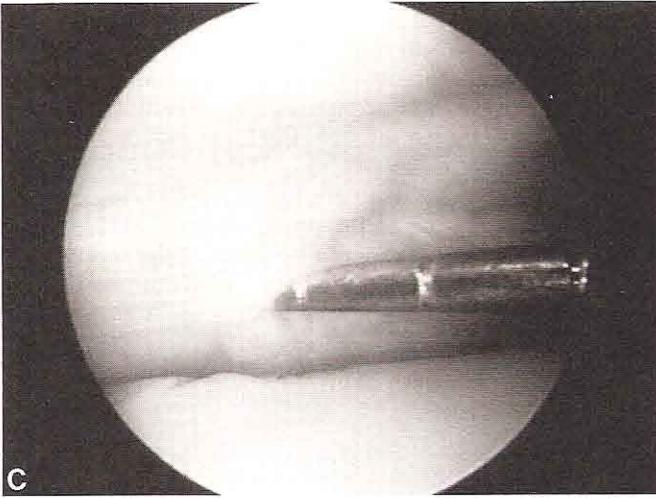
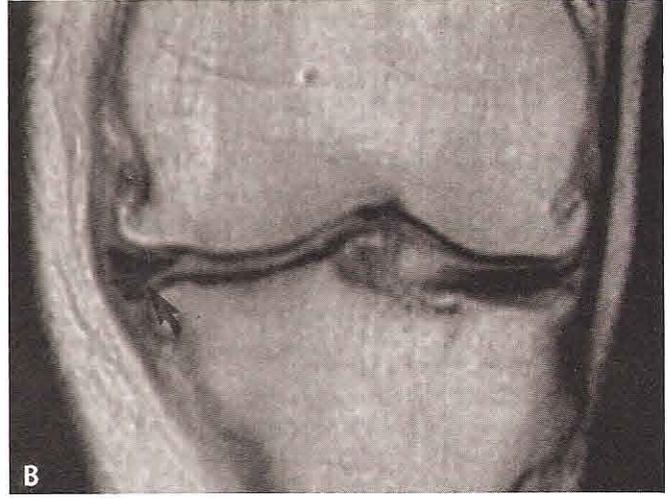
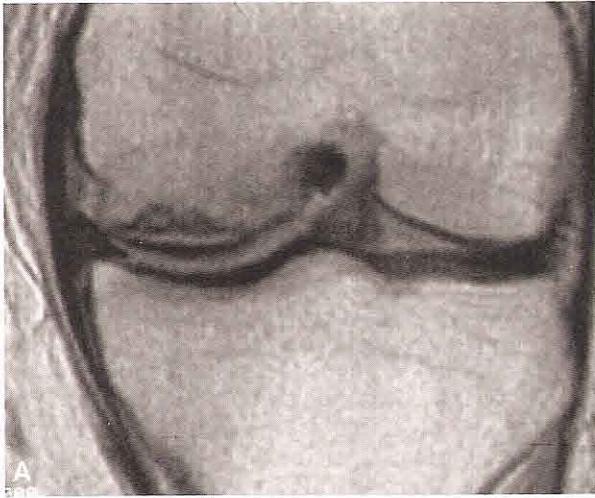


FIGURE 90-3. *A*, Magnetic resonance image of the knee of a 74-year-old man demonstrating concomitant spontaneous osteonecrosis of the medial femoral condyle. *B*, Complex degenerative horizontal cleavage tear of medial meniscus (*arrow*). *C*, Arthroscopic appearance of the degenerative medial meniscus with probe in substance of tear. *D*, Associated chondral lesion with exposed subchondral bone. *E*, Partial medial meniscectomy to a stable rim.

diagnosis of a tear. MRI is less accurate in diagnosing degenerative tears and will often result in identification of increased signal intensity where no lesion is found. This may represent a lesion predisposing to degenerative tears and structures such as the meniscofemoral ligaments and the bursa of the popliteal ligament, can be mistaken for meniscal tears on MRI.²³⁻³⁰

Operative arthroscopy is the standard diagnostic and treatment modality for meniscal lesions. Long-term outcome studies have not shown a distinct advantage over open meniscectomy, but patient recovery may be enhanced by the more limited approach. Access to the menisci is much easier with the arthroscope, and partial meniscectomy is possible. Arthroscopy is a major surgical procedure, and significant complications have been noted including infection, phlebitis, neurovascular injury, and anesthesia death. Conservative treatment therefore is warranted in most cases except when a "locked" meniscus causes great pain and limited motion. Because of the high diagnostic capability of clinical examination correlated with MRI, there is little place for "look see" diagnostic arthroscopy. This approach fails to find pathologic changes in a high proportion of cases.

Meniscectomy is the treatment of choice for detached and unstable fragments of meniscus. The long-term effects of this treatment include narrowing of the joint space, ridge formation, flattening of the femoral condyle, and predisposition to OA. Articular cartilage degeneration results from the loss of the 30 to 60% load transmission normally passing through the menisci. Partial meniscectomy, leaving the peripheral portion intact, had a less dramatic biomechanical effect. Leaving the meniscal rim intact after removing a torn bucket handle has resulted in significantly fewer long-term degenerative changes. Also fewer arthritic changes also were found in dogs after a partial meniscectomy. The standard arthroscopic technique at this time is partial meniscectomy debriding the meniscus to a stable rim.³¹⁻³³

Because of the late arthritic changes after meniscectomy, meniscal repair has been advocated for vertical peripheral tears occurring in the vascular zone within 2 mm of the meniscosynovial junction. These tears must be short, and there should be no tear extending into the substance. Finally, associated ligamentous instability involving the ACL should be repaired at the same time.

The late results after meniscectomy are definitely related to the amount of pre-existing degenerative changes identified at the time of surgery by radiography and by arthroscopy. Excellent late functional results occur after meniscectomy if the articular cartilage is well preserved, but results are less satisfactory if significant arthritis was present. Nonetheless, locking and joint line pain are eliminated in most patients despite the arthritis.

LIGAMENTOUS INSTABILITY

Disruption of knee ligament alters the limits of joint motion. The normal kinematics of joint function are changed, with exaggerated stresses on the articular car-

tilage, leading to degenerative arthritis. Instability or abnormal motion resulting from a torn ligament can occur in the anterior, posterior, medial, or lateral direction. The four primary ligaments limiting this motion are the ACL, posterior cruciate, medial collateral, and fibular collateral. Each ligament provides restraint regarding its specific motion depending on the angle of flexion of the knee. As the knee extends, isolated ligament restraint lessens and the concerted function of several ligaments or secondary restraints increases. For example, the medial collateral ligament (MCL) is the primary restraint to valgus angulation with the knee in 30° of flexion. The MCL is also a secondary restraint to anterior displacement, which is controlled by the ACL. If the ACL is disrupted, the MCL then is one of several structures limiting anterior displacement.³⁴⁻³⁸

With major ligament disruption, rotational instability occurs. The femur rotates abnormally on the fixed tibia. This is an important clinical finding because the typical episode of buckling or "giving way" seen with functional instability reproduces this phenomenon. ACL disruption results in anterior displacement of the tibia, but anterolateral rotatory instability also occurs. The lateral tibial plateau rotates and subluxes anteriorly with knee extension and then reduces as the knee moves into flexion (the pivot shift sign).³⁹⁻⁴¹

The diagnosis of ligamentous disruption is usually made by physical examination. Anesthesia may be needed if the injury is acute because of the associated pain and swelling. Combined with instrumented measurements such as the KT 1000 or stress radiographs, 90% diagnostic accuracy can be achieved. In most clinical grading systems, grade I laxity represents up to 5 mm of motion; grade II, 6 to 10 mm; grade III, 11 to 15 mm; and grade IV, greater than 15 mm. The opposite normal knee is used as the reference point.⁴²

The most sensitive test of a torn ACL is the anterior drawer sign. This maneuver is performed with the knee flexed to 25° (Lachman's test) (Fig. 90-4). Posterior cruciate ligament integrity is best measured by the posterior drawer test, which is done with the knee flexed to 90°. Medial collateral ligament laxity is best tested using a valgus stress with the knee flexed 30°. Fibular collateral ligament integrity and lateral capsule laxity are tested using a varus stress with the knee flexed to 30°. It is important that the knee is first in neutral position before testing. In the 90° flexed position, one looks for posterior sag of the tibia compared to the opposite normal knee, which indicates posterior cruciate ligament disruption. The neutral position for varus, valgus tests in the coronal plane is the position in which the medial and lateral joint surfaces are in contact. Rotational tests include the pivot shift test, indicating anterior subluxation of the lateral tibial plateau secondary to ACL disruption; reverse pivot shift test, which is posterior subluxation of the lateral tibial plateau (secondary to posterolateral capsule disruption); and anteromedial subluxation (secondary to injuries of the anteromedial structures) (Fig. 90-5).⁴³⁻⁴⁶

The most common disabling knee injury in athletes

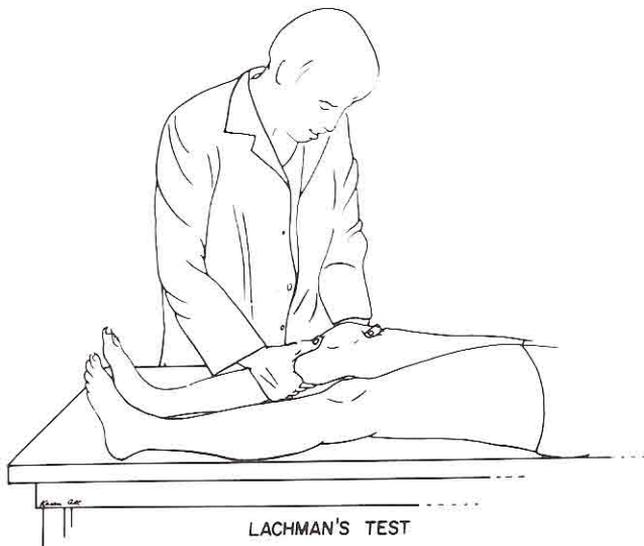


FIGURE 90-4. Lachman's test for anterior cruciate stability done with the knee at 25° flexion.

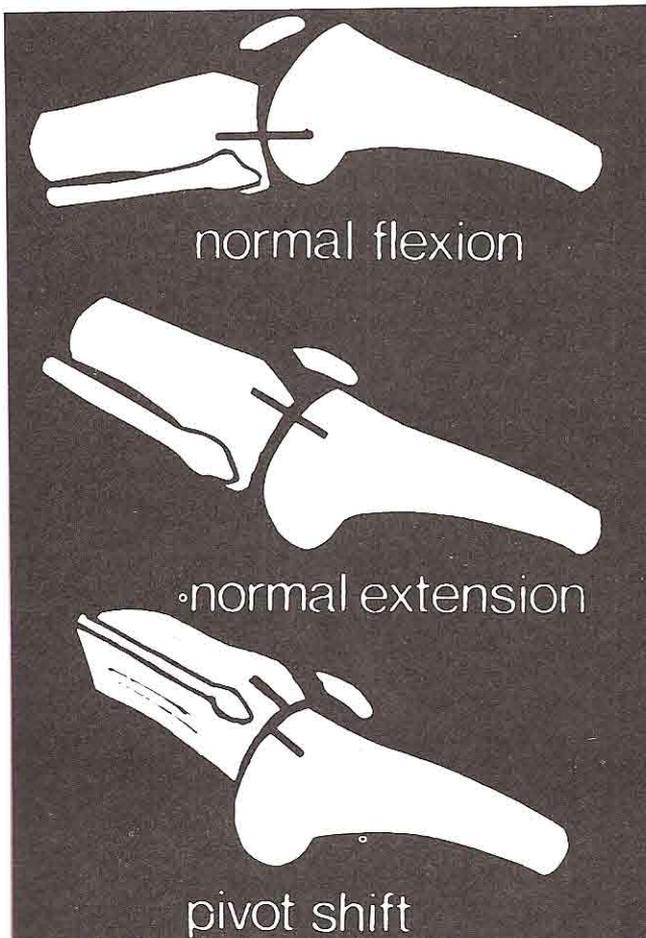


FIGURE 90-5. Principle of "pivot shift" phenomenon, anterior lateral tibial subluxation at full extension with reduction as the knee flexes past 20°.

is the ACL disruption. Over 70% of ACL injuries occur during sports activities and involve jumping and cutting motions during which abnormal valgus rotation forces cause disruption of the ligament. Severe swelling occurs within 2 to 6 hours. Over 80% of knees with traumatic hemarthrosis have acute ACL injuries. Most cases can be diagnosed by physical examination. Arthroscopy is reserved for cases with suspected associated damage such as torn menisci, osteochondral fractures, or loose bodies.⁴⁷⁻⁵¹

Treatment of ACL injuries is based on the activity level and functional expectations of each individual. Patients with grade I or II laxity remain functionally stable after rehabilitation and modification of their activities. Those with grade IV knee laxity can be severely disabled whether the individual is athletic or not. Surgical reconstruction is usually recommended in such cases. Patients with grade III laxity fall into the "gray zone." Such knees may need surgical intervention if the patient wishes to be athletic, or if other associated ligaments are injured, or if there is a reparable meniscus. If doubt exists in the patient's mind regarding surgical reconstruction, then conservative measures are generally indicated. There is no conclusive evidence that surgical repair avoids late complications such as meniscal tear or degenerative arthritis.⁵²⁻⁵⁷

A medial collateral ligament injury usually heals satisfactorily without surgery, but there may be some late morbidity after unrepaired grade III disruptions. Treatment usually consists of a hinged cast or brace for 3 to 6 weeks. When MCL disruption is combined with ACL disruption, surgical repair is usually indicated. Not uncommonly, disruption of the medial retinaculum and vastus medialis is associated with patellar dislocation. Lateral ligamentous disruptions, like those of the medial ligaments, are usually associated with ACL disruption. Surgical repair of the fibular collateral and posterolateral structures (arcuate ligament complex and popliteus tendon) are generally recommended.^{58,59}

Posterior cruciate ligament disruption is rare, occurring only 10% as frequently as ACL injury. Associated meniscal injuries are infrequent, but articular surface damage is not. This may result from the exaggerated pull of the quadriceps tendon on the patella to stabilize the knee in flexion. Many patients are only modestly impaired from this injury, but the incidence of late degenerative arthritis is high. Surgical repair is indicated when bone fragment is avulsed, enabling a stable fixation of the ligament. Repair of midsubstance tears are unsuccessful. Reconstructions are variably successful.⁶⁰⁻⁶²

Rehabilitation is important in the treatment of any knee injury. Normally, the hamstrings are two thirds as strong as the quadriceps, but after ACL injuries, the goal is to make the hamstring/quadriceps strength ratio 1 : 1. On the other hand, in posterior cruciate ligament injury, the goal is strengthening of the quadriceps maximally for knee stability. Similarly, after lateral ligament injuries with posterolateral instability, quadriceps strength

is maximized and flexion exercises are eliminated to remove the detrimental effect of the biceps femoris.

OSTEOCHONDRITIS DISSECANS

Although the term denotes inflammation of bone and cartilage, osteochondritis dissecans presents with little inflammation. The condition occurs when an island of subchondral bone with its articular cartilage separates from the femoral condyle. This island, usually 1 to 2 cm in diameter, often "dissects" from the main condylar mass. It can be thought of as a small fracture that develops chronic nonunion. Eighty-five percent of these lesions are found in the knee joint, but they have been reported in many sites, including the femoral head, the dome of the talus, and the capitulum humeri.⁶³⁻⁶⁵

Osteochondritis dissecans is a condition of unknown origin in which 85% of the lesions appear on the medial or central portion of the medial femoral condyle and 15% occur laterally. These lesions are possibly shear fractures, but their occasional "mirror-image," bilateral occurrence suggests other anatomic or developmental causes. Prominent tibial spines have been associated with the condition and have been implicated as sites of impingement with rotation of the tibia against the condyles. The small bony island is probably alive at the time of separation or fracture. The edges of the bony fragment become necrotic, but the overlying articular cartilage, nourished by synovial fluid, remains alive. Studies on the blood supply of the condyles indicate a rich, anastomotic arterial network that makes localized infarction an unlikely cause. The lesion has been reported in families as an autosomal dominant condition.

This lesion is most common in adolescents and young adults, less common in patients in their 30s and 40s, and rare in the elderly, in whom degeneration overshadows the primary cause. Males are affected three times more than females. Shed or free fragments of bone loose in the knee joint are common in adults and rare in children. There are two types of lesions. One occurs in the skeletally immature individual, and healing almost always follows restriction of activity and splinting. The other occurs in the skeletally mature patient in whom there is a high incidence of fragment separation.

MRI can demonstrate the subchondral fracture but may not always permit detection of cartilage disruption. Arthroscopy is the best way to assess whether the lesion is a fragment in situ, a partially detached fragment, or a free body. Treatment of in situ lesions may be done by drilling the fragment in cases where healing is delayed. For the more usual partially detached fragment, bone grafts may be applied either directly under the articular cartilage or from a superior hole directed into the lesion using the arthroscope. Stabilization of free fragments includes inserting pins with the arthroscope. If a free fragment cannot be replaced, the free body is removed and the remaining bed is trephined to allow for fibrocartilaginous ingrowth and healing.⁶⁶⁻⁶⁸

Long-term followup studies have revealed that many

patients with osteochondritis dissecans develop degenerative arthritis. Thirty-eight of 48 patients seen at an average of 33 years after diagnosis had arthritis. At least 20 years of observation are often required before arthritis is manifest. The arthritic process is accelerated in athletes.

TRAUMATIC CHONDRAL FRACTURES

Isolated cartilage fractures of the articular surface are not uncommon arthroscopic findings when meniscal pathology is suspected. Patients can usually describe an injury such as contact with an automobile dashboard or torsion during athletics. Symptoms usually include pain and chronic effusion, and there may be localized joint line tenderness. Terry has described three types of lesions: (1) stellate with central defect and radiating fissures, (2) flap with attached base, and (3) crater-type with exposed defect. In all cases, the underlying subchondral bone was exposed. Bony contact may be an important factor in these injuries. The tibial spine can be opposed to lesions on the medial femoral condyle, and in other cases, the lateral femoral condyle and tibial plateau lesions match in a weight-bearing position.

Treatment of these lesions remains controversial because of the limiting healing potential of damaged cartilage. Mankin has suggested that once a critical threshold of injury is reached with articular cartilage, permanent damage occurs and the occurrence of osteoarthritis is invariable. Thus, chondral fractures have a worse prognosis than typical meniscus tears. The long-term outcome of arthroscopically proven cases is unknown. These lesions are of interest relative to the pathophysiology of cartilage degeneration and illustrate the morbidity that may result from seemingly benign knee injuries.⁶⁹⁻⁷²

PATELLOFEMORAL MALALIGNMENT

Anterior knee pain is an important and common affliction often ascribed to chondromalacia patella, a morbid softening, fissuring degeneration process of the patellar cartilage. For the most part, however, this condition is primarily associated with patellar malalignment. The association between patellar crepitus and pain has been noted for many years. More recently, specific data have accumulated defining how malalignment can be studied and treated.⁷³ Using a specific 45° tangential radiograph of the patella, Merchant separated chondromalacia cases into two types: (1) those with normal alignment and (2) those with abnormal alignment resulting in lateral subluxation.⁷⁹ This problem has been further studied using tangential computed tomography with the knee in slight flexion. A classification scheme has evolved such that the direct arthroscopic appearance of the articular lesion can be correlated with findings of subluxation, subluxation and patellar tilt, patellar tilt alone, or normal alignment.⁷⁴

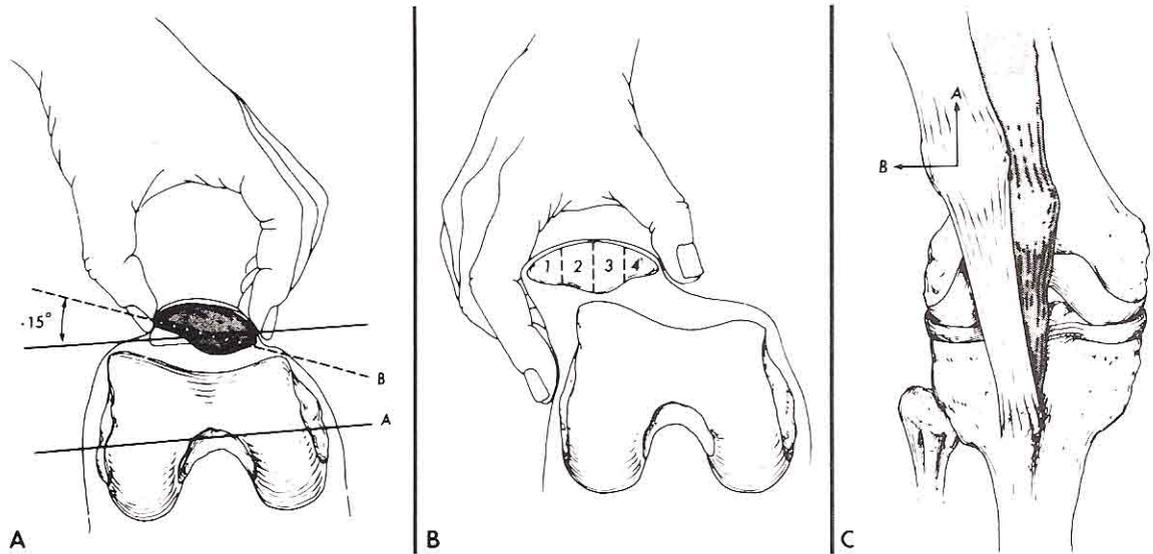


FIGURE 90-6. *A*, Passive patellar tilt indicates laxity of lateral ligamentous restraints. A patellar tilt of 0° or less indicates tight lateral retinacular ligaments. *B*, Passive lateral glide test demonstrates the ability to laterally displace the patella with the knee in full extension, subluxation beyond one half of its width indicates laxity of medial retinacular restraints. *C*, Increased “functional” Q angle is present if lateral excursion (*B*) exceeds proximal excursion (*A*).

The clinical evaluation of patellar alignment requires a detailed history of the onset and progression of patellar instability and pain. Bilateral pain and recurrent dislocation are usually associated with malalignment. A direct blow to the anterior knee does not cause malalignment. Symptoms consist of pain with activities involving knee flexion such as ascending or descending stairs, running, jumping, squatting, and progressive resistive exercise. Localized aching occurs after periods of immobility with the knee in the flexed position, such as watching television or working at a desk (the “movie sign”). Tenderness of the peripatellar muscle, tendon, and retinaculum may result from stretching of the lateral retinaculum when the extensor mechanism is abnormally aligned. This is to be distinguished from articular pain that is due to patellofemoral OA, which may be related to malalignment but can also be caused by trauma. Articular pain is usually increased by patellofemoral compression and is usually coupled with crepitus and effusion. Not all patients with crepitus have pain. Conversely, many have anterior knee pain without signs of chondromalacia or crepitus.^{75,76}

Physical examination of the knee may reveal noticeable tilting of the patella with active flexion and extension. Normally, the patella is firmly engaged in the trochlea at 30 to 40° of flexion. The passive patellar tilt test assesses patellar laxity when the lateral patellar edge is elevated from the femoral condyle. A 0° or a negative tilt angle usually reflects excessive lateral retinacular tightness. The passive patellar glide test estimates lateral patellar excursion. Lateral overhang exceeding half the width of the patella usually indicates lax or torn medial

patellofemoral restraints. The functional quadriceps (Q) angle measures the amount of lateral excursion compared with superior excursion as the patient contracts the quadriceps with the knee flexed 20° . If lateral excursion exceeds superior excursion, then an excessive lateral quadriceps vector exists. The overall alignment of the lower extremity should be assessed with the patient standing; look for anatomic variants that may increase the Q angle (i.e., the line of quadriceps pull from the patella and tibial tubercle) (Fig. 90-6). Vastus medialis

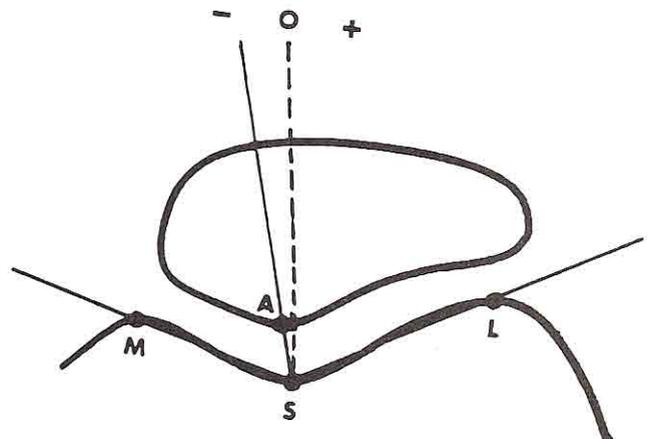


FIGURE 90-7. Merchant’s angle of congruence measured from the sulcus angle MSL (mean = 137°) and SO, which bisects the sulcus angle. The congruence angle is ASO (mean = 6° and abnormal if greater than 4°).

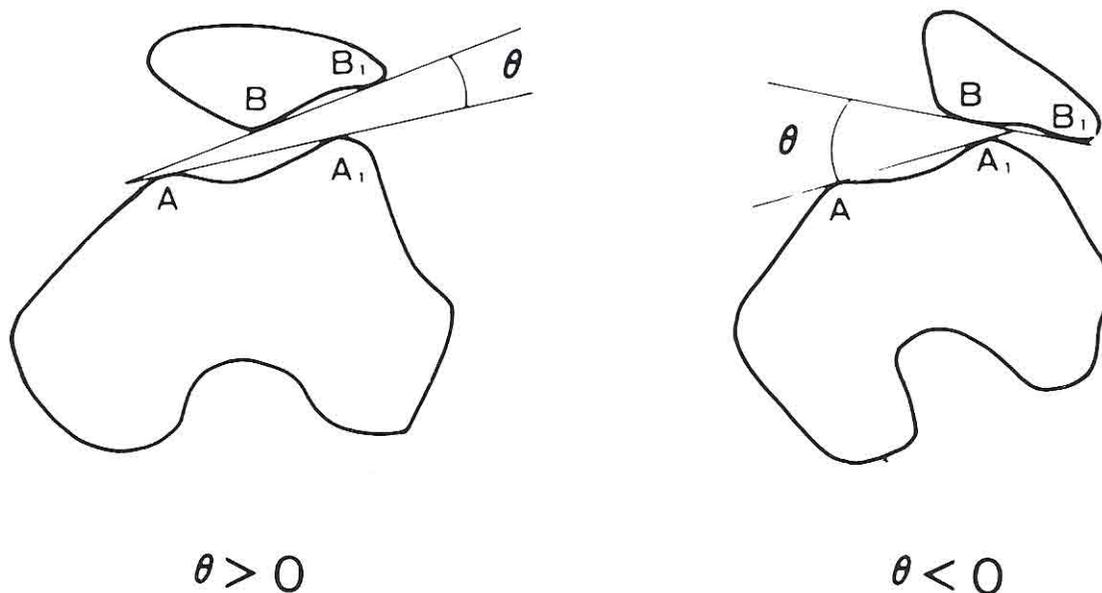


FIGURE 90-8. Angle of Laurin measures the lateral patellofemoral angle, where line A-A1 passes through the femoral condyles and B-B1 passes through the lateral patellar facet. The angle is positive if it opens laterally, and abnormal if it opens medially.

atrophy, laterally placed tibial tubercle, valgus deformity of the knee, and generalized laxity with forefoot pronation are findings associated with malalignment.⁷⁷

Radiographic analysis of the patellofemoral joint includes the so-called "sunrise" (tangential) view of the patella. Merchant measured the angle of congruence on a standard tangential (axial) radiograph done with knee flexed 45°, and the x-ray beam directed at a 30° angle from the horizontal. This angle is made from a line of the bisected femoral trochlea and a line of the apex of the patella to the trochlear groove.⁷⁸ Aglietti believed that this angle of congruence should be less than 4°. It usually is negative (Fig. 90-7). Patellar tilt can be measured by the angle of Laurin, which measures the lateral patellofemoral angle with the knee in 20° of flexion. This angle measures the line of the lateral patellar facet compared with the line drawn across the lateral femoral trochlea. In the normal patellofemoral joint, if this angle opens laterally, it is positive. If there is abnormal patellar tilt, it opens medially and is negative (Fig. 90-8).⁷⁷⁻⁸⁰

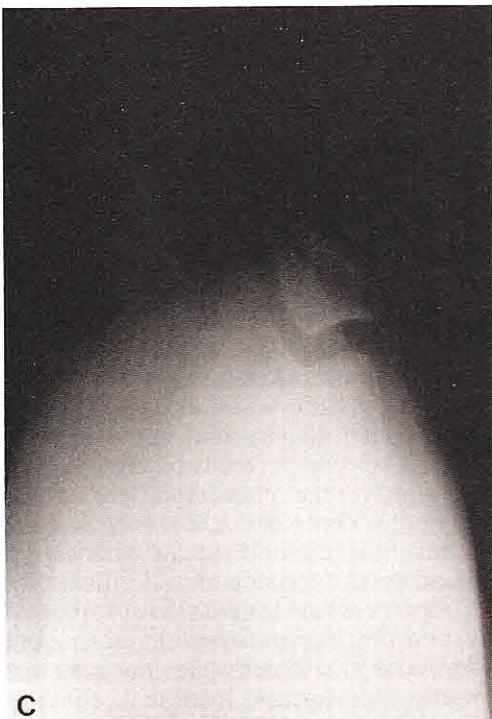
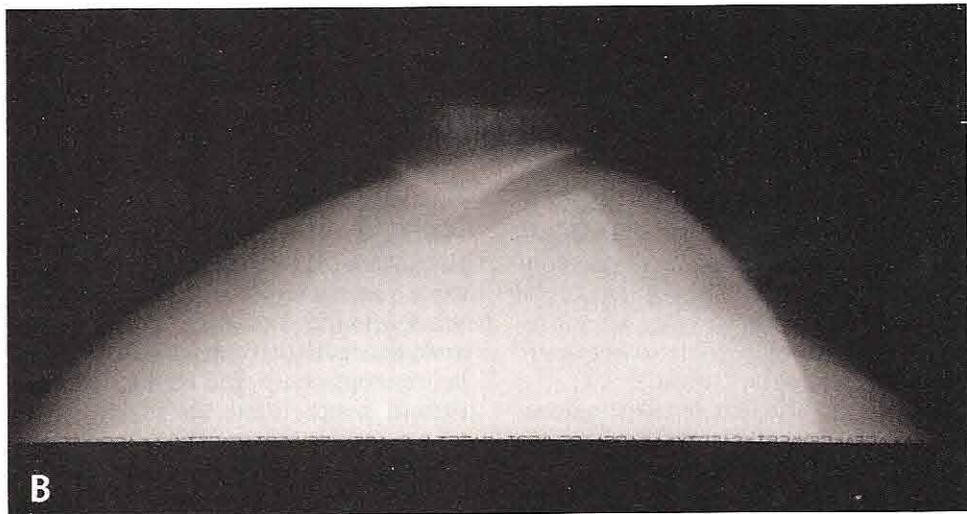
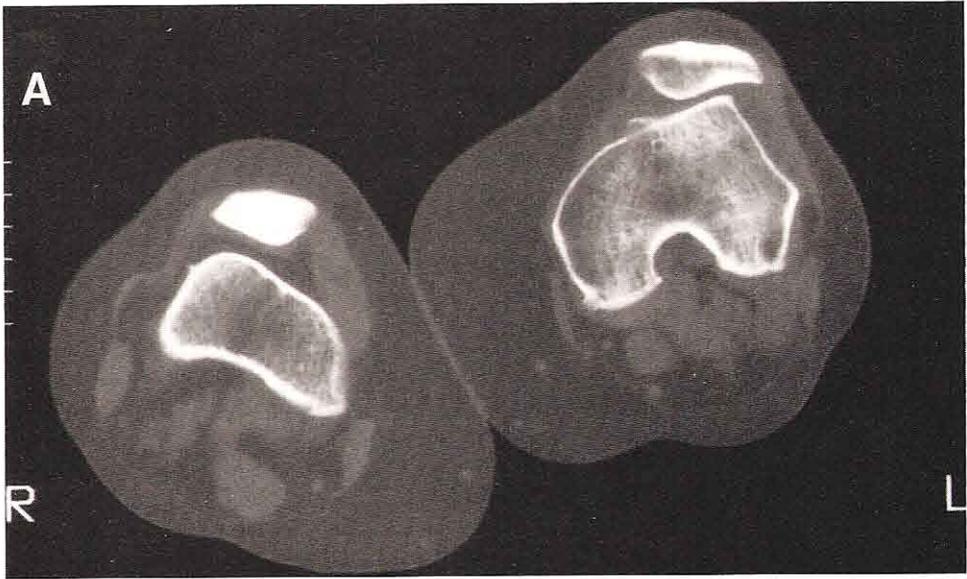
Schutzer used computed tomography to define the normal patellar alignment. Using midpatellar transverse cuts at 0, 10, 20, and 30° of flexion, the patella enters the trochlea early in flexion of the knee with the angle of congruence equal to 0° at 10 and 20° of flexion. The patella is centered and upright without tilt beyond 10°

of flexion.^{80a} Computed tomography (if properly done) more accurately defines the normal patellofemoral relationship than do radiographs as they are obtained with the knee in near full extension where the subluxation occurs (Fig. 90-9). Standard radiographs must be taken with the knee in at least 20 to 30° of flexion and are less able to show the problem. Finally, technetium bone scintigraphy is helpful in identifying patellofemoral arthrosis and planning surgery.⁸¹

Arthroscopy is invaluable for assessing the extent of damage to the articular cartilage. Grading is done by the classification scheme of Outerbridge: stage 1, softening of the cartilage only; stage 2, fibrillation of less than 0.5 inch in width; stage 3, fibrillation or more than 0.5 inch in width; and stage 4, exposed bone.⁸² Arthroscopic chondral debridement is indicated when recurrent effusion is caused by cartilage flaps and fibrillation. The underlying malalignment, however, must be corrected to prevent further progression of articular cartilage damage.

Patients may have varying degrees of patellar subluxation, tilt, or OA. Patients with subluxation alone may have transient episodes of instability more than pain. Osteoarthritis is not a prominent finding unless the patella has been damaged by dislocation. Patellar tilt without subluxation increases the risk of medial patellar OA from abnormal lessened contact and OA of the lateral

FIGURE 90-9. Radiographic evaluation of symptomatic patellar subluxation in a 38-year-old woman with patellofemoral arthrosis: A, Technetium bone scan demonstrates uptake in lateral patellofemoral joint. B, Hughston sunrise at 60° flexion shows no subluxation. C, Merchant's view at 30° flexion shows no subluxation. D, Computed tomography at 15° flexion shows significant subluxation.



facet (lateral facet syndrome) because of increased pressure. There is lateral retinacular pain as the retinaculum is adaptively shortened and stretches as the knee flexes. The most symptomatic patients have both tilt and subluxation, which causes lateral facet syndrome, medial facet arthrosis, and instability. These patients have retinacular pain early in the course and have both articular breakdown and even patellar dislocation.^{83,86}

Treatment of patellar malalignment is conservative initially. This includes exercises to stretch the retinaculum, hamstrings, and iliotibial band and to strengthen the quadriceps muscle, particularly the vastus medialis obliquus. Other modalities include elastic knee supports, anti-inflammatory medication, orthotics, and modification of activity. Short arc quadriceps exercises are important, and deep knee bends are to be avoided. Patients with crepitus and recurrent effusion are less likely to respond to this program. Other intra-articular pathology must be ruled out.⁸⁷

Operative treatments aim at either correcting malalignment or reducing the symptoms of OA. For patellar subluxation, retinacular lateral release may be all that is needed, but the results of this intervention alone are inconsistent. Proximal medial advancement of the vastus medialis obliquus or even anteromedial transfer of the tibial tubercle may be needed to restore normal alignment. Patients with patellar tilt and no OA demonstrated by computed tomography will respond to lateral release alone. In patients with tilt and OA, lateral release and anteromedialization of the tibial tubercle are needed to reduce the forces on the patella.⁸⁸

When degeneration of the articular cartilage reaches stage 3 or 4 on the lateral facet, lateral retinacular pain may resolve as the tension on the ligament shortens with cartilage loss. If bone scintigraphy is positive, articular damage is already significant, and lateral release is unlikely to be helpful. Patellectomy has been done in the past but often leaves the patient in chronic pain because of pre-existing damage to the femoral trochlea. Other problems include extensor lag and instability. Anterior tibial tubercle transfer by the Maquet procedure has been advocated, and significant relief of pain occurs if 10 to 15 mm of anterior transfer is done. This procedure has a significant complication rate including skin breakdown, infection, and nonunion of the graft. An alternative is the anteromedialization of the tibial tubercle described by Fulkerson: The tibial tubercle is advanced both anteriorly and medially for a distance of up to 17 mm. This procedure is for those cases with both OA and malalignment and has fewer reported complications. Accurate diagnosis is needed in treating these cases, and poorly planned procedures are likely to be ineffective or cause iatrogenic complications.⁸⁸⁻⁹⁰

SPONTANEOUS OSTEONECROSIS

The syndrome of osteonecrosis was defined by Ahlback and Bauer as sudden onset of pain in an older patient localized over the medial femoral condyle with a posi-

tive bone scan and the eventual development of a subchondral radiolucent lesion. Women over 60 years of age are most commonly affected. The onset of pain is so severe that many patients can describe the exact moment when it began. Night pain is common; activity only moderately increases pain. Exquisite tenderness exists over the medial femoral condyle and joint effusions are common. Early on, the hallmark is an intense uptake of bone-seeking radionuclide. These lesions also can be detected by MRI.^{91,92}

The treatment and prediction of ultimate outcome depend on the radiographic features as well as the clinical picture. If bone scan and MRI findings are positive but standard radiographs are normal, the lesion is probably too small to be of consequence, and these patients can be treated with protracted protected weight bearing and analgesics. When the radiographic appearance is abnormal, these lesions can predictably be placed in one of two groups, depending on the size of the defect as a percentage of the affected condyle. If the lesion is less than 50% of the affected condyle, the prognosis is good and should be treated conservatively unless and until secondary degenerative changes occur. If medial compartment OA develops, high tibial osteotomy or unicompartmental arthroplasty is indicated.⁹³⁻⁹⁵

In those cases involving more than 50% of the articular surface of the medial femoral condyle, there is a more progressive downhill course with bone loss and the development of contractures. Curettage of the lesion and proximal tibial osteotomy is not helpful. Unicompartmental arthroplasty should be done early, once the clinical pattern is recognized. For severe cases, bicompartmental total arthroplasty may be needed.^{96,97}

A similar condition has been recognized in the proximal tibial plateau, but these lesions are usually small and respond to conservative treatment. The cause of osteonecrosis of the knee remains unknown, but most authors regard it as a subchondral stress fracture as it more typically occurs in older osteoporotic women. Healing of these lesions is variable, and the ultimate outcome depends on the size of the lesion.⁹⁸

OSTEOARTHRITIS

The cause of OA (see also Chapter 102) has been related to both injury and chronic occupational overuse. In one study, 4.5 times as many OA patients had a history of knee injury compared to controls. Obese people were at least 3.5 times more likely to have OA; individuals engaged in heavy manual labor were two to three times more likely to develop this condition. Long-distance running, on the other hand, was not likely to cause a higher incidence of OA, but such runners are not likely to be obese, and individuals with mechanical disorder or early OA probably exclude themselves and are not likely to become long-distance runners.⁹⁹

Bony fracture is an important cause of late joint problems and OA. Elderly patients have generally poorer results after fracture treatment, either conservative or



FIGURE 90-10. Anterior posterior (A) and lateral (B) radiograph demonstrating typical late findings of degenerative arthritis.

surgical. Violent injuries resulting in joint-comminuted and displaced fractures were found more likely to lead to OA. Also, most patients who were considered treatment failures developed arthritis early within the first 6 to 8 years. Anatomic restoration of articular congruity and joint alignment with early active and passive motion of the joint are the goals of early fracture treatment.¹⁰⁰

From the foregoing discussion, the final common pathway of all mechanical disorders is secondary OA typified by cartilage loss, joint space narrowing, osteophyte formation, and angular deformity (Fig. 90-10). Clinical findings of importance include chronic pain unrelieved by nonsteroidal anti-inflammatory drugs, crepitus, joint line tenderness, significant loss in range of motion, and external varus or valgus deformity. Radiographically, it is important to define the degree of joint space narrowing. This can best be done with a 45° posteror anterior flexed weight-bearing radiograph, because the areas of increased contact of the articular surfaces and subsequently degeneration occur with the knee flexed 30 to 60°. If the joint space is narrowed more than 2 mm compared to the opposite normal side, then grade 3 or 4 degeneration of cartilage is likely to be present.¹⁰¹

The late treatment of knee OA is initially conservative and includes weight reduction, rest, restriction of activity, nonsteroidal anti-inflammatory medication, occasional injection of corticosteroids, and physical therapy. In cases for which this treatment is ineffective, alternatives include osteotomy to correct severe angular deformity and joint arthroplasty. With continued technical improvements, total joint replacement becomes an even more attractive salvage procedure in older patients.¹⁰²

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