CHAPTER 32

# Avascular Necrosis and Osteochondritis Dissecans

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## AVASCULAR NECROSIS

### Introduction

*Osteonecrosis* is synonymous with *aseptic necrosis* and *avascular necrosis* (AVN). After the hip, the knee is the second most common location for osteonecrosis but represents less than 10% of all cases of osteonecrosis (7,8). Three general categories of AVN include spontaneous, atraumatic, and traumatic. Spontaneous or idiopathic AVN of the knee was first described by Ahlback in 1968 and has since been referred to as Ahlback's disease (1). Atraumatic or secondary AVN is also caused by the use of corticosteroids, Gaucher disease, caisson worker's disease, and sickle cell disease (2–6). Traumatic AVN of the knee is theoretically seen after fractures of the distal femoral condyles or tibial plateau or after surgeries, such as high tibial osteotomy or posterior cruciate ligament reconstruction, related to femoral condyle tunnel preparation. This section focuses primarily on spontaneous or idiopathic AVN and discusses secondary osteonecrosis under Differential Diagnosis.

### Epidemiology

Spontaneous or idiopathic osteonecrosis typically affects women older than 60 years. The ratio of affected women to men is three to one and the ratio of those affected older than 60 years to those younger than 60 years is four to one. Unlike atraumatic or secondary osteonecrosis, which more commonly presents bilaterally, spontaneous osteonecrosis is found bilaterally in less than 20% of cases (9,10).

Spontaneous osteonecrosis most commonly affects the weightbearing portion of the medial femoral condyle followed by the lateral femoral condyle. It is only rarely seen in the periphery or intercondylar notch region (10,11). Spontaneous osteonecrosis also occurs in the tibial plateau where it is more common in the medial plateau mimicking a medial meniscal tear (12). Spontaneous osteonecrosis of the patella occurs rarely (13).

Other than being female and aged more than 60 years, there are few other known risk factors associated with spontaneous AVN, although obesity has been found in 60% of cases (15).

Despite the preponderance of AVN of the medial femoral condyle, there is no association with varus alignment (14).

## Presentation

Patients with spontaneous AVN will often complain of acute onset of severe pain, with only 10% of patients reporting a history of trauma. The pain is located over the involved condyle, most commonly over the medial aspect of the knee. Pain in the acute phase is often reported as being worse at night and can last 6 to 8 weeks (10). Mechanical symptoms are rare unless the articular cartilage surface is unstable and fragmented, but patients may present with severe muscle spasm, giving the knee a locked appearance. After the acute phase, the pain typically dissipates, but in some cases, pain may become chronic.

On physical examination, effusions are uncommon, and knee range of motion is usually preserved, being limited only by muscle spasm when present. Pain is elicited with palpation over the medial femoral condyle. This finding can often mislead the examiner into thinking that the patient has a degenerative meniscal tear—a much more common diagnosis in patients of this age group.

## Basic Science

The vascular supply in osteonecrotic bone is compromised by one of four mechanisms (Table 1). The most common known etiologies of atraumatic or secondary osteonecrosis are associated with alcohol or corticosteroid use. The mechanism by which alcohol or corticosteroid use causes AVN is unknown, although it is hypothesized that fat emboli from a fatty liver or from the bone marrow can occlude the arterial vessels (17).

The etiology behind spontaneous osteonecrosis is unknown, but theories are based on the same pathophysiology as idiopathic osteonecrosis of the femoral head (16,17). Two hypotheses have been espoused. The vascular hypothesis proposes that injury to the tenuous blood supply of the subchondral bone in the femoral condyle leads to ischemia and bone edema. This edema leads to increased intraosseous pressure, leading to worsening ischemia. As the ischemia advances, the bone collapses. With revascularization of the subchondral bone, the symptoms from osteonecrosis abate.

The traumatic hypothesis is based on the fact that most of these cases occur in women older than 60 years. This population has a high incidence of osteoporosis, and it is thought that microfractures in the subchondral bone lead to an influx of synovial fluid into the marrow cavity, which elevates the intraosseous pressure, resulting in osteonecrosis.

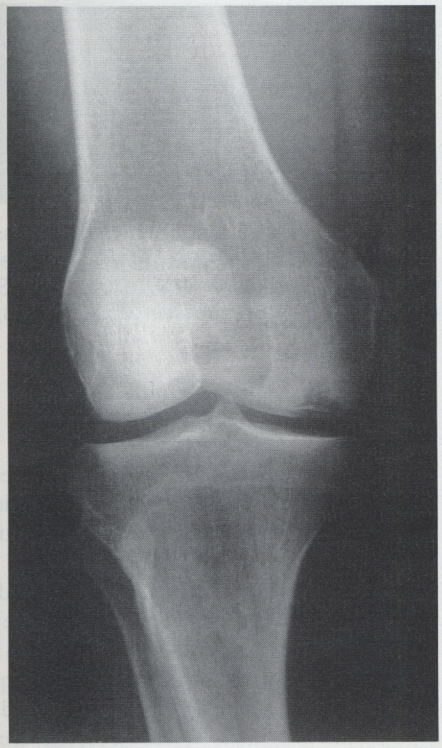
A third etiologic category of osteonecrosis can be classified as iatrogenic. For example, there are anecdotal reports of osteonecrosis developing after posterior cruciate ligament reconstruction due to the proximity of the bone tunnel to the articular surface of the medial femoral condyle. Similarly, there is significant overlap between the presentation of osteonecrosis and meniscal pathology. Although it is the author's opinion that many of these cases represent cases of unrecognized early-stage osteonecrosis diagnosed as meniscal tears and chondral injuries, the literature is replete with cases of AVN after partial meniscectomies and chondroplasty. For example, there are at least three studies in the literature representing 24 patients who were reported to develop AVN after arthroscopic partial meniscectomies (18–20). It is thought that without the protective effect of the meniscus, the condyle is subjected to a greater amount of force, resulting in micro fractures, edema, and ischemia (Fig. 1A). However, the average age of the patients in these studies was 65 years old, and the majority of the patients were women. It is contended that this population had preexisting spontaneous osteonecrosis before their partial meniscectomies. It is impossible to determine whether the arthroscopic surgery may have caused the osteonecrosis or simply exacerbated that which was present before surgery.

Other authors have reported the development of AVN after arthroscopic laser meniscectomy (21–21b). These case reports suggest that the damage to the subchondral bone from the energy of the laser precipitated osteonecrosis. Although this might be true, another study showed that when the laser is used at appropriate settings, the risk of AVN is no higher than the risk associated with routine arthroscopic instrumentation (22).

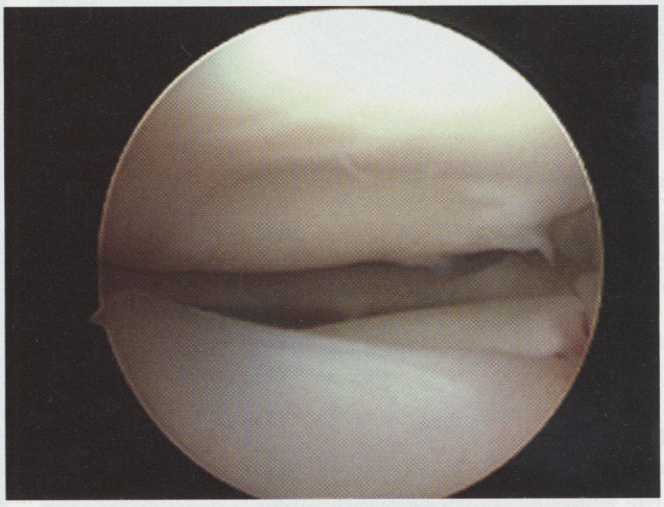
Recently, with the advent of monopolar and bipolar radiofrequency devices used to perform thermal chondroplasty, there has been increasing concern that some of these devices may actually lead to thermal injury to the subchondral bone. Non–temperature-controlled bipolar radiofrequency devices may cause full thickness chondrocytes death to the level of the subchondral bone, but a direct cause and effect relationship remains to be established (Fig. 2) (23,24).

**TABLE 1.** *Etiologies of vascular injury in osteonecrosis*

| |
|---|
| Mechanical disruption of vessels |
| Fractures |
| Dislocation |
| Stress fracture |
| Occlusion of arterial vessels |
| Circulating fat |
| Alcohol use |
| Corticosteroid use |
| Nitrogen bubbles |
| Caisson worker's disease |
| Abnormal cells |
| Sickle cell disease |
| Injury/pressure on arterial walls |
| Intimal damage |
| Vasculitis |
| Radiation exposure |
| Angiospasm |
| External pressure |
| Osteomyelitis—inflammatory cells |
| Gaucher's disease—macrophages filled with cerebroside |
| Alcohol use—hypertrophied fat cells |
| Occlusion to venous outflow |
| Venous pressure exceeds arterial pressure |
| Osteomyelitis |
| Corticosteroid use |
| Alcohol use |



A



B

**FIG. 1.** Avascular necrosis of the medial femoral condyle developing within 4 months of a simple posterior horn medial meniscectomy in a 48-year-old male. **A:** Routine weightbearing anteroposterior radiograph demonstrating focus of avascular necrosis. **B:** Arthroscopic evaluation in the same patient demonstrating softening over area of collapsed subchondral bone.

The pathologic changes of osteonecrosis of the knee parallel those changes seen in the hip. Early in the disease process, the gross appearance of the articular cartilage may be normal with slight discoloration and softening appreciated by gentle probing (Fig. 1B). The chondrocytes undergo cloning, and there is fibrillation of the cartilage with thickening of the tidemark (Fig. 3). With disease progression, a flap of articular cartilage develops over the area of necrotic bone. With further progression, the defect left behind by the cartilage flap is filled by fibrocartilage. Microscopic examination shows changes typical of osteonecrosis with empty

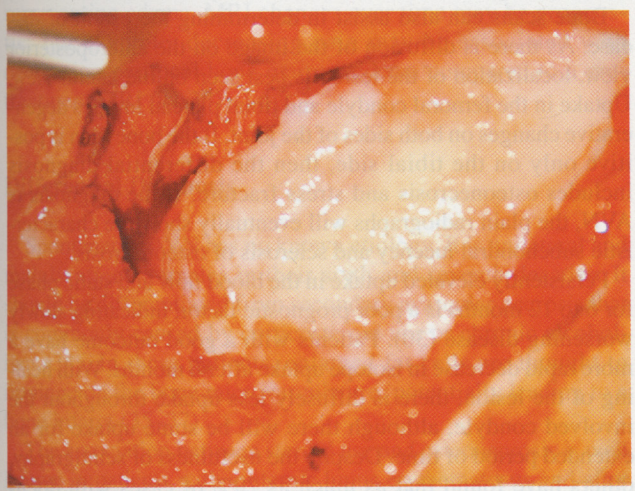


**FIG. 2.** Avascular necrosis of the medial femoral condyle after thermal chondroplasty, using a bipolar radiofrequency device used to treat a grade III lesion of localized chondromalacia.

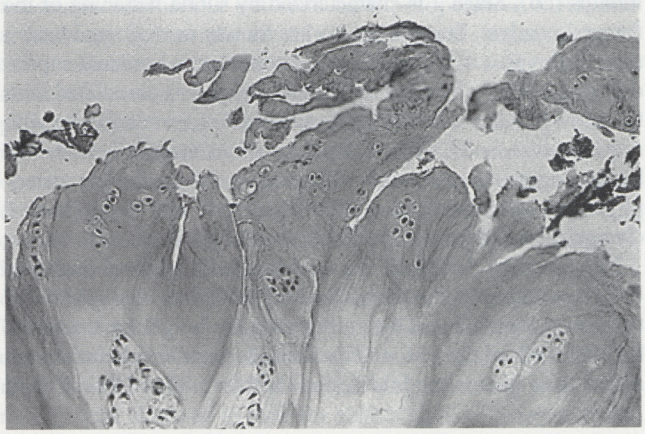


**FIG. 3.** Fibrillation of the articular cartilage and chondrocytes cloning. (From Mankin HJ, Mow VC, Buckwalter JA. Articular cartilage repair and osteoarthritis. In: Buckwalter JA, Einhorn TA, Simon SR, eds. *Orthopaedic basic science, biology and biomechanics of the musculoskeletal system*, 2nd ed. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2000:478, with permission.)

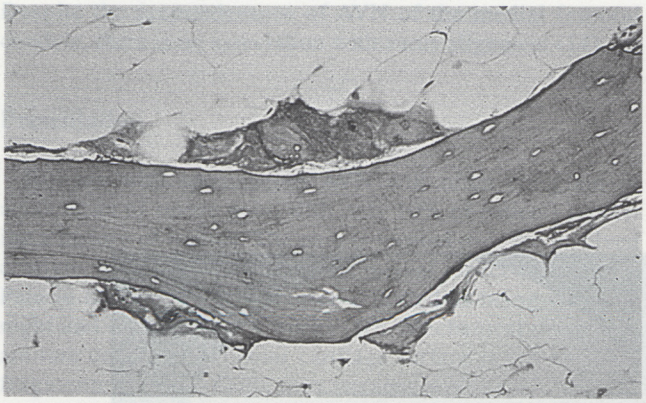


**FIG. 4.** Osteonecrotic bone with osteoblastic rimming. (From Day SM, Ostrum RF, Chao EYS, et al. Bone injury, regeneration, and repair. In: Buckwalter JA, Einhorn TA, Simon SR, eds. *Orthopaedic basic science, biology and biomechanics of the musculoskeletal system*, 2nd ed. Rosemont, IL: American Academy of Orthopaedic Surgeons, 2000:374, with permission.)

osteocyte lacunae and fatty degeneration of the marrow. Dead bony trabeculae are surrounded by osteoblastic activity, cartilage formation, and fibrovascular granulation tissue (Fig. 4).

### Imaging Studies and Staging Systems

Imaging studies are necessary to make the diagnosis of AVN. There are five plain radiographic stages (15,25). Stage 1 is determined by a normal radiographic study with the diagnosis being made on bone scan. Fifty percent of patients can present with normal x-rays, which, together with the medial sided joint pain on physical exam, often leads to a mistaken diagnosis of a symptomatic degenerative meniscus tear. Of the patients with normal x-rays, 70% will develop radiographic findings over the course of 7 months (26). Stage 2 is characterized by subtle flattening of the femoral condyle. Stage 3 lesions are frankly necrotic and have a radiolucent area in the subchondral bone that is bordered superiorly and laterally by a sclerotic halo. Stage 4 is associated with thickening of the sclerotic halo, subchondral bone collapse with sequestration, and cartilage flap formation. Stage 5 heralds full blown degenerative changes with joint space narrowing, osteophyte formation on the affected condyle, and degenerative changes in the tibial plateau (Table 2 and Fig. 5).

The extent of the lesion can be determined by three methods. Using radiographs corrected for magnification, the volume of the lesion is calculated by multiplying the height by the width by the length of the lesion (27). Small lesions are characterized as less than 10 $cm^3$, medium lesions between 10 and 20 $cm^3$, and

**TABLE 2.** *Radiographic stages of avascular necrosis*

| | |
|---|---|
| Stage I | Normal radiographs, bone scan positive |
| Stage II | Flattening of condyle |
| Stage III | Radiolucency with halo of sclerotic bone |
| Stage IV | Calcified sequestrum |
| Stage V | Narrowing of joint space |

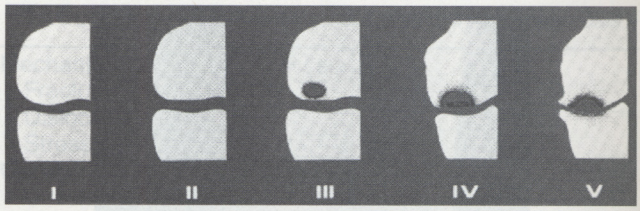



**FIG. 5.** Schema of the five stages of osteonecrosis. (From Insall JN, Aglietti P, Bullough PG, et al. Osteonecrosis. In: Insall JN, Windsor RE, Scott WN, et al., eds. *Surgery of the knee*. New York: Churchill Livingstone, 1993:616, with permission.)

large lesions more than 20 $cm^3$ (28). The combined necrotic angle is another method to determine the size of the lesion (28). On the anteroposterior view, a line is drawn tangentially along the medial and lateral border of the lesion (Fig. 6). The angle that is formed by the intersection of these lines is added to the angle measured on the lateral view. Small lesions are defined as those when the sum is less than 150 degrees, medium if between 150 and 250 degrees, and large if greater than or equal to 250 degrees (28). The percentage of the condyle involved also gives an indication of the prognosis. The larger the percent of the condyle involved, the worse the prognosis. The percentage involved is measured on plain films by dividing the width of the condyle by the width of the lesion (29) (Fig. 7).

Scintigraphy is useful to make the diagnosis of osteonecrosis, especially in the face of normal radiographs (Fig. 8) (9). Technetium-99m phosphate compounds are effective as the isotope adheres to hydroxyapatite crystals, immature collagen, alkaline phosphatase, and calcium phosphate. A three-phase scan is recommended with blood flow, blood pool, and delayed images, providing an accurate diagnosis of early osteonecrosis (30). Early in the disease, the blood flow phase shows hyperemia. Focally intense uptake is seen in the affected condyle on the delayed images, also known as the *static phase*.

Bone scans can also assist in determining the extent of disease. As there is diffusion of the image on the anteroposterior view, localization of the lesion is best seen on the lateral view. Uptake in the femoral condyle and tibial plateau implies degenerative changes on both sides of the joint. If the bone scan is positive only on the tibial side, then the differential diagnosis includes osteonecrosis and stress fracture. If the diagnosis is AVN, the plain radiographs will eventually show the characteristic subchondral lucency and sclerosis of osteonecrosis.

Persistently elevated uptake in the blood flow and static phase for 6 to 12 months is a poor prognostic factor, indicating slowly worsening disease (30). Patients with a good outcome have a more rapid decrease in isotope uptake after 6 months. With healing of the lesion, there is diffusely less uptake throughout the involved condyle, although there will be some uptake for an extended period of time. Despite the usefulness of scintigraphy, others have found that as much as 29% of cases with documented AVN can have negative bone scans (28).

The role of magnetic resonance imaging (MRI) in osteonecrosis has not been fully delineated. Some studies report that MRI is not particularly sensitive with respect to early AVN (9). False-negative reports of 25% on T2 and 12.5% on T1 have been

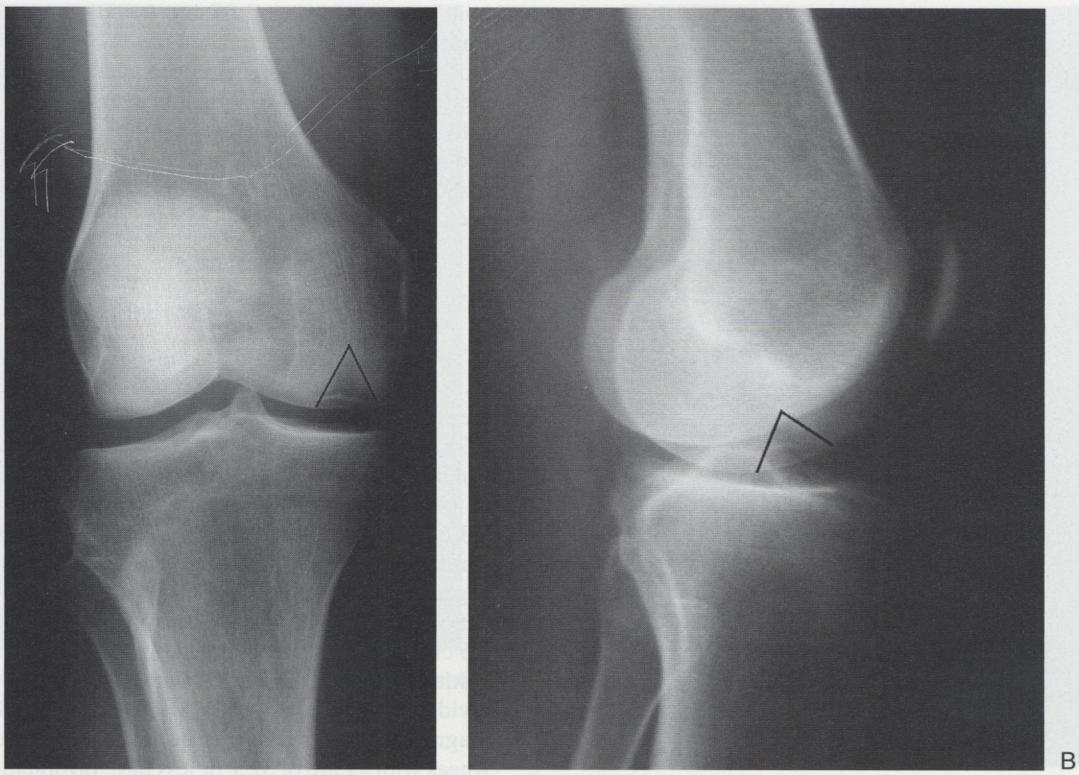



**FIG. 6.** The combined necrotic angle in osteonecrosis measured with tangential lines drawn to the edges of the defect on both the anteroposterior **(A)** and lateral **(B)** radiographs.

documented in the literature (31). Other authors support the use of MRI, stating that it is better than scintigraphy by being noninvasive and more sensitive (32).

When MRI does detect AVN, the T1 and T2 images have distinct findings. On the T1 images, the bright signal of the marrow is replaced with a subchondral area of low signal intensity (Fig. 9). On the T2 sequences, there is an area of low signal corresponding to the lesion surrounded by a high signal representing edema. MRI changes over time may be prognostic. For example, decreasing pain may be associated with decreasing perifocal bone marrow edema compared to a pretreatment MRI (31,33).

## Differential Diagnosis

Spontaneous AVN has no known etiology. It is unilateral and mainly affects females in their sixth and seventh decades. It should be differentiated from secondary avascular necrosis that is commonly caused by corticosteroid use or chronic alcoholism (34). In these patients, enlarged fat cells are thought to impinge on the bone microvasculature, potentially leading to a bone infarct (35–37).

The association between steroid use and osteonecrosis is well known in patients with systemic lupus erythematosus and after renal transplantation. The prevalence of osteonecrosis in these patients has been reported as high as 8% to 12% (38,39).

Unlike spontaneous osteonecrosis, secondary osteonecrosis can affect any age, any population, and is frequently asymptomatic. It may affect other joints such as the hip and shoulder as well. Secondary osteonecrosis of the knee tends to be bilateral and involves the lateral femoral condyle more often than spontaneous osteonecrosis (9).

A diagnosis of medial meniscus pathology is the most common alternative diagnosis to spontaneous osteonecrosis. Similarities between the two include acute onset of pain, mild effusion, and locking of the knee joint. There are cases in which avascular necrosis is not seen on a preoperative MRI, and arthroscopy is recommended. In these clinical situations, a partial medial meniscectomy is performed for a degenerative meniscus tear—a common finding in this age group (40). If the patient's symptoms worsen postoperatively, possibly due to increased tibiofemoral contact force after the meniscectomy, one should be suspicious for the development of osteonecrosis (Fig. 1) (41). The conclusion that partial meniscectomy has caused osteonecrosis, however, must be made with caution, as it is more likely that early phase (i.e., pre-radiographic) osteonecrosis was present at the time of the meniscectomy (31). In cases in which the diagnosis is in doubt, a bone scan or MRI can prove useful to help differentiate these entities. Initial treatment is observation with protected weightbearing with serial radiographs obtained to look for classic signs of osteonecrosis.

Symptomatic osteoarthritis is differentiated from spontaneous osteonecrosis by the temporal pattern of clinical deterioration. Typically, osteoarthritis has a gradual onset with a slow progression. Alternatively, osteoarthritis associated with spontaneous osteonecrosis is associated with an acute onset and rapid deterioration (10). Imaging studies can be useful in differentiating osteoarthrosis from osteonecrosis. Radiographically, joint space narrowing without the classic findings of

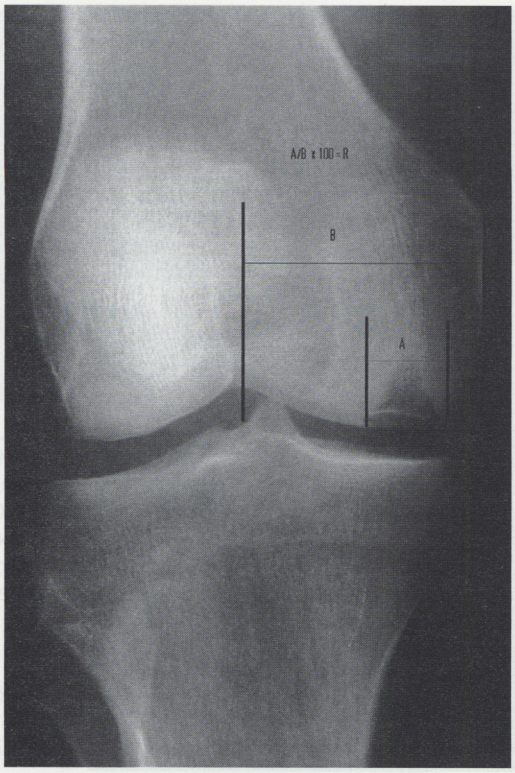



**FIG. 7.** The percentage of the femoral condyle involved can be measured by dividing the width of the condyle **(B)** by the width of the lesion **(A)** on the anteroposterior radiograph.

osteonecrosis is seen with osteoarthritis. Bone scans in osteoarthritic patients demonstrate bipolar uptake, as opposed to unipolar uptake, in osteonecrosis. Additionally, uptake is rarely as intense as is seen in patients with osteonecrosis (42).

Other conditions confused with osteonecrosis include hip osteoarthrosis, pes anserine bursitis, and deep venous thrombosis. With hip osteoarthrosis, referred pain to the medial side of the knee may be the presenting complaint. Hip radiographs, selective injections, and a bone scan help to differentiate this from osteonecrosis of the knee. With pes anserine bursitis, acute medial-sided knee pain is seen below the joint line at the hamstring insertion. Physical examination and selective injections are diagnostic.

Osteonecrosis can present with radiating leg pain distally and posteriorly, potentially mimicking a deep venous thrombosis. In one study, 3 of 40 patients were misdiagnosed as having a deep venous thrombosis as evidenced by negative venograms (26).

Osteochondritis dissecans (OCD) is also included in the differential diagnosis of osteonecrosis. Whereas spontaneous osteonecrosis is most common in women older than 60 years, OCD occurs between the ages of 10 and 50 years and males are 2 to 3 times more frequently affected. Osteonecrosis mostly affects the weightbearing zone of the medial femoral condyle, and OCD classically involves the lateral aspect of the medial femoral condyle (43,44). At least one-half of patients with OCD report a history of trauma—an uncommon finding in patients with osteonecrosis (43,44). Pain tends to be insidious in OCD with delayed osteoarthritic change, as opposed to the acute onset of pain with osteonecrosis associated with expeditious joint space narrowing (1,27,42). Radiographically, OCD rarely leads to collapse of the femoral condyle. Loose bodies are common with OCD and rare with AVN. On bone scan, OCD lesions are typically cold in contrast to osteonecrosis (45). Histologically, OCD has a fibrocartilaginous layer between the fragment and the condyle, whereas osteonecrosis has fibrovascular granulation tissue. Necrosis is rare in OCD unless the fragment has been chronically detached (46).

## Prognosis

The natural history and prognosis of spontaneous osteonecrosis is predicated by lesion area, the ratio of the lesion's size to the condyle involved, and the lesion's stage. Muheim and Bohne (27) determined that smaller lesions (average size of 2.4 $cm^2$) had a more favorable outcome than larger lesions (average size of 10.4 $cm^2$). Other studies have shown that lesions averaging 2.3 $cm^2$ or less did well when treated conservatively, whereas similarly treated lesions averaging 3.7 $cm^2$ or more developed degenerative changes (42). Based on these studies, recommendations include nonoperative treatment for lesions smaller than 3.5 $cm^2$ and operative treatment for those larger than 5.0 $cm^2$.

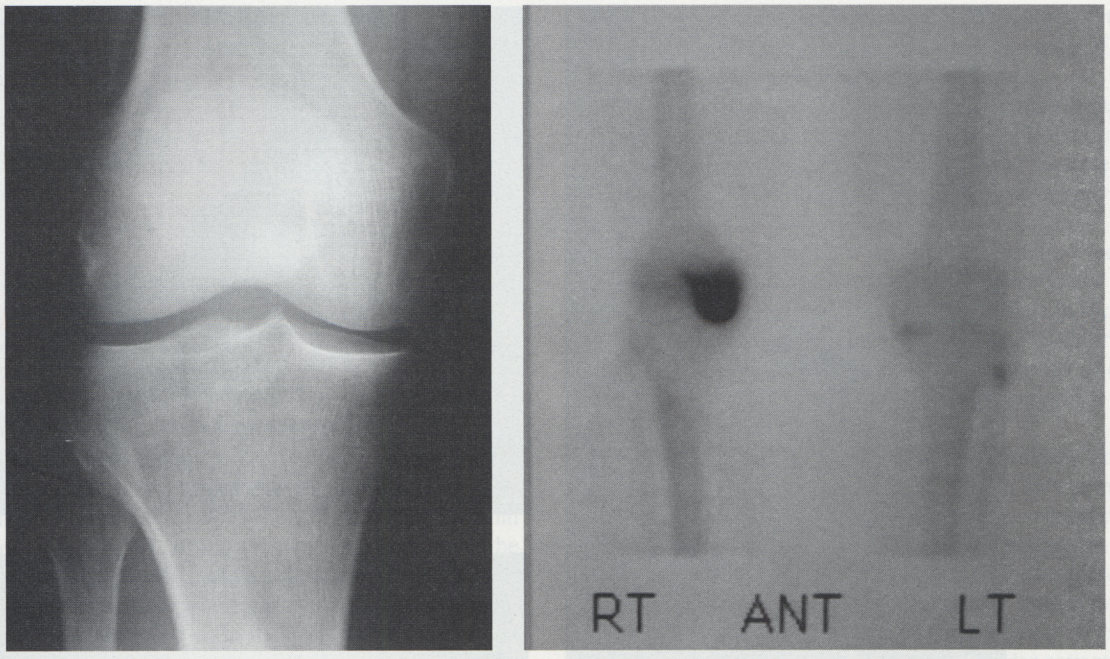
Measuring the medial to lateral diameter of the lesion and dividing this number by the width of the condyle eliminates magnification error and is believed to be prognostic (29). Knees with a ratio of 32% or less have favorable outcomes, and knees with a ratio of 57% or more have poor outcomes. A ratio of 50% has been defined as the borderline between good and bad outcomes (29).

Similarly, but less reliably, the earlier the stage of the lesion at the time of diagnosis, the better the prognosis. In particular, defining stage III lesions has made this method difficult in practice (47).

## Treatment

Nonoperative treatment for osteonecrosis is reserved mainly for stages I and II. These patients have normal radiographs or slight flattening of the involved condyle. Lesions less than 3.5 $cm^2$ are considered small and also do well with conservative care. Nonoperative treatments, including protected weightbearing with the use of a cane, analgesics such as acetaminophen, and quadriceps rehabilitation, are important. This regimen leads to 70% to 90% good to excellent results with small lesions (15,29,42). However, a recent long-term study noted that only 20% of initially symptomatic knees treated nonoperatively had a successful clinical outcome at 8 years (28). Roughly 30% of the osteonecrotic lesions remained the same, 30% enlarged, and 30% decreased in size. Although degenerative changes are eventually seen in most cases, they are typically less severe with smaller lesions (10).

Core decompression is used in patients who fail conservative treatment and who have lesions that are stage III or less. At 7 years' follow-up, 70% of patients who underwent a single core decompression had a good or excellent result (28). Like the hip, a guidewire is inserted into the osteonecrotic bone under fluoroscopic control. A cannulated 8-mm reamer is then used to decompress the bone. Other studies demonstrate deteriorating results with advancing stages of the lesion. Jacobs et al. (48) demonstrated that, although more than 90% of patients with stage I or II had good results, only 50% of patients with stage III had good results.




A B

**FIG. 8. A:** Normal radiograph in a patient suspected of having osteonecrosis, with evidence on technetium bone scan **(B)**. ANT, anterior; LT, left; RT, right.

Patients who fail core decompression can be treated several ways. When the continued pain is thought to be primarily from the bone, repeat core decompression can be performed, but results decrease to 60% good to excellent (28). Arthroscopy is indicated when pain is believed to be articular in origin and is ameliorated with an intraarticular corticosteroid injection. At 3 years, 60% of arthroscopically treated patients who failed core decompressions had satisfactory results (28). Arthroscopic treatment alone for stage III or worse osteonecrosis has not been shown to change the natural history of the disease (9). A total or unicompartmental knee arthroplasty is a good option in patients who fail core decompression (48). Rarely, young patients with localized osteonecrosis and normal alignment, may be successfully treated with fresh osteochondral allograft reconstruction (Fig. 10).

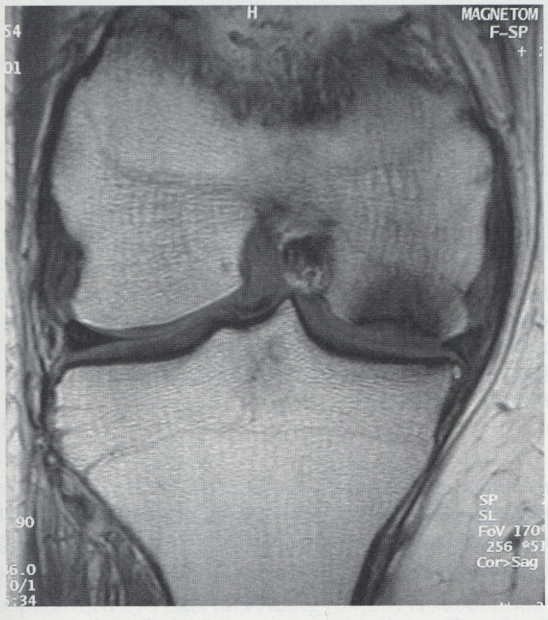



**FIG. 9.** A T1-weighted magnetic resonance image demonstrating a well-circumscribed lesion of avascular necrosis of the medial femoral condyle, with decreased signal intensity within the subchondral bone marrow.

Osteotomy performed with concomitant arthroscopic débridement of the necrotic lesion may be successful (42, 47). Although the realignment osteotomy is not recommended in patients with a normal mechanical axis, it has been used when the standing tibiofemoral angle is in 1 to 5 degrees of valgus. The goal of surgery is to correct the anatomic axis to 6 to 15 degrees of valgus. With osteonecrosis of the lateral femoral condyle, the tibiofemoral angle should be corrected to 4 degrees of varus. Good results have also been seen in patients with concomitant drilling and bone grafting at the time of osteotomy (47).

When osteonecrosis has advanced to the stage of collapse, particularly when associated with advanced patient age, low physical demand, ligamentous instability, flexion contractures, and synovitis, total knee replacement is the treatment of choice. Overall, total knee replacements provide 85% to 95% good to excellent results at 10 years of follow-up (49,50). Results are not as good for patients who have secondary or steroid-induced osteonecrosis. Only 55% of 31 knee replacements had good to excellent results at an average follow-up of 8 years in this later population (7). Others have reported an 84% survivorship of the prostheses at 5 years (51).

Unicompartmental arthroplasty is also an option in patients whose disease is limited to one compartment. In short-term studies, results have been very encouraging with a low incidence of loosening or revision and a high rate of pain relief (52). Unicompartmental arthroplasty allows patients to maintain

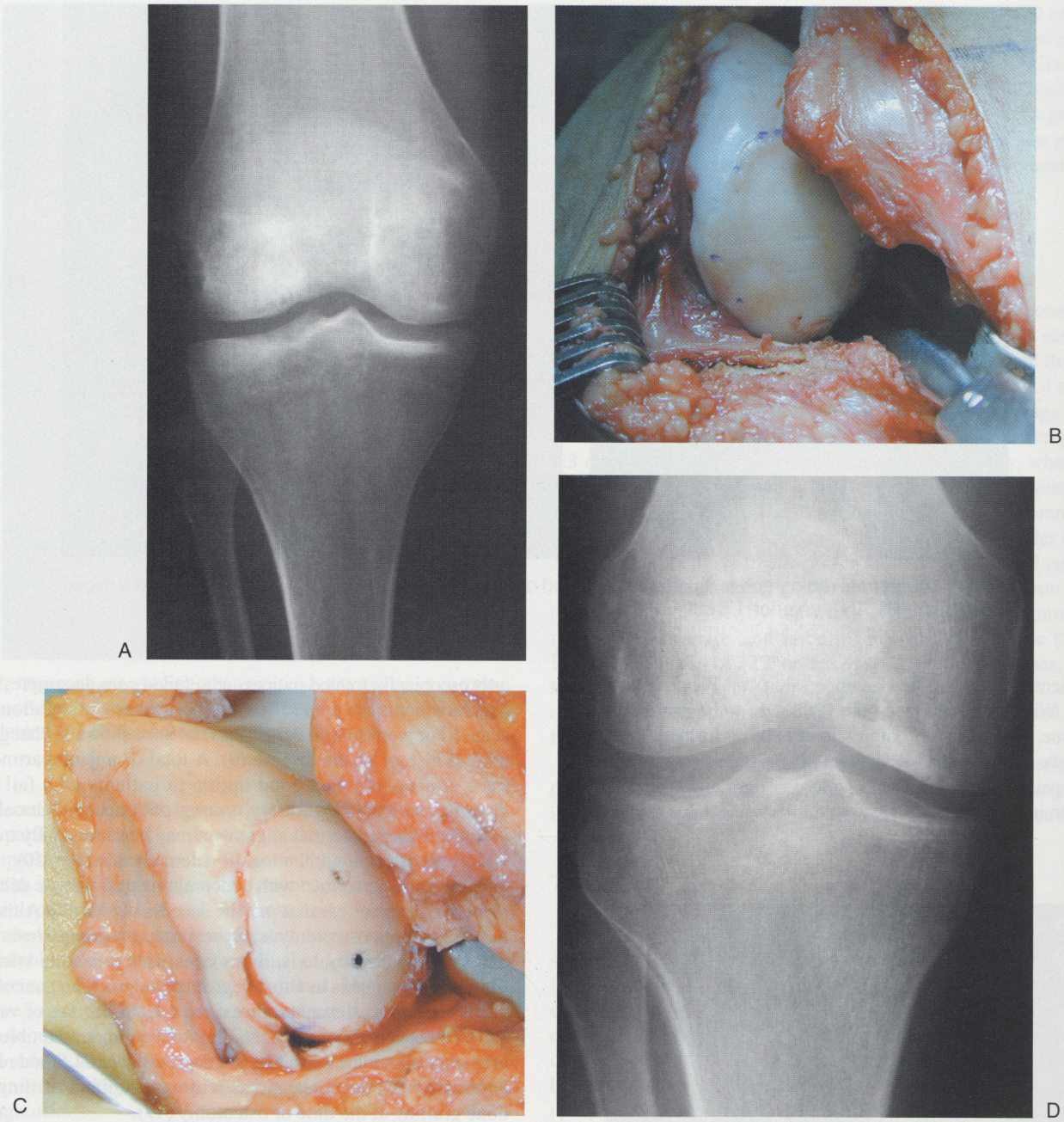



**FIG. 10.** Clinical example of an 18-year-old woman with localized steroid-induced avascular necrosis of the medial femoral condyle with anteroposterior radiograph demonstrating minimal flattening of the medial femoral condyle **(A)**. **B:** Intraoperative photograph of softened articular surface consistent with subchondral collapse treated with fresh osteochondral shell allograft **(C)**, and demonstrating excellent incorporation without collapse on her 12-month postoperative anteroposterior radiograph **(D)**.

excellent range of motion and proprioception without sacrificing the bone necessary to perform a total joint replacement.

## Conclusion

Spontaneous osteonecrosis is a relatively common cause of medial-sided knee pain in female patients in their sixth and seventh decades. The exact etiology is unknown and the prognosis seems predicated on the stage and size of the osteonecrotic segment. Nonoperative treatment is warranted for smaller lesions and consists of protected weightbearing and nonnarcotic pain medication. Operative treatment can include arthroscopy, core decompression, osteotomy, and arthroplasty. Frequently, patients in this age group have coexisting degenerative meniscal tears, and arthroscopic partial meniscectomies do not relieve the pain

caused by osteonecrosis. The physician should always think of osteonecrosis in patients with medial-sided knee pain in the autumnal years of life.

## OSTEOCHONDRITIS DISSECANS

### History

OCD is a condition in which an area of subchondral bone undergoes avascular change. Without spontaneous healing, the bone and adjacent cartilage fragments separate and become loose bodies. Although most commonly occurring in the knee, other target joints include the elbow, ankle, shoulder, and hip.

The history of OCD goes back many centuries. In 1558, Pare (53) described removing looses bodies from a joint. Paget (54) termed the condition *quiet necrosis* in 1870—a reference to ischemic changes seen in the subchondral bone with minimal clinical symptoms. In 1888, Konig (55) was the first to use the term *osteochondritis dissecans*, as he initially believed that the condition was an inflammatory disease.

Other sources for loose bodies in the knee joint include synovial chondromatosis, fractured osteophytes, meniscal fragments, and chondral or osteochondral fractures. Osteochondral injuries commonly result from patellar dislocations, pivot-shifting injuries, or direct blows. Morscher (56) reported that the majority of loose bodies from fractured articular surfaces were the result of patellar dislocations, in which the medial edge of the patella or the lateral femoral trochlea shears off.

### Classification

OCD remains a poorly understood entity, and there is no consistent definition in the literature. For that reason, authors often use the same term when describing different conditions. Dividing OCD into a juvenile- or adult-onset form is reasonable, because these variants have different prognostic implications. *Juvenile OCD* (JOCD) is defined as a lesion that occurs when the physes are open (57,58). Adult OCD has a poorer prognosis, probably due to the inherently limited ability to heal after physeal closure. This classification, however, fails to explain why many patients with OCD are young adults who report a history of knee pain in their youth (58). It is possible that OCD may develop during adolescence but remains clinically quiescent and symptoms do not manifest until after physeal closure. Other classification systems describe the lesion stability (intact, partially detached, fully detached, *in situ*, or loose) (59).

### Etiology

The etiology of OCD remains obscure, but many causes have been proposed, including trauma, vascular insult, genetics, endocrinopathies, hormonal abnormalities, ossification abnormalities, and multifactorial causes. The traumatic theory popularized by Paget and Konig (54,55) is subdivided into endogenous and exogenous trauma. *Endogenous trauma* refers to the medial tibial eminence creating a sheer stress against the lateral aspect of the medial femoral condyle with internal rotation of the lower leg (60). This mechanism is questionable, because the articular cartilage of the medial femoral condyle is generally normal in the region of the medial tibial spine (61).

*Exogenous trauma* is subdivided into microtraumatic and macrotraumatic causes. *Microtraumatic* implies repetitive stress over time on a particular area of the femoral condyle, leading to a subchondral stress fracture, resulting in loose body formation. *Macrotrauma* is supported by the fact that 25% to 50% of patients with OCD report a definitive traumatic event that may cause an osteochondral fracture (43). The traumatic etiology is supported by the fact that children who engage in sports at a relatively young age seem to be at increased risk. As girls and boys are becoming involved in sports at an earlier age, the incidence of JOCD in girls is increasing, and the average age of children with JOCD is decreasing (58).

Enneking (62) proposed a vascular etiology to OCD by acknowledging the tenuous blood supply to the epiphysis in rapidly growing individuals. The epiphyseal and secondary centers of ossification are supplied by the epiphyseal artery, which has numerous branches and arcades leading to end arterioles. Theoretically, a traumatic event may result in the interruption of blood supply to the epiphysis and secondary centers of ossification, resulting in an osteochondritic lesion. In older patients, repetitive trauma may cause vascular injury with impaired healing of a subchondral stress fracture, leading to fragment detachment.

It is possible that patients with JOCD may have a mild variant of an epiphyseal dysplasia. Multiple epiphyseal dysplasia is an autosomal dominant disease resulting in a deficient proliferative layer of endochondral ossification. The severe Fairbanks type can lead to angular deformities, and the milder ribbing type results in the formation of multiple osteochondritic lesions. Because JOCD is often bilateral, it is possible that it is a variant of normal growth, and its expression is determined by an individual's genetic makeup.

OCD has also been associated with endocrinopathic conditions leading to fat emboli or thrombosis of the end arterioles in the subchondral bone. Ligamentous laxity has also been associated with JOCD. Adolescents with angular deformities, such as genu varum, that result in stress accumulation on the femoral condyles are at risk for JOCD (58).

### Presentation

OCD usually presents in the 10- to 20-year-old age group but can be seen in patients up to the age of 50 years. It has an incidence of 3 to 6 per 100,000 individuals. Males are three times more commonly affected than females, although, with the recent increase in female sports participation, the ratio of males to females affected with OCD may be approaching 2:1. The medial femoral condyle is affected three times more often than the lateral femoral condyle (Fig. 11). OCD has also been described on the inferior aspect of the patella (63,64). In JOCD, the incidence of bilaterality is 30% to 40%, whereas the adult form is bilateral in only 5% of cases (65).

Patients without a recent history of trauma present with a vague, poorly localized, aching pain, typically of several months duration. They also complain of stiffness and activity-related swelling. Symptoms typically worsen with increasing activity as stable lesions detach, causing catching and locking. Physical findings may be confused with patellofemoral pain, as

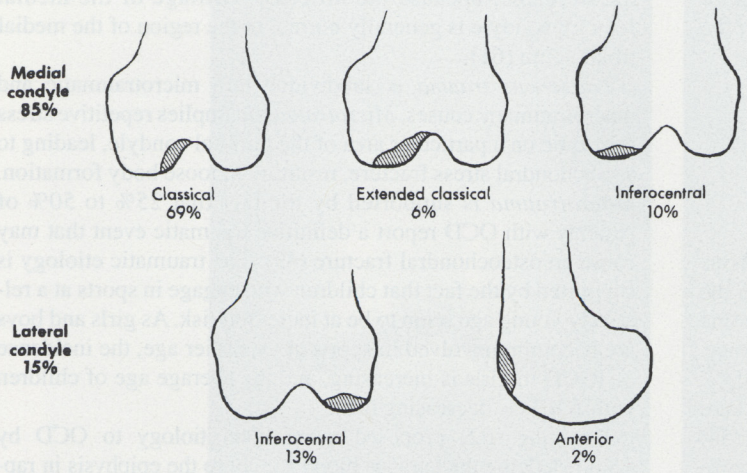



**FIG. 11.** Distribution of osteochondritis dissecans. (From Sisk TD. Knee injuries. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*, Vol. 3, 7th ed. St. Louis: C.V. Mosby, 1987:2470, with permission.)

the patient is often tender to palpation along the femoral condyle near the trochlea. Decreased range of motion and effusions are common. When symptomatic, patients often have quadriceps atrophy and walk with an antalgic gait. With lesions on the lateral aspect of the medial femoral condyle, patients may walk with the tibia externally rotated to minimize the chance of hitting the involved area with the medial tibial spine.

Wilson's sign is a physical examination test that is specific for these medial femoral condyle lesions (66). The knee is flexed to 90 degrees with the tibia internally rotated. The knee is then slowly extended. A positive test is defined by recreating the patient's symptoms with the knee in 30 degrees of flexion as the tibial spine abuts the medial femoral condyle. Externally rotating the tibia should alleviate the discomfort.

## Imaging Studies and Staging Systems

The clinical benefit of classifying OCD lesions to guide treatment recommendations is questionable, but they do gen-

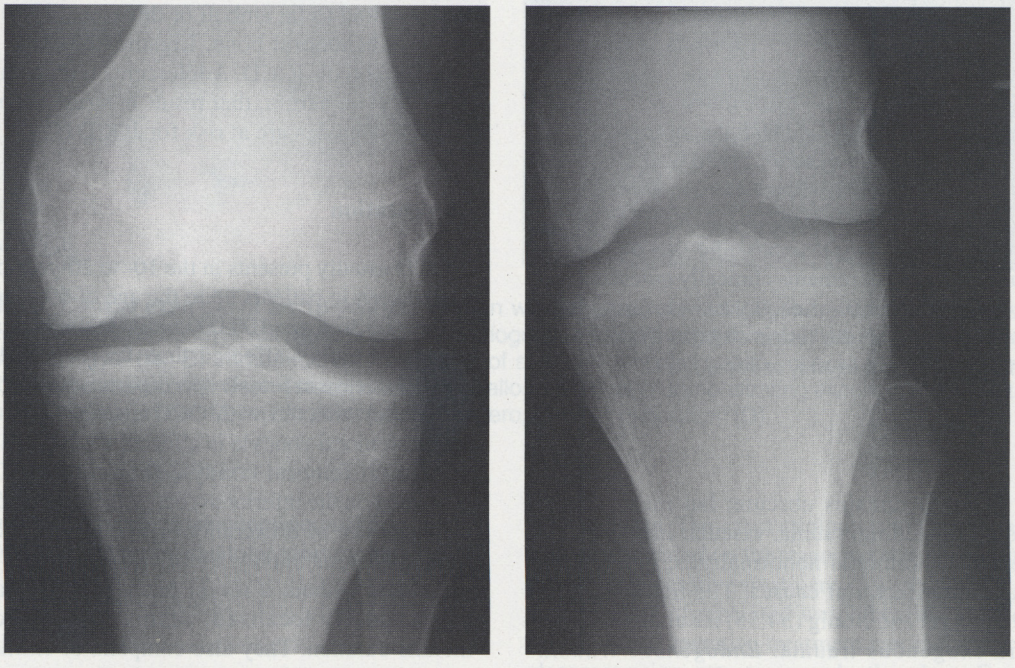



**FIG. 12.** Chronic osteochondritis dissecans of the lateral aspect of the medial femoral condyle in a 28-year-old male barely visualized in extension weightbearing radiograph **(A)** and imaged more dramatically and in a 45-degree posteroanterior weightbearing radiograph **(B)**.

erally emphasize the importance of mapping the lesion's location with diagnostic imaging. Plain radiographs are usually diagnostic. Views that should be obtained include the weightbearing anteroposterior view with the patient's body weight evenly distributed on both legs, 45-degree nonweightbearing lateral view, a merchant view, and a 45-degree flexion weightbearing posteroanterior view. This latter view is possibly the most helpful in diagnosing the classic lesion on the posterolateral aspect of the medial femoral condyle, which can be missed on the anteroposterior view (Fig. 12). Adolescents should always have their contralateral limb radiographed to detect bilateral lesions (Fig. 13). Caffey et al. (67) believed that marginal irregularities with islands of bone in craters were at higher risk for developing OCD (67). If, despite normal x-rays, JOCD is still suspected, then a bone scan can detect the OCD in the symptomatic knee.

Berndt and Harty (68) developed the first radiographic system for OCD (for lesions in the talus), which is modified for the knee (Table 3). *Stage I lesions* are defined as not being visible on plain radiographs. *Stage II lesions* are defined as visible fragments that are still attached, presumably by the overlying articular cartilage. *Stage III lesions* are unattached lesions that are not displaced. *Stage IV lesions* are displaced fragments. The osteochondritic lesion is demonstrated as a well-circumscribed area of subchondral bone separated by a crescent-shaped radiolucent outline of the sclerotic fragment.

On lateral radiographs, Harding (69) noticed that 90% of medial lesions were situated in a specific zone of the condyle, defined by an area intersected by lines drawn through the posterior cortex of the femur and Blumensaat's line (Fig. 14). Garrett (70) reported that 75% of lateral condyle lesions were posterior to a line drawn along the posterior cortex of the femur.

The classification system with scintigraphy has not been found to be of prognostic value, but pinhole views can be useful to monitor lesion healing (58,71). There are five stages that have been defined by bone scan.

**TABLE 3.** *Radiographic stages of osteochondritis dissecans*

| | |
|---|---|
| Stage I | Not visible on x-ray |
| Stage II | Visible fragments still attached by overlying cartilage |
| Stage III | Nondisplaced, unattached fragments |
| Stage IV | Displaced fragments |

Stage 0: Negative bone scan with a negative radiograph.
Stage I: Negative bone scan with a lesion seen on plain radiographs. This represents an accessory center of ossification seen in normal joints or a healed lesion.
Stage II: Bone scan with increased uptake in the region of the lesion.
Stage III: Bone scan with increased uptake of the entire femoral condyle (Fig. 15).
Stage IV: Bone scan with uptake involving the corresponding articular surface consistent with bipolar arthritic change.

Most lesions present at stage III or IV. Activity levels are generally tolerated when the bone scan reverts to low-level uptake (i.e., stage I or II). Typically, patients with JOCD treated conservatively will improve one scintigraphic level over the course of 6 months and return to full competitive sports by 10 months (58,72).

MRI can confirm the extent of the lesion and the integrity of the underlying subchondral bone (Fig. 16). Standard spin-echo techniques combined with fat suppression improve contrast between cartilage and fluid. Ideally, articular cartilage is evaluated using a

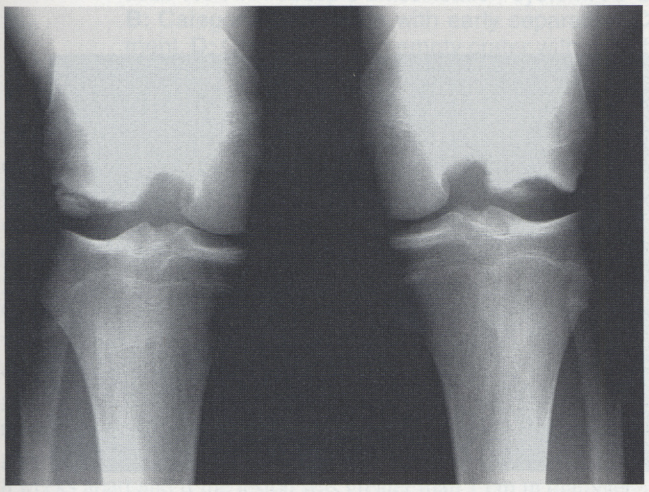


**FIG. 13.** Forty-five–degree posteroanterior weightbearing radiograph demonstrating bilateral osteochondritis dissecans of the lateral aspect of the lateral femoral condyles.

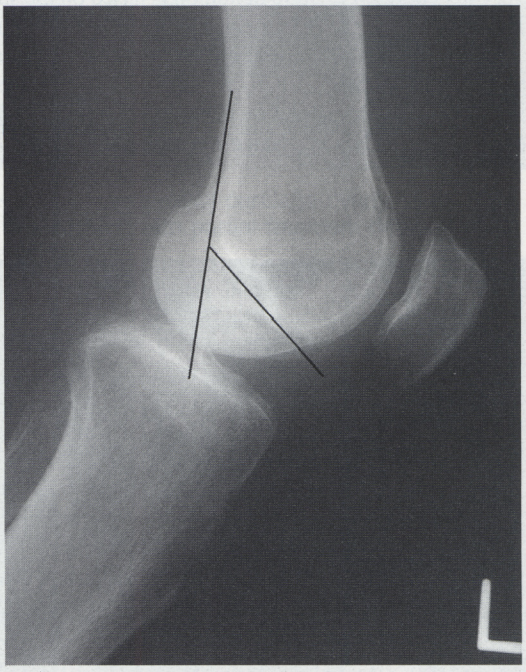


**FIG. 14.** Forty-five–degree flexion lateral view demonstrating classic area of osteochondritis dissecans lesion defined by an area intersected by lines drawn through the posterior cortex of the femur and Blumensaat's line.

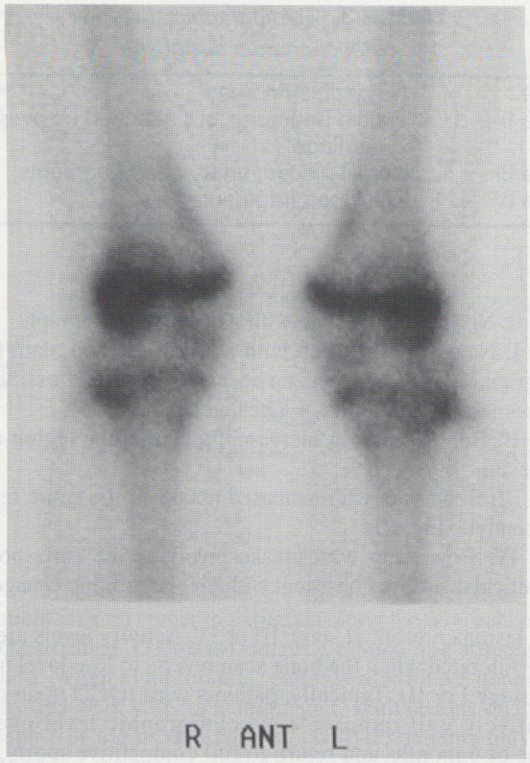



**FIG. 15.** Delayed-phase bone scan on same patient imaged in Figure 14, demonstrating bilateral intense localized uptake in the entire lateral femoral condyle.

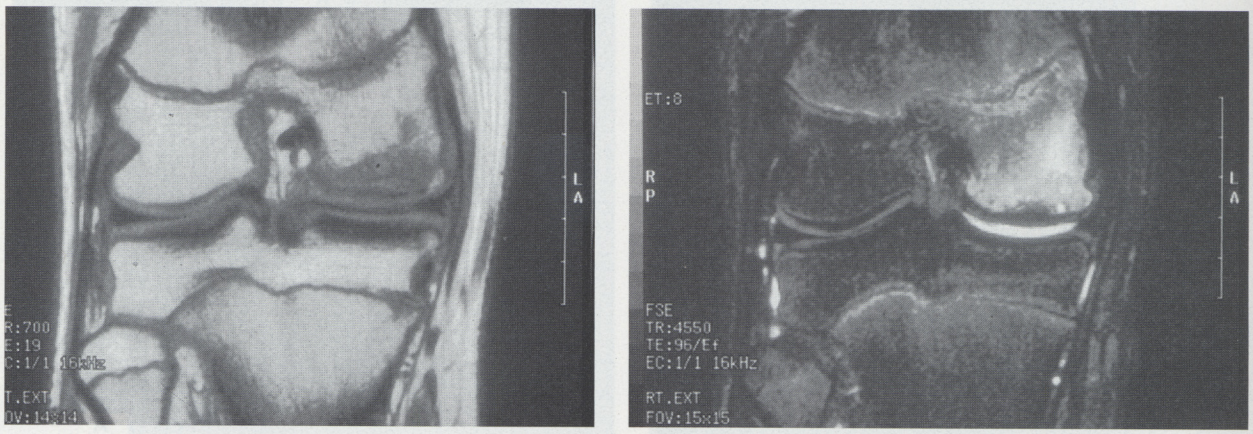
spoiled gradient echo sequence with fat suppression and three-dimensional acquisition. Differentiating OCD lesions from long-standing osteochondral fracture nonunion is based on the shape of the fragment. A loose OCD fragment is convex on both surfaces, whereas its corresponding crater is concave. An osteochondral fracture, on the other hand, usually has a flat fracture surface. The MRI staging system developed by Dipaola et al. (73) can also be used to follow lesion healing. Stage 1 is characterized by thickening of the articular cartilage and a decreased T2 signal. Stage 2 has a breach of the articular cartilage and a decreased signal consistent with fibrous tissue surrounding the OCD. Stage 3 is defined by a separation of the articular cartilage and increased signal on T2 behind the fragment consistent with synovial fluid. Stage 4 is a loose body.

Staging OCD is most accurately accomplished with arthroscopy. The surgeon must pay close attention to seemingly normal-appearing articular cartilage. Any depressions or a bluish-gray color in the articular surface typifies an intact OCD lesion. An arthroscopic classification system has been developed where category 1 is a lesion with intact cartilage overlying it. Category 2 lesions have early separation, and category 3 lesions are partially detached. Category 4 lesions are craters with or without a loose body (Fig. 17) (74).

## Natural History and Nonoperative Treatment

The natural history of OCD is primarily determined by patient age. In patients younger than the age of 15 years, the OCD lesion is firmly attached in 55%, in contrast to only 9% in patients older than 15 years (75). Pappas (76) developed a classification based on outcome. Girls younger than 11 years and boys younger than 13 years had the best outcomes, with healing in greater than 90% over the course of 3 to 6 months. On the other hand, girls and boys older than the age of 20 years had the worst prognosis. Treatment variables depend on patient age, lesion size, and lesion stability. Large fragments in the weight-bearing zone of the condyle tend to do poorly. Unstable fragments have the least chance of healing.

Nonoperative treatment is reserved for skeletally immature patients or those near maturity with stable lesions and is based on protective weightbearing. Casting is reserved for the noncompliant patient, because it leads to stiffness, atrophy, and cartilage degeneration and may actually decrease the healing potential of OCD (77). Knee braces can be used, but knee range of motion and strength must be maintained. After 6 to 8 weeks of protective weightbearing, the patient is allowed to resume full weightbearing,




A B

**FIG. 16. A:** T1-weighted sagittal image. **B:** T2-weighted sagittal magnetic resonance image of an unstable osteochondritis dissecans lesion of the medial femoral condyle, with fluid behind lesion consistent with an unstable fragment.

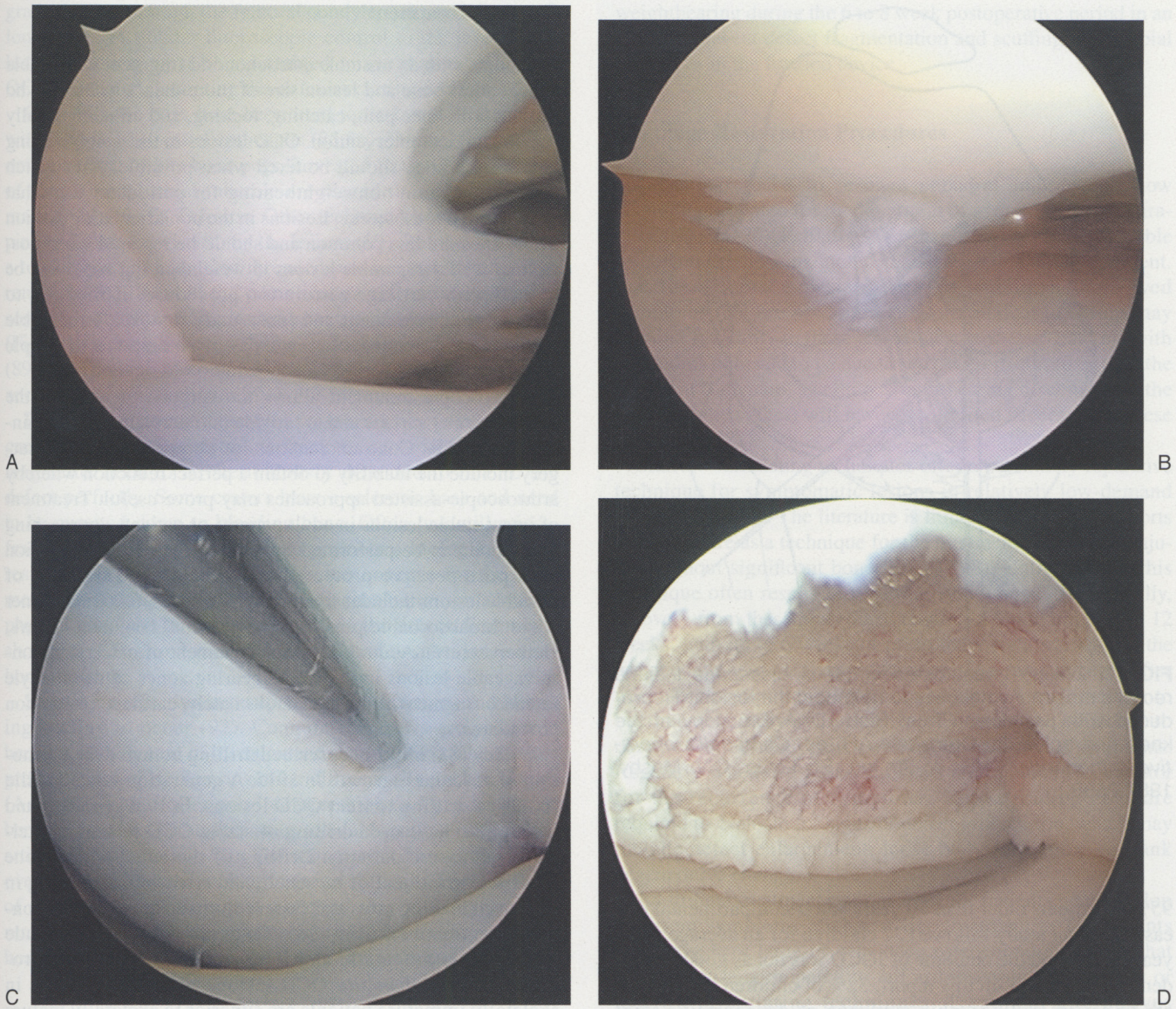



**FIG. 17.** Arthroscopic classification system. **A:** Category 1—a lesion with intact cartilage overlying it. **B:** Category 2—a lesion with early separation. **C:** Category 3—a lesion with partial fragment detachment. **D:** Category 4—an empty crater with or without a loose body.

assuming symptoms allow. Bicycling, swimming, and lower-extremity strengthening is advanced when the pain is gone and there is radiographic evidence of healing. Competitive sports are discouraged until complete radiographic healing occurs—a process that can take 4 to 12 months. Fifty percent of stable lesions in young compliant patients with open growth plates will heal in 10 to 18 months (72). Stable lesions in young adults can also be watched. It usually takes at least 4 months for these lesions to heal, and the patient is at risk for fragment displacement later. If radiographs are not conclusive in determining the healing process, then serial bone scans can be obtained. Guhl (74) recommended surgery if the OCD remained hot on consecutive scans taken at least 8 weeks apart and if the lesion was greater than 1 cm in diameter. These somewhat aggressive treatment recommendations are not necessarily followed by the authors.

Surgery is recommended if the lesion becomes detached or unstable, if the pain worsens in a compliant patient, if the patient is approaching epiphyseal closure, or if the bone scan does not improve. Surgery is also recommended in all symptomatic adults with OCD, even if they have stable lesions, because the risk of fragment degeneration and loose body formation is high, making a potentially reparable lesion irreparable.

Lesion healing is not predictive of which patients will later develop degenerative arthritis. Linden (57) reviewed 76 knees with an average follow-up of 33 years. The long-term results of JOCD were excellent with only 3 of 23 children developing mild arthritic changes. Of the 48 adults studied, there was a high incidence of loose bodies and chondrocalcinosis. There was a 20-year latency before degenerative changes of the joint appeared. At age 40 years, none of the patients had radiographic evidence of degenerative joint disease. By age 50 years, 70% of the patients with adult-onset OCD lesions had radiographic evidence of tricompartmental disease. More than

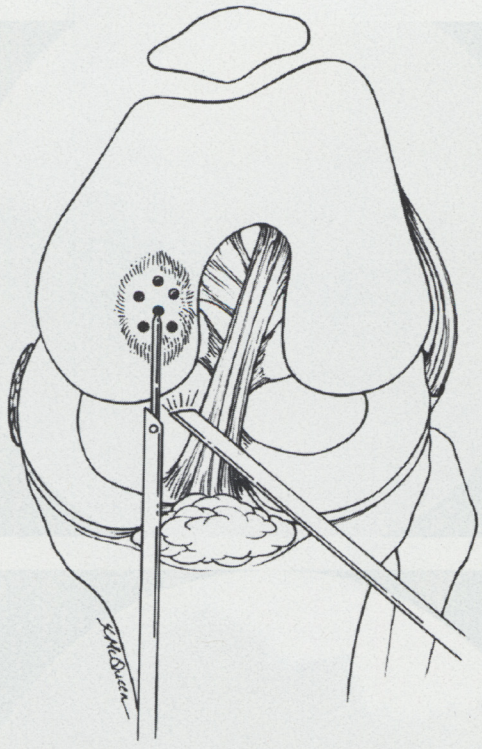


**FIG. 18.** Drilling intact osteochondritis dissecans lesions recruits mesenchymal cells to help heal the lesion. (Reproduced with permission from Sisk TD. Arthroscopy of the knee and ankle. In: Crenshaw AH, ed. *Campbell's operative orthopaedics*, Vol. 4, 7th ed. St. Louis: C.V. Mosby, 1987:2598.)

95% of patients older than 70 years had tricompartmental disease. Another study followed 22 children for an average of 34 years and found the outcome of JOCD was not as good as Linden had described. In this report, 32% of children had moderate to severe radiographic evidence of degenerative joint disease on follow-up. Especially poor results were seen in children with large defects and those involving the lateral femoral condyle (78).

## Surgical Treatment

Patients with an unstable osteochondral fragment with viable subchondral bone and lesion size of more than 1 to 2 $cm^2$ who present with knee pain, catching, locking, and effusion usually require surgical intervention. OCD lesions in the weightbearing zone of the knee should be fixed when possible, after which patients maintain nonweightbearing for periods of time that range from 6 to 12 weeks. Lesions in the nonarticulating portion of the knee are less common and should be repaired when possible as well. Irreparable lesions in weightbearing zones can be treated with cartilage restoration procedures if they fail to respond to débridement and loose body removal. Irreparable lesions in the nonarticulating areas often respond to simple débridement (79,80).

Arthroscopic treatment allows the surgeon to evaluate the entire articular surface and to provide initial treatment with minimal morbidity. Contraindications for purely arthroscopic surgery include the inability to obtain a perfect reduction whereby arthroscopic-assisted approaches may prove useful. Treatment of intact stable lesions in adult patients or patients approaching adulthood can be performed by drilling the bed of the lesion with postoperative-protected weightbearing. Treatment of unstable lesions includes fixation by bone pegs, 0.062 Kirschner wires, bioabsorbable pins, or screws (small fragment screws, Herbert screws, Acutrak screws). Treatment of macerated nonsalvageable lesions in the weightbearing zones of the condyle includes fragment removal and, ultimately, cartilage restoration procedures.

Wildey (81) initially described drilling bone for the promotion of vascular ingrowth in 1915. A generation later, Smillie (82) used drilling to treat OCD lesions. In 1989, Bradley and Dandy (83) have used drilling of stable OCD lesions in skeletally immature patients. Drilling of the subchondral bone leads to recruitment of mesenchymal stem cells, resulting in improved healing rates and faster returns to activity than conservative treatment (Fig. 18). Techniques vary and include arthroscopic antegrade and fluoroscopically guided retrograde drilling of *in situ* OCD lesions. Generally, results in skeletally immature patients are superior to skeletally mature patients.

Bone grafting of stable OCD lesions has been reported to lead to lesion healing after 6 months. The technique requires retro-

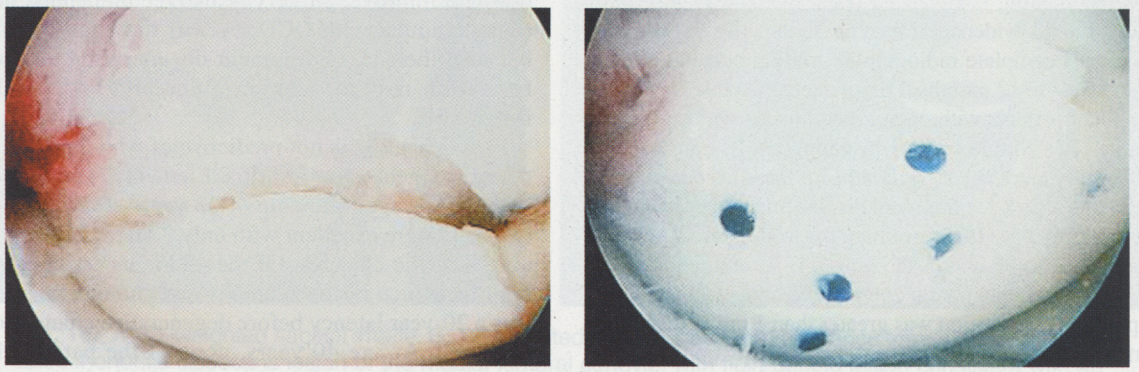


**FIG. 19. A:** Example of an *in situ* lesion of osteochondritis dissecans repaired with 2-mm polylactic acid bioabsorbable pins **(B)**.

grade drilling through the femoral condyle and packing cancellous bone graft under fluoroscopic control to the level of the lesion. Caution must be maintained to avoid penetrating the OCD lesion and disrupting the articular cartilage (84).

Cortical bone pegs have been used to fix large OCD lesions with 80% good to excellent results (85,86). Through an arthrotomy, matchstick-like bone pegs harvested from the medial side of the proximal tibial tubercle are placed through drill holes made within the OCD lesion. Other techniques include metallic pins or Kirschner wires to fix the lesions (87). Smooth pins have a tendency to migrate, but threaded pins are more difficult to remove (88). To avoid the complications of hardware, bioabsorbable polylactic acid pins have been used with some success (Fig. 19), although foreign body reactions have been reported (89,90). The lack of compression with the use of these pins is another potential limitation (91).

Fragments that are detached have a tendency to continue to grow as they receive nutrients from the synovial fluid. These fragments need to be meticulously débrided to fit into the defect perfectly. Some craters have a sclerotic base and need to be curetted down to bleeding bone. Often, we will microfracture the base of the defect to promote a biologic healing response. If the resulting crater is too concave for the fragment, cancellous bone from the adjacent nonarticular metaphyseal bone can be harvested to fill in the defect. Fragments should be left 1 to 2 mm proud to allow for subsidence after bone grafting.

Screws are effective in compressing the fragment and allowing healing to occur (92). Cannulated screws are preferred because of their ease of insertion and removal (75). The authors prefer the use of Herbert or similar differentially pitched screws (Acutrak), which offer good compression and can be buried beneath the articular surface so as to avoid scuffing of the tibial articular cartilage (Fig. 20). Unfortunately, a fibrous cap forms over the screw's head making removal difficult. The downside of cannulated screws is that a second surgery is required to remove the screws, although consideration for leaving recessed headless screws may be appropriate. Patients are made non-weightbearing during the 6 to 8 week postoperative period in an effort to prevent defect fragmentation and scuffing of the tibial plateau from the fixation device.

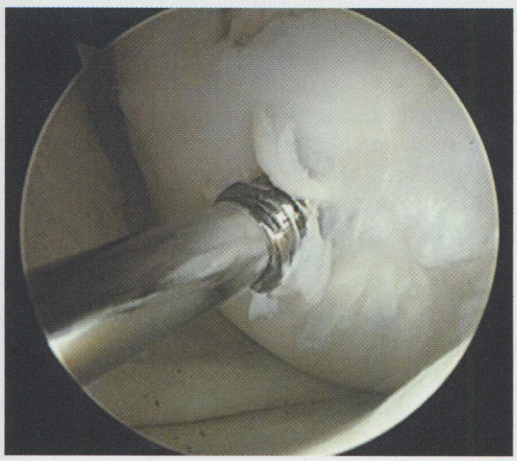


**FIG. 20.** Example of an *in situ* lesion of osteochondritis dissecans repaired with a headless screw, offering significant compression across the defect.

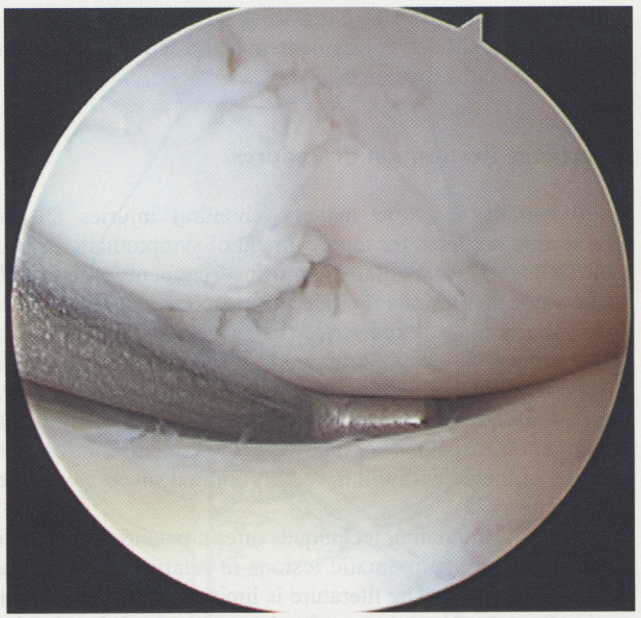
## Cartilage Restoration Procedures

Techniques used to manage chondral injuries are now becoming accepted for the treatment of symptomatic irreparable OCD (93–96). Palliative first-line treatment for irreparable osteochondral lesions includes arthroscopy and débridement. It is the authors' experience that patients with short-lived symptoms that are predominately mechanical in nature may respond favorably to these techniques. However, patients with symptoms believed to be due to the defect itself rather than the fragment (i.e., pain, swelling, weightbearing discomfort in the region of the defect) will have only limited success with these techniques (72).

Marrow-stimulating techniques offer a potentially reparative technique for symptomatic lesions in relatively low-demand patients (Fig. 21). The literature is limited and largely supports microfracture as a technique for the treatment of chondral injuries without significant bone loss (97,98). Unfortunately, this technique often results in recurrent symptoms (99). Typically, recovery from these procedures requires a minimum of 8 to 12 weeks, with up to 6 weeks of protected weightbearing and the use of continuous passive motion for 4 to 6 hours each day. After microfracture, patients may require up to 4 to 6 months to become relatively symptom free. Patients are educated to observe for recurring pain, swelling, or mechanical symptoms. Regularly scheduled follow up visits are important to monitor for the recurrence of symptoms. Repeat radiographs are required to evaluate for signs of defect progression, which may include defect enlargement, loose body formation, or frank degenerative change.

Additional treatment options exist for patients who develop recurrent symptoms after first-line treatment or for patients who have very large or very deep osteochondral lesions that are otherwise anticipated to fail arthroscopic débridement or marrow-stimulating techniques. Treatment alternatives include restorative techniques such as osteochondral grafting and autologous chondrocyte implantation (ACI). Presently, there is no optimal defect size or depth considered as an absolute indication for osteochondral grafting or ACI. Generally speaking and depending on patient activity level and defect location, defects larger than 2 to 3 $cm^2$ and deeper than 4 to 6 mm are likely to demonstrate recurrent symptoms after débridement or microfracture and, thus, may benefit from osteochondral grafting or ACI.

There are essentially three restorative options that attempt to establish normal articular cartilage to the injured area: osteochondral autografts, osteochondral allografts, and ACI. Osteochondral autograft procedures are predominately indicated for smaller lesions with minimal bone loss due to concerns for graft stability, which is otherwise compromised with larger uncontained lesions. Autografts are taken from relatively nonweightbearing sites such as the lateral trochlea or intercondylar notch. These composite bone and cartilage grafts maintain their viability by the nutrients supplied by synovial fluid and the surrounding subchondral bone bed. Limitations due to donor site availability and the associated donor morbidity limit



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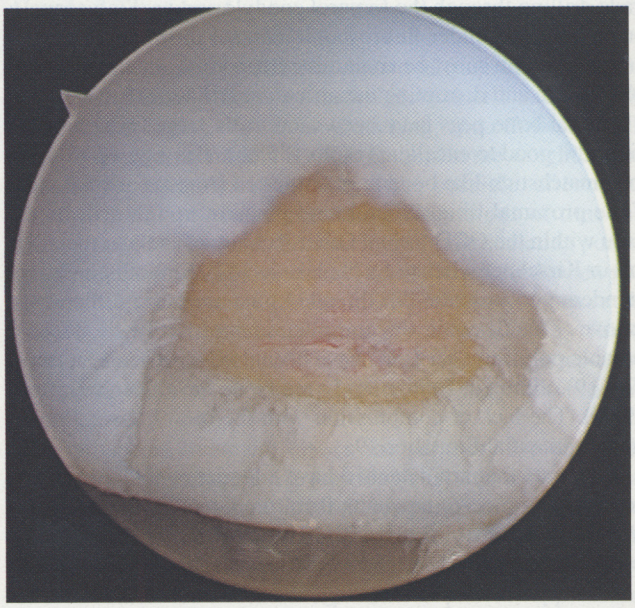

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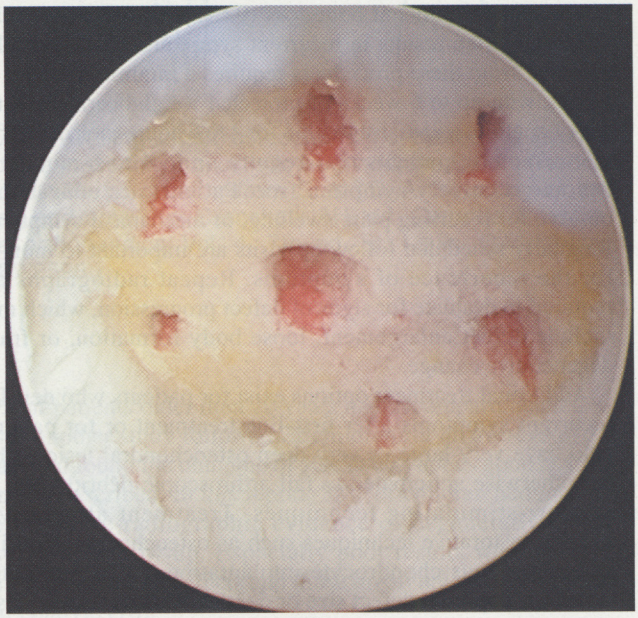

C

**FIG. 21.** Microfracture of unstable osteochondritis dissecans fragment with remaining portion of lesion considered stable. **A:** Lesion appearance before débridement. **B:** Débridement through the calcified layer. **C:** Microfracture with holes spaced 2 to 3 mm apart.

the primary indications to relatively small symptomatic defects of the weightbearing surface of the femoral condyle. Similar to microfracture techniques, the literature reporting the results of osteochondral autograft procedures largely deals with patients who have chondral defects of the femoral condyle of less than 2 to 3 $cm^2$ without significant bone loss (Fig. 22) (100–102).

Osteochondral allografts offer another excellent treatment option for the symptomatic patient with relatively large OCD lesions. Gross et al. (103–105) popularized the use of fresh osteochondral allografts, especially those used for joint restoration. He found that fresh cartilage allografts maintained nearly 100% cartilage cell viability at retrieval. Osteochondral allograft processing is typically carried out within 24 hours of the death of the donor. The limitations of fresh osteochondral allograft transplantation are logistical and partially related to the need for implantation before the final determination of bacterial contamination. Currently, extensive research in the area of prolonged-fresh cartilage preservation is underway. These techniques use tissue culture methodology to main allograft cell viability. Once perfected, fresh transplantation will be less of a logistical concern.

Current indications include larger (i.e., more than 2 to 3 $cm^2$) unipolar lesions due to localized degenerative disease, posttraumatic arthritis, AVN, and OCD. Ligament instability and malalignment must be corrected concomitantly or in a staged fashion. In the setting of meniscal deficiency, combined allograft meniscus transplantation may be considered. Contraindications include inflammatory arthritis, steroid dependency, uncorrected comorbidities (i.e., malalignment,

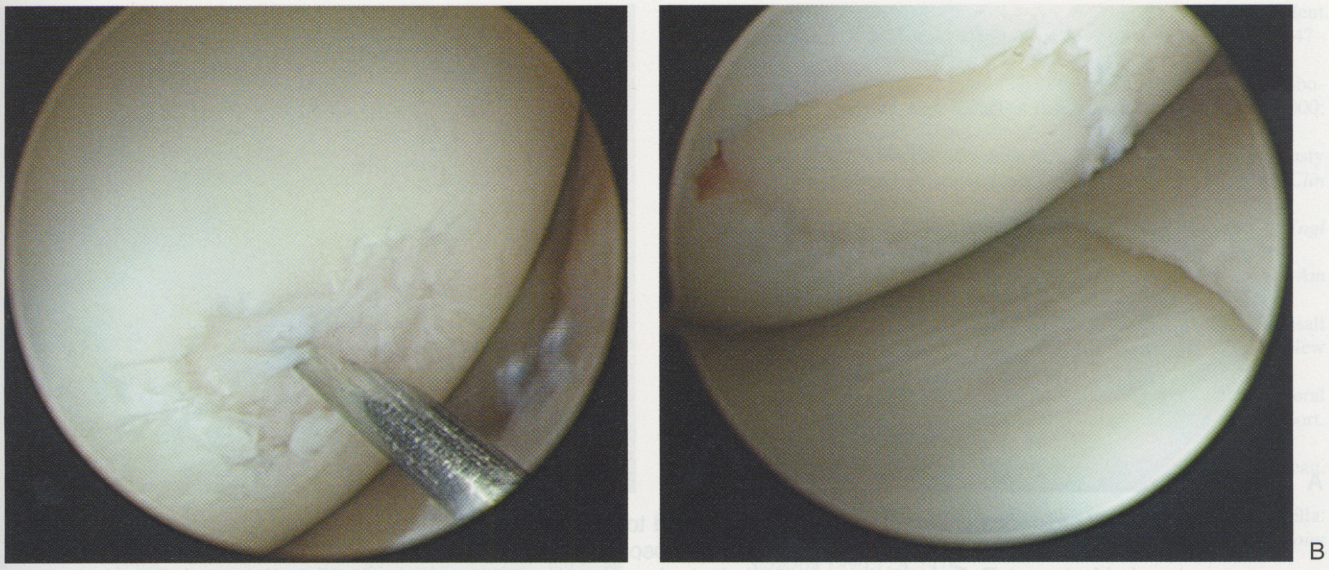

**FIG. 22. A:** Typical symptomatic chondral defect considered ideal for osteochondral autograft transplant using a single 9-mm plug **(B)**.

ligament insufficiency, subtotal meniscectomy), and any other general medical condition that may affect graft incorporation. Relative contraindications include bipolar (i.e., kissing lesions) disease.

Fresh osteochondral allograft transplantation is the author's preferred method of treatment for patients with OCD who have relatively deep lesions (i.e., deeper than 8 to 10 mm) or in patients who are older than 35 to 40 years, even with shallow OCD lesions owing to a more rapid recovery compared to ACI (see later) (Fig. 23). Relatively young patients (i.e., younger than 25 to 30 years) who have lesions considered amenable to either osteochondral allograft transplantation or ACI are often indicated for the latter because of the relative preservation of subchondral bone when performing ACI. This allows other treatment options, such as revision to osteochondral allograft transplantation, should ACI fail.

Gross and others have demonstrated a clinical success rate at 5 years of 95%, at 10 years of 77%, and 66% at 20 years (103–107). The results of fresh osteochondral grafting for bipolar lesions are considerably less favorable. The grafts are size-matched based on plain radiographs corrected for magnification. Typically, the width of the proximal tibia 1 cm below the joint line on the anteroposterior radiograph is sufficient to determine the appropriate size match. It is important that the graft is not used to correct malalignment. Instead, osteotomy should be used to correct mechanical axis abnormalities simultaneously or in a staged fashion. Postoperatively, patients are made nonweightbearing for 6 to 8 weeks with liberal use of

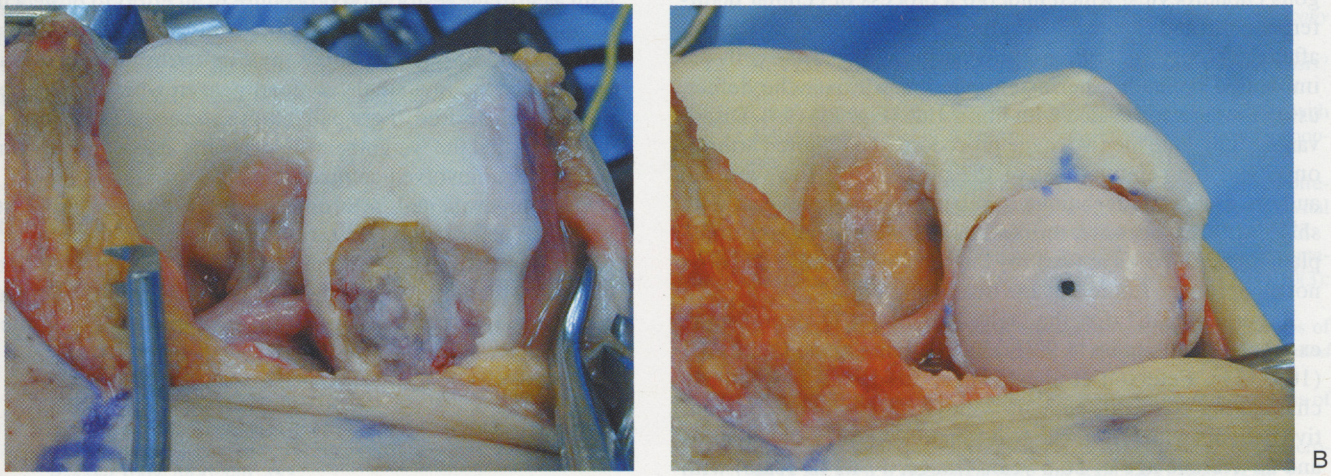

**FIG. 23. A:** Large lesion of osteochondritis dissecans occupying the majority of the weightbearing femoral condyle treated with fresh osteochondral allograft transplant fixed with a single bioabsorbable pin **(B)**.

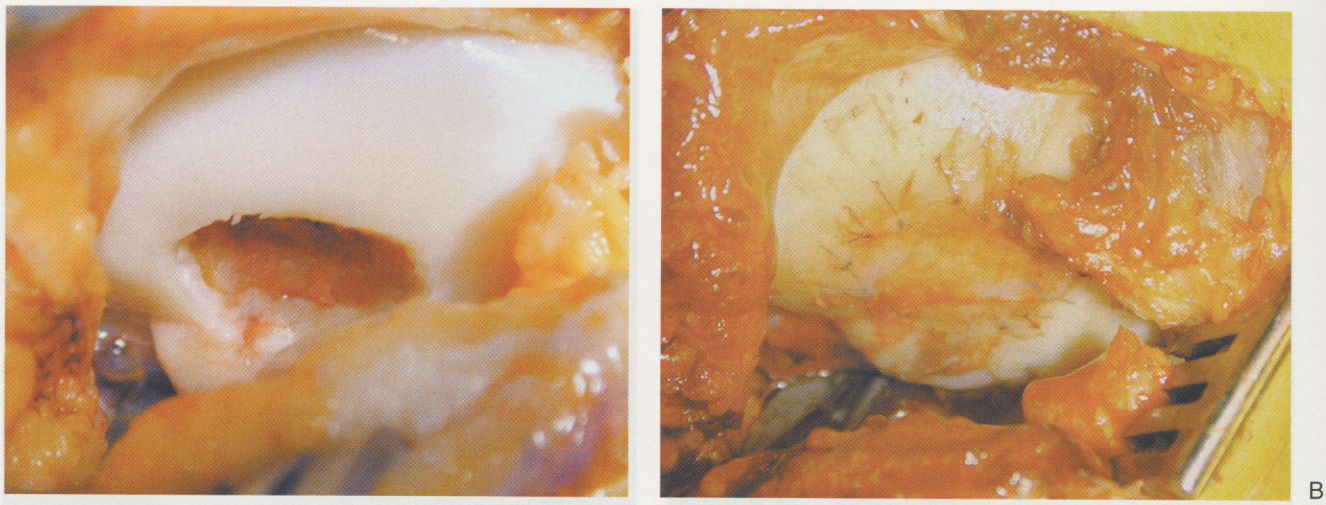

**FIG. 24.** Autologous chondrocyte implantation used to treat the unstable lesion in the same patient from Figure 19, which had failed prior fixation with bioabsorbable pins. **A:** Débridement of the necrotic osteochondral fragment. **B:** Defect covered with periosteal patch, which is sewn and glued into place. Autologous chondrocytes are subsequently injected beneath the periosteal patch.

continuous passive motion. Return to high-level activities is generally delayed until graft incorporation is noted radiographically, which can take up to 12 months.

ACI is another option for the treatment of symptomatic lesions of OCD. Reported first by Brittberg et al. (108) for the treatment of symptomatic chondral injury, ACI has become an accepted treatment option for lesions of the femoral condyle, trochlea, and, recently, the patella (94,109–111). With the exception of defects larger than 10 to 12 $cm^2$, the indications and contraindications for ACI are similar to fresh osteochondral allograft transplantation. The treatment of OCD with ACI without bone grafting is considered acceptable for shallow defects with no more than 6 to 8 mm of bone loss. Deeper lesions can be treated with staged bone grafting and healing followed by ACI. Single-staged bone grafting with a "periosteal sandwich" of cultured chondrocytes between cambium layers is described but is still considered largely investigational (110,111).

The procedure involves arthroscopically harvested autologous chondrocytes, which undergo a process of cellular dedifferentiation and expansion in culture. At a minimum of 3 weeks after the biopsy, an arthrotomy is performed, and the cells are implanted beneath a periosteal patch sewn over the defect, using a suture and sealed with fibrin glue (Fig. 24) (94). Disadvantages include the two-stage procedure, the required arthrotomy, and the relative cost of the procedure. However, the authors prefer this technique in relatively young patients with shallow OCD, because, unlike osteochondral allograft transplantation, ACI does not destroy the subchondral bone and does not compromise future salvage treatment options.

Reports of ACI for the treatment of OCD demonstrate excellent results in up to 90% of all patients treated with ACI (109–111). Peterson et al. (109) reported on a particularly challenging cohort of patients with OCD. Eighteen consecutive patients with OCD who had a history of 53 prior surgeries and an average age of 26.6 years (range, 17 to 46 years) with an average defect size of 4.7 $cm^2$ (range, 1.5 to 12.0 $cm^2$) were treated with single stage ACI with a mean follow-up of 3.1 years (range, 2.0 to 6.3 years). Their clinical results were excellent in 13, good in 3, fair in 1, and poor in 1. Sixteen of 18 (89%) patients felt that the surgery had met their expectations for clinical outcome. These results are similar to that achieved by the senior author (94).

## Reconstructive Options

Mechanical malalignment can excessively load the affected joint surface, leading to increased symptoms, decreased potential for lesion healing, and the possibility of failure of any of the described cartilage restoration procedures. Performed in isolation for the severely malaligned limb with OCD, osteotomy can be successful in reducing a patient's symptoms (112). However, unlike the malaligned osteoarthritic knee, patients with OCD are typically younger and expect to remain or become more active after treatment. Thus, the authors routinely perform an osteotomy, when indicated, in combination with a cartilage restoration procedure in an effort to protect the reconstruction and to maximize the clinical benefit of the procedure (Fig. 25). Also, unlike the osteoarthritic patient with bipolar disease in a single compartment in which overcorrection is often desired, these patients are corrected to just beyond neutral so that the weightbearing axis falls at least into the first half of the uninvolved compartment.

Unicompartmental arthroplasty, total knee arthroplasty, and knee arthrodesis are rarely indicated in this relatively young population and certainly would not be the initial treatment considered in the adolescent or young adult. These are considered as salvage procedures when other treatment options have failed.

## Conclusion

OCD is relatively common and has multiple etiologies, including trauma. The treatment of OCD in the knee is based on disease chronicity, patient age, skeletal maturity, response to

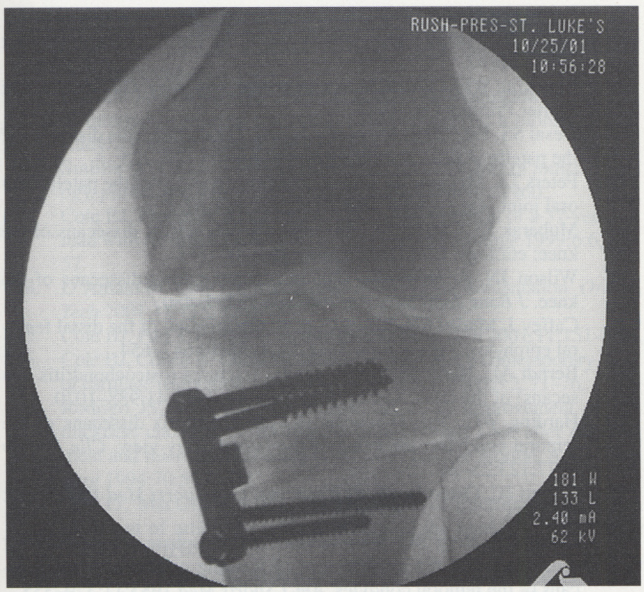


**FIG. 25.** Intraoperative fluoroscopic view of opening wedge osteotomy and fresh osteochondral allograft performed concomitantly in a patient with chronic osteochondritis dissecans and varus malalignment.

previous treatments, and the depth and quality of the subchondral bone. Most commonly, patients with open physes with nondisplaced lesions will heal with appropriate conservative treatment, including relative rest and restricted weightbearing. Drilling of stable lesions or drilling and anatomic fixation of unstable lesions is indicated in skeletally immature patients with persistent symptoms or in the skeletally mature patient with OCD when the fragment remains *in situ*. When fragments are not amenable to fixation, removal can be beneficial, especially when mechanical symptoms prevail. However, these patients, as do those treated with marrow-stimulating techniques such as microfracture, often become symptomatic with increasing follow-up time and levels of activity. In symptomatic patients who fail fixation or whose defects are not reparable, restorative options may be indicated. These most commonly include fresh osteochondral allograft transplantation or ACI. A low threshold for performing a corrective osteotomy is paramount to the success of these procedures. The decision making for any treatment option must be evidenced based when possible and only further prospective study will permit this to occur.

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