

# Knee Cartilage: Diagnosis and Decision Making

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Articular cartilage is vulnerable to irreversible traumatic injury and degenerative disease. Damaged articular cartilage has a limited ability to heal without intervention owing to two primary factors: lack of a vascular response, and relative absence of an undifferentiated cell population to respond to injury. The rationale for early surgical intervention for articular cartilage injuries is based on the symptomatic nature of focal chondral lesions and the potential for these lesions to progress.

The surgical management of articular cartilage defects is based on several underlying principles, including the reduction of symptoms, improvement in joint congruence and force distribution, and prevention of additional cartilage damage. This chapter reviews the anatomy and biomechanics of articular cartilage, discusses the clinical evaluation of these injuries, and provides a practical approach to the treatment of symptomatic articular cartilage injuries.

## Anatomy and Biomechanics

The function of articular cartilage is to provide for smooth, pain-free gliding of the joints during skeletal motion. The architecture of articular cartilage is such that it provides a low coefficient of friction to allow smooth motion throughout a lifetime. However, normal function requires maintenance of the structural properties and the metabolic function of cartilage.

Articular cartilage is composed of a large extracellular matrix including type II collagen and proteoglycan aggregates. Collagen fibers give cartilage its form and tensile strength, and water constitutes 75% to 80% of the extracellular matrix, functioning largely in compression. In addition, chondrocytes synthesize and degrade proteoglycans and are responsible for cartilage homeostasis.

The structure of articular cartilage can be divided into three zones—superficial, transitional, and deep—each of which imparts mechanical properties contributing to the ultimate function of the articular surface. The superficial zone is composed primarily of collagen fibers oriented parallel to the joint surface, and it primarily resists shear forces. The middle, or transitional, zone is composed of obliquely oriented collagen fibers and primarily resists compressive forces. The fibers in the deep zone are oriented perpendicular to the subchondral plate, and this zone resists both compressive and shear forces. Injury to any one of these layers, the chondrocytes, or the subchondral bone can disrupt the normal biomechanical properties of articular cartilage, leading to further degeneration.

## Historical Aspects

Both partial- and full-thickness lesions have limited capacity for repair. The avascular nature of articular cartilage and the limited stem cell population limit the healing response following injury. In addition, the constant load of articular cartilage, particularly in the knee, creates a challenging mechanical environment for an appropriate healing response.

The natural history of asymptomatic focal chondral defects is not well documented. It is thought that chondral injuries lead to the development of degenerative arthritis, although this has not been proved. Recent studies of unipolar, unicompartmental, full-thickness articular cartilage lesions following debridement have shown progression to radiographic joint space narrowing.<sup>22</sup> Symptomatic lesions, however, are unlikely to become quiescent without significant activity restriction or some form of surgical intervention.

## Clinical Evaluation

### History

Patients with chondral or osteochondral injuries typically report either a twisting, shearing-type injury combined with an axial load or significant blunt trauma causing an impaction injury. In addition, these lesions are commonly associated with other soft tissue injuries about the knee, including condylar lesions from ligament rupture (anterior cruciate ligament tears), or patellar or trochlear lesions following patellar dislocation. Full-thickness chondral injuries can account for 5% to 10% of the pathology following acute hemarthrosis and must be suspected in sports- or work-related injuries.<sup>26</sup>

Symptomatic chondral lesions typically present as knee pain localized to the affected compartment: the medial or lateral hemijoint for medial or lateral condyle injuries, and the patellofemoral joint for patellar or trochlear lesions. Weight-bearing activities typically aggravate symptoms from lesions on the medial or lateral femoral condyle. Activities such as sitting, stair climbing, and squatting aggravate patellofemoral lesions. In addition, recurrent effusions, catching, and locking can occur with symptomatic chondral lesions. Patients with documented lesions who have atypical symptoms should be critically evaluated to prevent inadvertent treatment of coexisting incidental lesions.

### Physical Examination

Patients are typically tender along the ipsilateral joint line or condyle. Patients with patellar or trochlear lesions typically have patellar crepitation and a positive patellar grind and inhibition test. An effusion may be present as well. It is essential to evaluate for concomitant pathology that would modify treatment recommendations, such as malalignment or ligament deficiency. For medial or lateral condyle injuries, careful attention must be paid to any varus or valgus limb alignment. For patellar or trochlear lesions, maltracking of the patella, including a tight lateral retinaculum or high Q angle, must be evaluated. In addition, signs of meniscal pathology must be evaluated, because coexisting disease is frequently present. Finally, any ligamentous injury must be noted; failure to address these injuries can lead to early failure of any articular cartilage repair technique.

### Imaging

Standard diagnostic imaging should include a standard weight-bearing anteroposterior radiograph of both knees in full extension, a non-weight-bearing 45-degree-flexion lateral view, and an axial view of the patellofemoral joint. In addition, a 45-degree-flexion weight-bearing posteroanterior radiograph is recommended to identify subtle joint space narrowing that traditional extension views may miss. If there is any degree of clinical malalignment, a long-cassette mechanical axis view should be ordered to evaluate the mechanical axis of the limb.

Magnetic resonance imaging can be helpful in delineating the extent of articular cartilage lesions, especially in the setting of completely normal radiographs. It can define the location, size, and depth of chondral injuries and can evaluate any subchondral fractures, bone bruises, or osteochondritis dissecans lesions. Magnetic resonance imaging can also assess the stability of an osteochondral lesion: fluid behind the lesion indicates an unstable lesion that may be amenable to surgical stabilization. Evolving techniques, including two-dimensional fat suppression, three-dimensional fast spin-echo sequences, and gadolinium enhancement, provide accurate information on the presence and size of articular cartilage lesions and may assist in the evaluation of patients after cartilage restoration procedures.

### Classification

Focal chondral defects of the femur are a specific subset of articular cartilage injuries. The Modified International Cartilage Repair Society Chondral Injury Classification System classifies chondral injuries based on the amount and depth of the cartilage lesion. Most commonly, these lesions are classified using the modified Outerbridge system (Table 54-1).<sup>27</sup> Other important factors that affect the ability of cartilage lesions to heal with operative treatment include the location and size of the lesion, the depth and condition of the subchondral bone, the condition of the surrounding normal cartilage, and coexisting knee pathology. In addition, it is important to recognize any bony deficiency that may alter the treatment plan for the repair of chondral injuries.

In addition to classifying lesions by depth and size, it is important to consider other factors when determining the appropriate treatment. These include whether the defect is acute or chronic, the defect's location, associated ligamentous instability, integrity of the meniscus, and tibiofemoral or patellofemoral malalignment. Many patient factors must also be considered, including age, activity level, occupation, expectations, body weight, presence of systemic disease, and results of previous treatment attempts.

### Associated Injuries

The most common associated injuries are ligament and meniscus tears. The meniscus functions in both load

**Table 54-1** Modified Outerbridge Classification of Cartilage Lesions

Grade	Description
I	Softening of articular cartilage
II	Fibrillation or superficial fissures of cartilage
III	Deep fissuring of cartilage without exposed bone
IV	Exposed subchondral bone

From Outerbridge R: The etiology of chondromalacia patellae. *J Bone Joint Surg Br* 43:752-757, 1961.

distribution and shock absorption. The detrimental effects of meniscal deficiency, leading to excessive articular cartilage load and the development of osteoarthritis, have been well documented.<sup>17,33,34</sup> Although meniscal and chondral injuries can occur concomitantly, it is likely that many chondral injuries occur secondary to meniscal deficiency. Alternatively, a highly irregular articular surface may predispose a patient to a meniscus tear. Every attempt should be made to preserve and repair the meniscus, especially in the presence of a chondral defect. If a chondral defect is present in a meniscus-deficient knee, it is essential that the meniscal deficiency be addressed (e.g., with meniscal allograft transplantation) when treating the articular cartilage lesion. In addition, any ligamentous instability must be addressed before or at the time of articular cartilage repair. Knee instability will lead to excessive shear forces and early failure of articular cartilage repair.

If varus malalignment exists in the presence of medial condyle disease, a valgus-producing high tibial osteotomy should be performed to improve the predictability of the repair. Similarly, valgus malalignment should be treated with distal femoral osteotomy. When treating patellar or trochlear chondral lesions, there is an increasing trend toward distal realignment with anteriorization or antero-medialization of the tibial tubercle, even in the face of normal patellar tracking. This is performed primarily to unload the patellofemoral compartment and protect the cartilage repair site.

## Treatment Options

### Nonoperative

Nonoperative treatment for chondral injuries is generally reserved for asymptomatic lesions. Small, incidental chondral lesions can be treated with benign neglect in the absence of clinical symptoms, although defect progression is possible. Nonoperative treatment of symptomatic lesions is unlikely to be successful but includes a regimen similar to that for osteoarthritis. This includes nonsteroidal anti-inflammatory medications, physical therapy, intra-articular corticosteroid or hyaluronic acid injections, and nutritional supplementation with chondroitin and glucosamine sulfate. Unfortunately, in high-demand patients with symptoms attributable to the defect, nonoperative treatment is rarely successful. If symptoms persist despite nonsurgical treatment, surgical intervention is warranted. Although there are no definitive guidelines for the length of nonsurgical treatment, it is generally believed that symptomatic chondral lesions should be treated aggressively, because progression and further cartilage deterioration may limit the benefits of cartilage restoration.

### Operative

The principal goals in the surgical management of symptomatic chondral defects are to reduce symptoms, improve joint congruence, and prevent additional

cartilage deterioration. Primary repair should be attempted for all traumatic lesions and symptomatic unstable osteochondritis dissecans lesions with a viable osteoarticular fragment of at least 1 cm<sup>2</sup> and an adequate bony bed for fixation.

For those lesions that cannot be primarily repaired, the treatment options can be characterized as palliative, reparative, or restorative. Palliative procedures, such as debridement and lavage, are used for incidentally discovered lesions or symptomatic lesions in low-demand patients with a preponderance of mechanical symptoms or signs of meniscal pathology. In these instances, there is no attempt to repair or replace the damaged articular cartilage. Reparative procedures, such as marrow-stimulating techniques (drilling, abrasion arthroplasty, or microfracture), promote a fibrocartilage healing response in the area of the defect. Restorative techniques replace the damaged cartilage with new articular cartilage; these include autologous chondrocyte implantation, osteochondral autografting, and fresh osteochondral allografting. Taking into account the factors previously discussed, a treatment algorithm has been developed to guide the implementation of these options (Fig. 54-1).

### Primary Repair

For acute osteochondral lesions or in situ and unstable osteochondritis dissecans, primary repair should be attempted. The size and location of the lesion are the primary determinants of whether the fragment needs to be removed or can be stabilized. Every attempt should be made to fix large fragments (>1 cm<sup>2</sup>) from the weight-bearing portion of the femoral condyles. Some lesions are amenable to arthroscopic fixation, but an arthrotomy may be necessary for adequate reduction and fixation of the fragment. Fixation is usually performed provisionally with K-wires and then with bioabsorbable pins or metal screws (Fig. 54-2). Typically, patients are kept non-weight bearing following repair, and the screws are removed 8 to 10 weeks postoperatively. Continuous passive motion may be used, or patients are asked to perform 600 to 800 cycles/day without the use of a formal machine. If headless screws are used and are sufficiently recessed in the subchondral bone, there may be no need for removal.

### Debridement and Lavage

Palliative procedures such as debridement and lavage are reserved for lower-demand patients with incidentally discovered chondral lesions or those with small lesions (<2 to 3 cm<sup>2</sup>) and limited symptoms. Debridement can be particularly helpful for patients with mechanical symptoms from a loose chondral flap. However, relief from debridement and lavage may be incomplete and temporary. Postoperative rehabilitation is relatively straightforward and should include weight bearing and resumption of activities as tolerated.

Thermal debridement of partial-thickness articular cartilage injuries is currently being investigated. Proponents of thermal debridement advocate this treatment

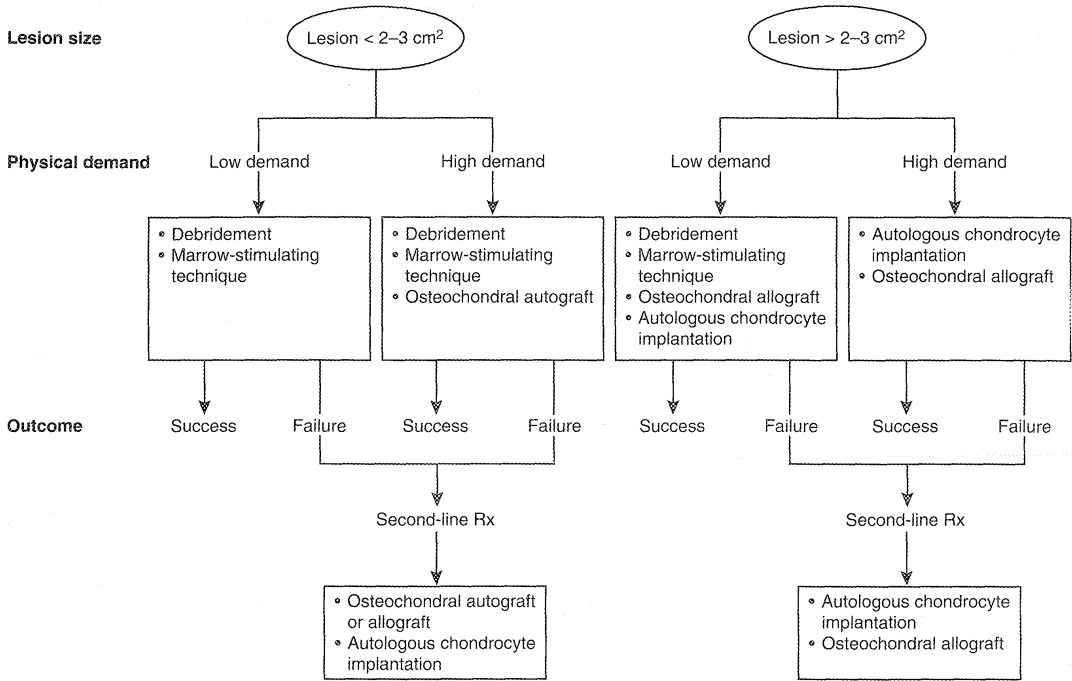


Figure 54-1 Treatment algorithm for articular cartilage lesions.

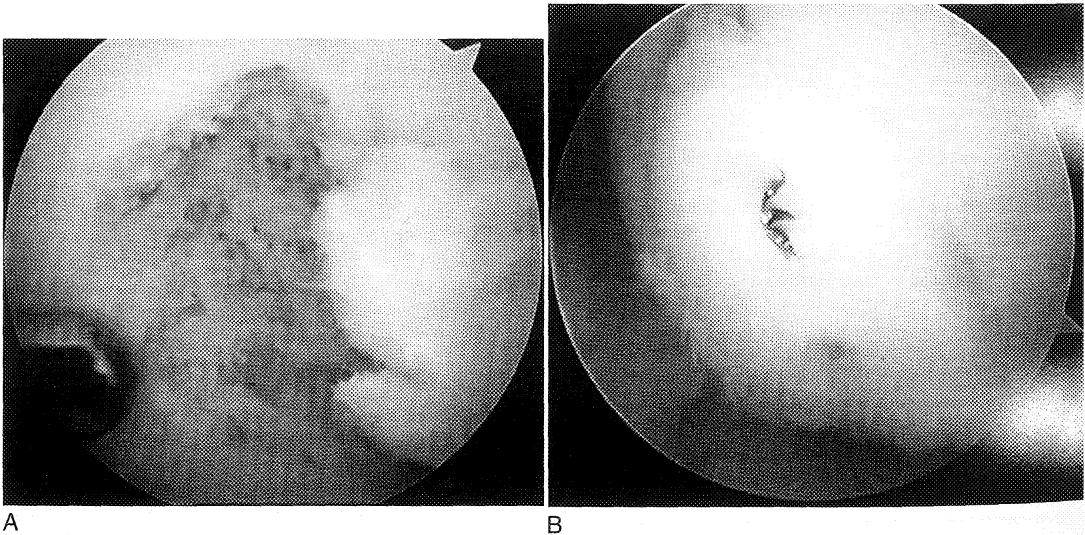


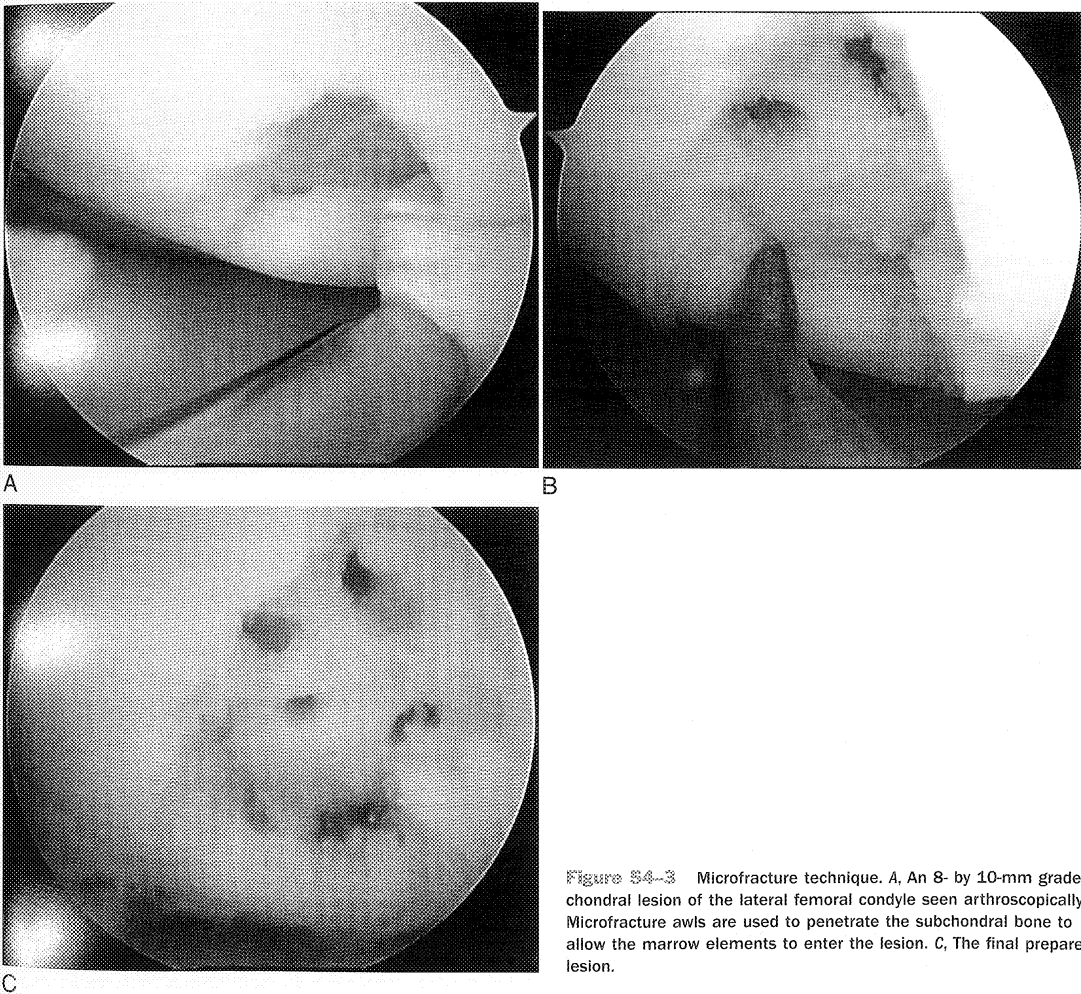
Figure 54-2 A, Arthroscopic example of an unstable osteochondritis dissecans lesion of the medial femoral condyle considered amenable to repair. B, Arthroscopic picture of the lesion repaired with two variably pitched headless screws.

for the smoothing of articular cartilage and the containment of articular cartilage lesions (preventing propagation). However, if improperly used, thermal treatment can cause injury to the underlying or surrounding normal cartilage and potentially affect the subchondral bone.<sup>7,21</sup> Therefore, thermal treatment of articular cartilage injuries must be approached with caution.

#### Marrow-Stimulating Techniques

For patients with small to moderate-sized lesions (1 to 5 cm<sup>2</sup>) and moderate demands, marrow-stimulating techniques such as drilling, abrasion arthroplasty, and microfracture can be used. All these techniques are used to stimulate fibrocartilage ingrowth into the chondral defect. Abrasion arthroplasty was initially developed by Pridie<sup>31</sup> as an open technique in 1959,<sup>18</sup> and it was later

modified for arthroscopic use by Johnson.<sup>19</sup> Abrasion arthroplasty has been used mostly for osteoarthritic knees rather than focal chondral defects. At this time, microfracture is the most commonly accepted technique for marrow stimulation (Fig. 54-3). It involves providing fibrocartilage repair tissue to the focal chondral defect by debriding the lesion through the calcified layer and penetrating the subchondral plate with specialized awls in an effort to expose the damaged area to progenitor cells present within the subchondral bone. In many cases, microfracture is used as a first-line treatment for focal chondral defects in the hope that larger cartilage restoration procedures can be avoided. Optimal results following microfracture come from rigid adherence to the postoperative protocol, so the procedure should not be performed casually. Postoperative management requires a prolonged period of non-weight bearing (4 to



**Figure 54-3** Microfracture technique. A, An 8- by 10-mm grade IV chondral lesion of the lateral femoral condyle seen arthroscopically. B, Microfracture awls are used to penetrate the subchondral bone to allow the marrow elements to enter the lesion. C, The final prepared lesion.

6 weeks) with continuous passive motion, or patients are asked to perform 600 to 800 cycles/day without the use of a formal machine.

### Cartilage Restoration Techniques

#### AUTOLOGOUS CHONDROCYTE IMPLANTATION

Autologous chondrocyte implantation (ACI; Fig. 54-4) is indicated for intermediate- to high-demand patients with symptomatic articular cartilage lesions who have failed at least an attempt at arthroscopic debridement. This technique is used primarily for larger (2 to 10 cm<sup>2</sup>) symptomatic lesions of the knee, principally of the femoral condyles. Recent literature supports its use for trochlear and patellar lesions, especially when combined with distal realignment procedures.<sup>29</sup> ACI is a two-stage technique in which 200 to 300 mg of autologous chondrocytes are biopsied arthroscopically in the first stage and implanted through an arthrotomy in the second stage. Coverage is obtained by a periosteal patch sewn with 6-0 Vicryl suture and sealed with fibrin glue. The repair tissue from this technique has been shown to be durable, mechanically firm, and hyaline-like in histology.<sup>29</sup> Lesions of osteochondritis dissecans are also appropriate candidates for ACI, provided that the depth of bone loss is less than 6 to 8 mm. The postoperative course is demanding, with a prolonged period of protected weight bearing and range of motion with continuous passive motion for 4 to 6 weeks. Symptom relief is generally predictable, but it may take 12 to 18 months for some lesions (e.g., patellofemoral lesions).

#### OSTEOCHONDRAL AUTOGRAFT TRANSPLANTATION

Osteochondral autograft transplantation (Fig. 54-5) is generally used for chondral lesions of the femoral condyle. These grafts are not generally recommended for the patella, owing to a mismatch of cartilage thickness between the donor and recipient site. In addition, care must be taken to match the curvature of the trochlea when such grafts are used for these lesions. They are generally used for small to medium-sized lesions (0.5 to 3 cm<sup>2</sup>), owing to limited donor site availability. Donor grafts can be harvested arthroscopically or through a small incision from the intercondylar notch or the lateral femoral trochlea. For larger lesions, the "mosaicplasty" technique of multiple plugs can be used. The advantages of osteochondral autografts are that they are autogenous tissue and have immediate normal hyaline architecture. There are several disadvantages of osteochondral autografts, however, including donor site morbidity, technical difficulty in achieving proper graft orientation and placement, residual gaps between the cartilage plugs, and the potential for cartilage or subchondral bone breakdown resulting from graft handling or improper placement.

#### OSTEOCHONDRAL ALLOGRAFT TRANSPLANTATION

Fresh osteochondral allograft transplantation (Fig. 54-6) involves the implantation of a composite cadaveric graft

that includes the subchondral bone and overlying hyaline cartilage in the site of the chondral defect. Osteochondral allograft transplants are used for medium to large articular cartilage lesions in relatively high-demand patients who tend to be somewhat older and often have associated bone loss (>6 to 8 mm) or for larger articular cartilage lesions (3 cm<sup>2</sup> up to an entire hemicondyle) in both low- and high-demand patients. These grafts are most commonly used on the femoral condyles but can also be used for the patella, trochlea, and medial and lateral tibial plateau along with the donor meniscus. Another relative consideration is patient age, with patients older than 40 years possibly being better candidates for allografting than for ACI because of biologic considerations and perhaps the patient's unwillingness to engage in the prolonged recovery process associated with ACI. Additionally, young patients with superficial chondral injury only may best be treated with ACI rather than osteochondral allografting simply because the subchondral bone is left undisturbed with the former procedure.

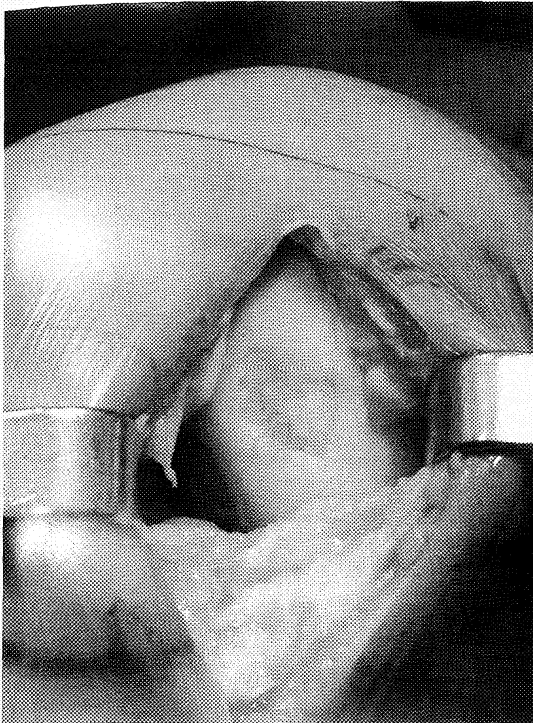
Osteochondral allograft transplantation depends on anatomic restitution of the articular surface with size-matched donor tissue. Fresh osteochondral tissue demonstrates good donor chondrocyte viability (60% or greater) at biopsy.<sup>11</sup> A major advantage of osteochondral allografts is the ability to replace large osteochondral defects with a single-stage procedure; disadvantages include availability, technical difficulty, cost, and possible disease transmission. Postoperatively, patients are kept non-weight bearing for 6 to 8 weeks and use continuous passive motion.

#### ARTHROPLASTY

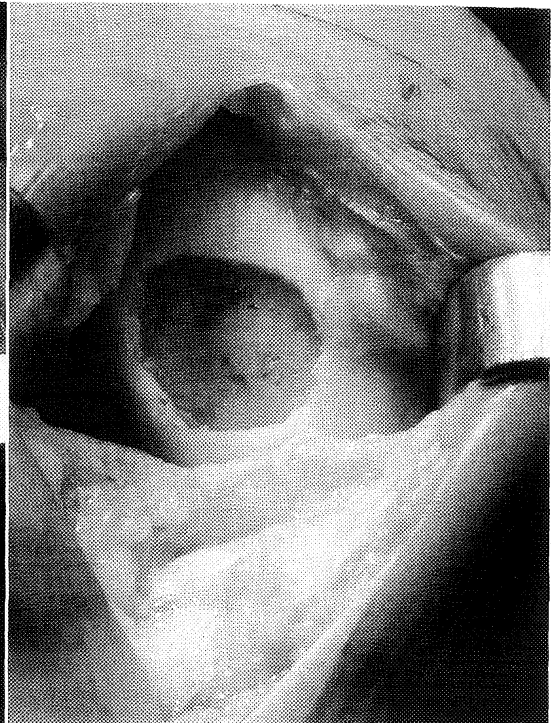
Although the focus of this chapter is on other alternatives for the repair or restoration of articular cartilage defects, prosthetic arthroplasty techniques, including unicondylar, patellofemoral, and total knee arthroplasty, remain viable options for the treatment of articular cartilage injuries.

### Rehabilitation Principles

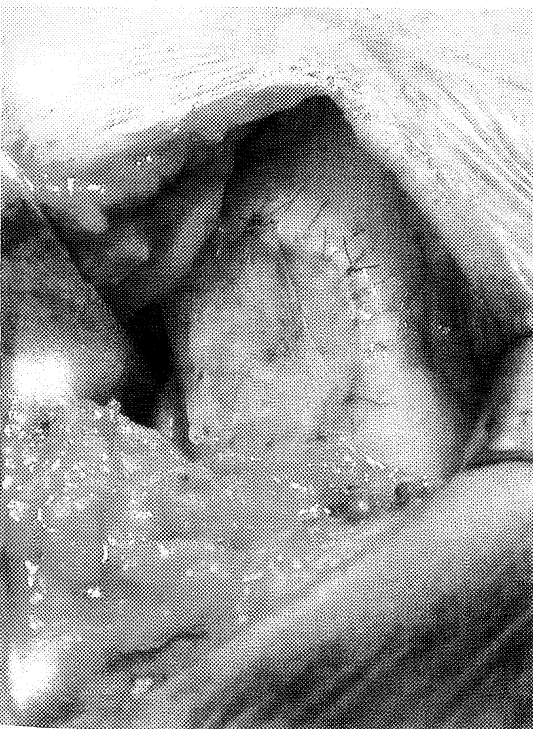
The specific rehabilitation regimens for each procedure for cartilage repair or restoration are outlined in the chapters on specific techniques. However, several general principles apply. Rehabilitation following palliative procedures allows a rapid return to activities with no restrictions on weight bearing or range of motion. Reparative procedures require strict protection of the lesion from loading to allow healing of the defect, while encouraging full range of motion for cartilage nutrition. Similarly, restorative procedures require protection of the healing articular surface from weight bearing, while encouraging range of motion. After 6 weeks of protection, weight bearing is usually advanced. The return to full activity depends on the procedure performed and can take from 4 to 18 months.



A



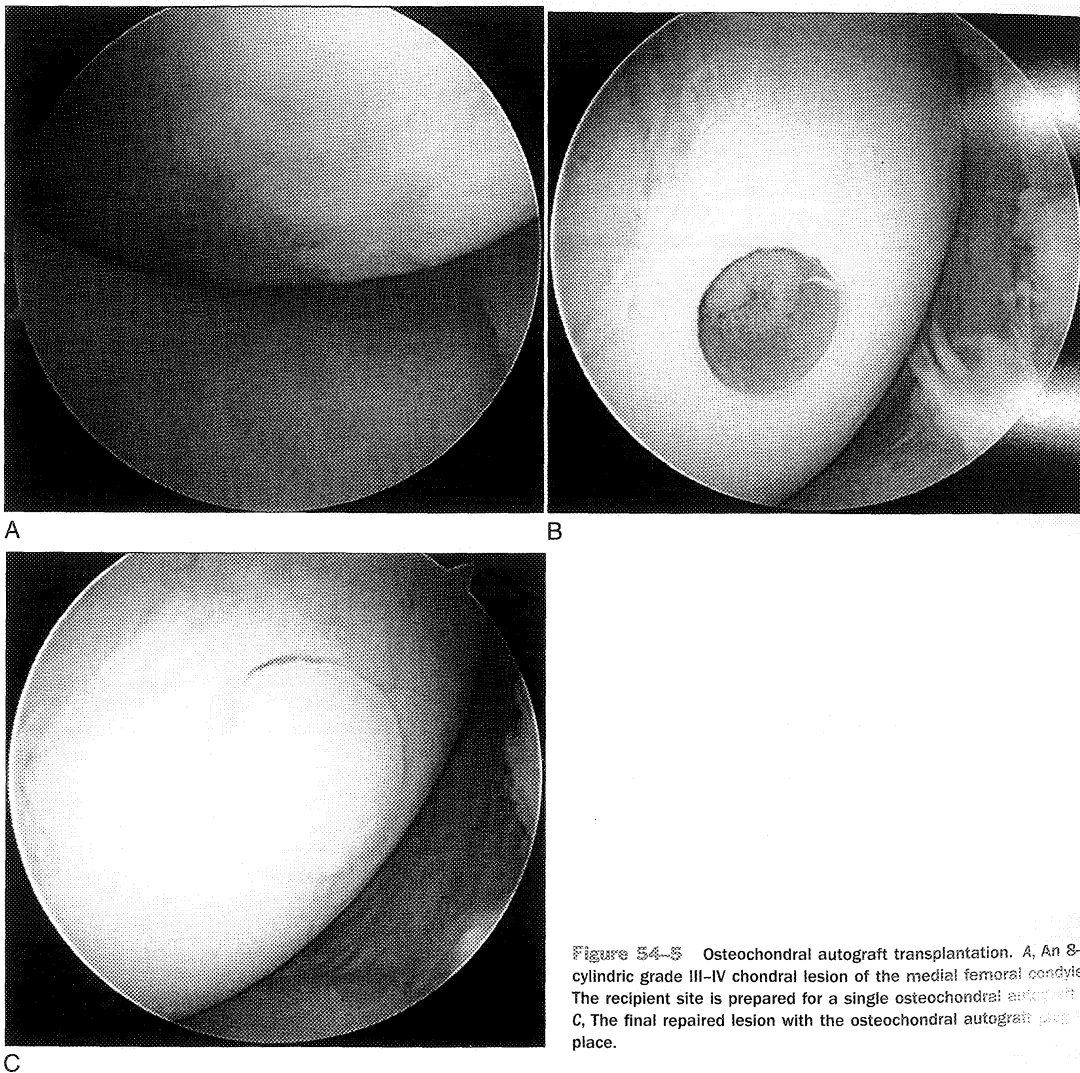
B



C

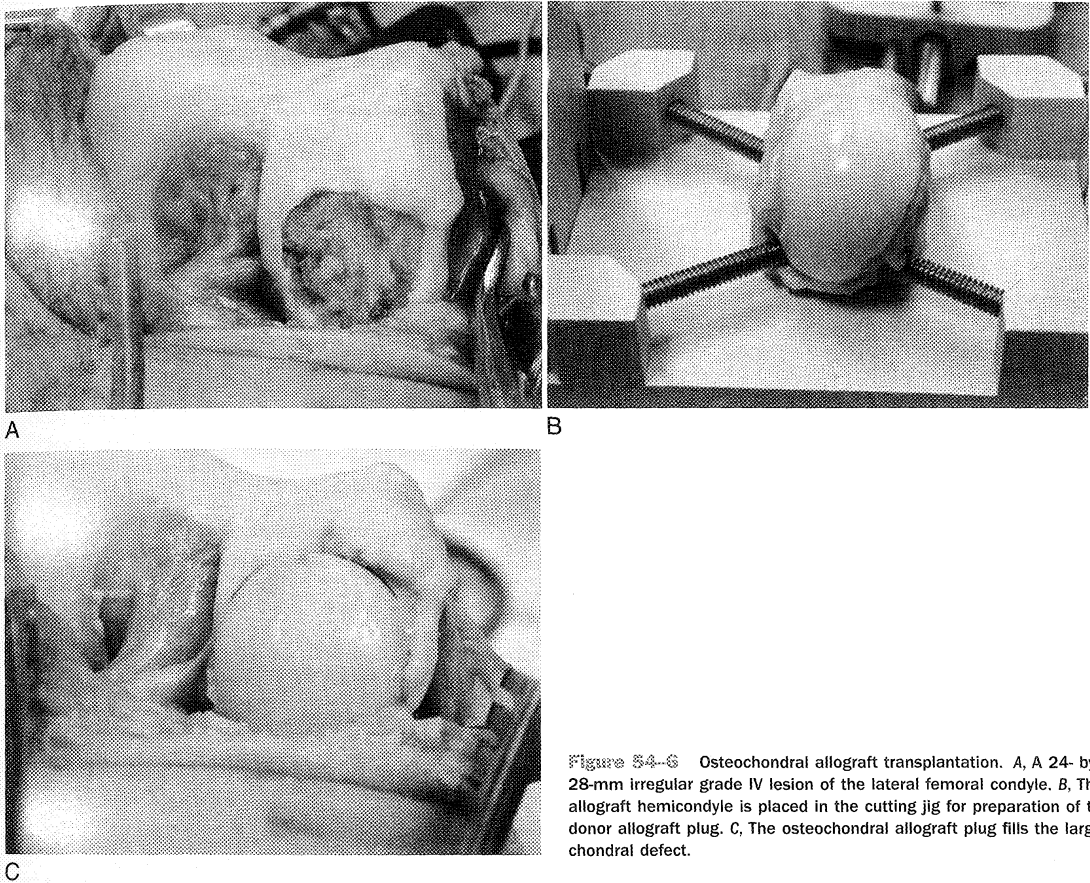
**Figure 54-4** Autologous chondrocyte implantation. *A*, A 20- by 15-mm grade IV chondral lesion of the medial femoral condyle seen via a mini-arthrotomy before preparation for transplantation. *B*, The lesion is prepared for transplantation. *C*, The periosteal patch is sewn in place and sealed with fibrin glue, and the cells are injected.





**Figure 54-5** Osteochondral autograft transplantation. A, An 8-mm cylindrical grade III-IV chondral lesion of the medial femoral condyle. B, The recipient site is prepared for a single osteochondral autograft plug. C, The final repaired lesion with the osteochondral autograft plug in place.





**Figure 54-6** Osteochondral allograft transplantation. **A**, A 24- by 28-mm irregular grade IV lesion of the lateral femoral condyle. **B**, The allograft hemicondyle is placed in the cutting jig for preparation of the donor allograft plug. **C**, The osteochondral allograft plug fills the large chondral defect.

## Results

There are many published reports on the results on each type of treatment for articular cartilage injuries; however, there are very few trials comparing treatment options. In addition, given the varying indications for each procedure, it is important to recognize the difficulty in comparing the results of treatment options for articular cartilage lesions. Population differences among trials, including patient age, lesion size, lesion location, and concomitant pathology, all affect the ability to make comparisons. In addition, it is important to recognize that good results can be achieved with each treatment technique in the appropriate patient population.

The results for articular cartilage debridement are limited to predominantly older studies in arthritic knees. Federico and Reider<sup>8</sup> performed simple mechanical debridement for traumatic and atraumatic chondromalacia patellae and achieved 58% good or excellent results in traumatic patients and 41% good or excellent results in atraumatic patients. Another study found that

superior clinical outcomes were obtained with patellar debridement using bipolar frequency versus a mechanical shaver for isolated patellar chondral lesions.<sup>28</sup> Simple debridement, with removal of loose cartilaginous flaps, can be performed for incidentally discovered asymptomatic lesions or small symptomatic lesions in low-demand patients.

The results for marrow-stimulating techniques, including abrasion arthroplasty and microfracture, are listed in Table 54-2. Overall, the results of abrasion arthroplasty have been unpredictable, and recurrent symptoms often develop within 2 to 3 years. Microfracture has shown promising results as first-line treatment in smaller chondral lesions, but there are few published clinical studies documenting the success rate of the procedure.

The results for cartilage restoration techniques (osteochondral autografts, osteochondral allografts, and autologous chondrocyte transplantation) are shown in Table 54-3. Success has been achieved in a high proportion of patients using each of these procedures, indicating that they are acceptable options for patients with articular cartilage lesions. However, the different patient populations

**Table 54-2****Clinical Results of Marrow-Stimulation Techniques**

Author (Date)	Technique	No. of Patients	Indications	Mean Follow-up	Outcome
Rand (1991) <sup>32</sup>	Abrasion arthroplasty	28	Degenerative arthritis	3.8 yr	39% good/excellent 29% unchanged/fair 32% worse/poor
Bert and Maschka (1989) <sup>2</sup>	Abrasion arthroplasty	59	Degenerative arthritis	60 mo	51% good/excellent 16% unchanged/fair 33% worse/poor
Friedman et al. (1984) <sup>9</sup>	Abrasion arthroplasty	73	Degenerative arthritis	>6 mo	60% good/excellent 34% unchanged/fair 6% worse/poor
Gill and MacGillivray (2001) <sup>12</sup>	Microfracture	100	Focal chondral defect	6 yr	Significant reduction in pain and swelling and improved function
Gill and MacGillivray (2001) <sup>12</sup>	Microfracture	19	Focal chondral defect	3 yr	74% minimal or no pain 63% good/excellent
Steadman et al. (2002) <sup>35</sup>	Microfracture	71	Focal chondral defect	11 yr	Lysholm improved Tegner improved (3.1–5.8, mean)

**Table 54-3****Clinical Results of Cartilage Restoration Techniques**

Author (Date)	Technique	Type/Location	No. of Patients	Mean Follow-up	Outcome
Hangody et al. (2001) <sup>15</sup>	OC autograft	Femur	461	>1 yr	92% good/excellent
		Patella, trochlea	93		81% good/excellent
		Tibia	24		88% good/excellent
Kish et al. (1999) <sup>20</sup> Bradley (1999) <sup>3</sup>	OC autograft	Femur	52	>12 mo	100% good/excellent
		—	145	18 mo	43% good/excellent 43% satisfactory 12% poor
Hangody et al. (1998) <sup>16</sup>	OC autograft	Femur, patella	57	48 mo	91% good/excellent
Aubin et al. (2001) <sup>4</sup>	OC allograft	Femur	60	10 yr	66% good/excellent 20% failure
Bugbee et al. (2000) <sup>5</sup>	OC allograft	Femur	122	5 yr	91% successful 5% failure
Chu et al. (1999) <sup>6</sup>	OC allograft	Femur, tibia, patella	55	6.3 yr	76% good/excellent 16% failure
Gross (1997) <sup>14</sup>	OC allograft	Femur, tibia, patella	123	7.5 yr	85% successful
Garrett (1994) <sup>10</sup>	OC allograft	Femur	17	3.5 yr	94% successful
Meyers et al. (1989) <sup>23</sup>	OC allograft	Femur, tibia, patella	39	3.6 yr	78% successful 22% failure
Peterson et al. (2002) <sup>29</sup>	ACI	Femur	18	>5 yr	89% good/excellent
		OCD	14	>5 yr	86% good/excellent
		Patella	17	>5 yr	65% good/excellent
		Femur, ACL	11	>5 yr	91% good/excellent
Minas (2001) <sup>25</sup>	ACI	Femur, tibia, patella, trochlea	169	>1 yr	85% significant improvement
Micheli et al. (2001) <sup>24</sup>	ACI	Femur, patella, trochlea	50	>3 yr	84% significant improvement
Peterson et al. (2000) <sup>30</sup>	ACI	Femur	25	>2 yr	92% good/excellent
		Patella	19	>2 yr	65% good/excellent
		Femur, ACL	16	>2 yr	75% good/excellent
		Multiple	16	>2 yr	67% good/excellent
		Multiple	16	>2 yr	67% good/excellent
Gillogly et al. (1998) <sup>13</sup>	ACI	Femur, patella, tibia	25	>1 yr	88% good/excellent
Brittberg et al. (1994) <sup>4</sup>	ACI	Femur, patella	16	39 m	88% good/excellent
		Patella	7	36 m	29% good/excellent

ACI, autologous chondrocyte implantation; ACL, anterior cruciate ligament; OC, osteochondral; OCD, osteochondritis dissecans.

and the nonstandardized reporting of results make it difficult to recommend one procedure over another on a scientific basis.

### Complications

Complications following the treatment of articular cartilage injuries are rare and mimic those seen following arthroscopy. The most common complication is incomplete resolution of symptoms or recurrence of pain. In such situations, one would typically advance from a first-line to a second-line treatment. Other major complications include postoperative stiffness, especially with combined procedures (e.g., meniscal allograft transplantation) or those performed on patellar or trochlear lesions. Reparative techniques are unlikely to cause complications other than recurrence of symptoms. Subchondral drilling, however, can cause thermal injury to bone, which is why microfracture is the preferred technique for marrow stimulation.

Reparative techniques have complications unique to each procedure. Some complications associated with ACI, including hypertrophy and detachment, are related to the periosteum. These may be amenable to arthroscopic debridement. In addition, osteochondral grafts can be complicated by dislodgment of the graft from the transplant site, which is rare with the press-fit technique. Additionally, graft collapse can occur through biomechanical overload or biologic failure of the chondral or subchondral components.

### Treatment Algorithm

Rigid adherence to the technical and postoperative requirements of each procedure is critical to the success of any treatment option; however, appropriate patient selection for a specific treatment is paramount to successfully reducing symptoms and improving function. In addition, there is substantial overlap between treatment options, and multiple factors must be considered, adding to the complexity of the treatment decision. Primary factors that must be considered include defect-specific factors such as size, depth, location, and degree of containment. Patient-specific factors include the results of prior treatment, comorbidities (e.g., ligament insufficiency, meniscal deficiency), patient age, current and

desired activity level, patient expectations, and surgeon comfort and experience.

When considering the three major factors in the treatment decision—lesion size, patient demand, and whether this is primary or secondary treatment—an algorithm can be formulated to help guide the decision (see Fig. 54-1). Our preferred treatment for each arm of the algorithm is highlighted. However, these treatment recommendations are commonly modified based on additional patient factors, as mentioned previously.

In general, we prefer simple debridement only in small to medium-sized lesions (<2 to 5 cm<sup>2</sup>) in low-demand patients. For small lesions in higher-demand patients, we initially attempt microfracture as a marrow-stimulating technique. If this fails to resolve symptoms, we progress osteochondral autografting. For medium-sized lesions in this population, ACI is frequently used. Lesions with significant bone loss (>6 to 8 mm) may be treated best with fresh osteochondral allografting.

For larger lesions, our primary treatment is debridement or microfracture for older, lower-demand patients. Although we may also microfracture these defects in higher-demand or younger patients, we often consider performing a biopsy for ACI, given the more guarded prognosis that microfracture has in this population. If there is a significant bony defect or the defect is particularly large, consideration is given to osteochondral allografting as a secondary treatment. Secondary treatment of patients who fail ACI may include osteochondral allografting as well. Although several factors can modify the treatment decision, including coexisting pathology that needs to be addressed concomitantly, having a general approach to these lesions helps determine the optimal treatment (Table 54-4 summarizes the decision-making process).

### Future Directions

Undoubtedly, cartilage restoration techniques will evolve over the next several decades. It is likely that gene therapy techniques will increase the capacity for natural healing of articular cartilage lesions. Alternative tissue techniques will be available to replace damaged articular cartilage, or modifications of existing technology will lead to better results or fewer complications. In addition, continued advances in arthroscopic techniques will allow procedures that are commonly performed through an open arthrotomy to be performed arthroscopically.

**Table 54-4****Summary of Decision Making in Articular Cartilage Injuries****Clinical Evaluation**

History	Pain in ipsilateral compartment; recurrent effusions
Physical examination	Pain in ipsilateral compartment; rule out coexisting pathology
Imaging	Standing radiographs, including 45-degree posteroanterior, and mechanical axis views; magnetic resonance imaging commonly shows articular cartilage lesion
Classification	Outerbridge classification (see Table 54-1)
Associated injuries	Evaluate for knee instability, meniscal deficiency, malalignment

**Treatment Options**

Nonoperative	Low-demand patients, small lesions (<1 cm <sup>2</sup> )
Operative	
Primary repair	Osteochondral lesions
Debridement and lavage	Low-demand patients Small to medium lesions (0.5-3 cm <sup>2</sup> )
Marrow stimulation (microfracture)	Moderate-sized lesions (1-3 cm <sup>2</sup> ) Low- or high-demand patients Fibrocartilage repair tissue
Osteochondral autograft	Small to medium lesions (1-3 cm <sup>2</sup> ) Autogenous tissue with normal hyaline architecture Consider donor site morbidity and availability
Autologous chondrocyte implantation	Medium to large lesions (2-10 cm <sup>2</sup> ) Hyaline-like tissue
Osteochondral allograft	Durable Medium to large lesions (up to hemicondyle) Allograft tissue
Postoperative rehabilitation	Good for lesions with bony defects Protect repair tissue from weight bearing for 6 wk Immediate range of motion Gradual return to weight bearing and activities based on technique

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