HOSPITAL PHYSICIAN®

Volume 2, Part 3 December 2005

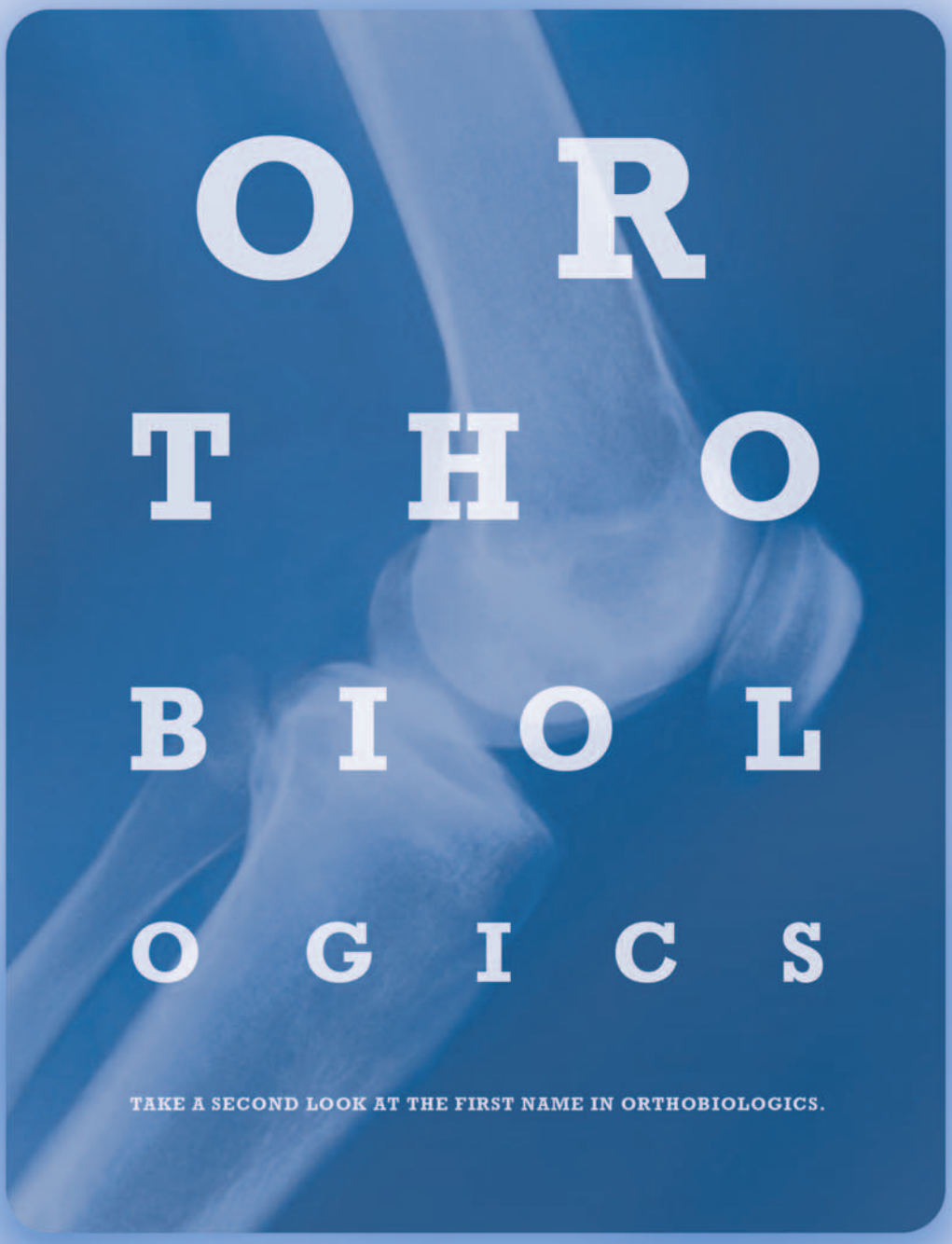
ORTHOPAEDIC SPORTS MEDICINE
Board Review Manual

Osteochondral Injury of the Knee

www.turner-white.com

AHME *Endorsed by the Association for Hospital Medical Education*

Your vision is our new bottom line.



The company long respected for advancing the science of cartilage repair has more to offer than you ever anticipated. An established leader in the development of biomaterials and cell therapies, Genzyme Biosurgery is excited to now be driving the marketing and distribution of Synvisc® (hylan G-F 20). And our pioneering research into novel OA and cartilage repair solutions is destined to redefine the field of orthobiologics. So take a second look at Genzyme Biosurgery. What you see may surprise you.

genzyme
Biosurgery

Genzyme Biosurgery
55 Cambridge Parkway
Cambridge, MA 02142
1-800-901-7251
www.genzymebiosurgery.com

HOSPITAL PHYSICIAN®

ORTHOPAEDIC SPORTS MEDICINE BOARD REVIEW MANUAL

STATEMENT OF EDITORIAL PURPOSE

The *Hospital Physician Orthopaedic Sports Medicine Board Review Manual* is a peer-reviewed study guide for orthopaedic sports medicine fellows and practicing orthopaedic surgeons. Each manual reviews a topic essential to the current practice of orthopaedic sports medicine.

NOTE FROM THE PUBLISHER:
This publication has been developed without involvement of or review by the American Board of Orthopaedic Surgery.

Endorsed by the Association for Hospital Medical Education

Osteochondral Injury of the Knee

Contributors:

Jason M. Scopp, MD
Director, Cartilage Restoration Center, Peninsula Orthopaedic Associates, PA, Salisbury, MD

Bert R. Mandelbaum, MD
Fellowship Director, Santa Monica Orthopaedic and Sports Medicine Group, Santa Monica, CA

Editor:

Andrew J. Cosgarea, MD
Associate Professor, Department of Orthopaedic Surgery, Johns Hopkins University School of Medicine, Baltimore, MD

Table of Contents

Cover Illustration by Photodisc

Osteochondral Injury of the Knee

Jason M. Scopp, MD, and Bert R. Mandelbaum, MD

INTRODUCTION

Chondral and osteochondral injuries are common and typically affect a young, athletic population. In a retrospective review of more than 31,000 knee arthroscopies, Curl et al[1] reported articular cartilage damage in 63% of patients, with more than 60% having a grade III or grade IV lesion. Failure to recognize these injuries can result in long-term disability.

The stresses created during athletic activity place the knee at risk for a range of osteochondral injuries. If injury occurs, it is imperative to recognize osteochondral status as being intimately linked with limb alignment, meniscal status, and ligamentous status. A deficiency in one part of this *functional unit* can have an impact on the others and, in the short term, can lead to a loss of athletic performance. If articular cartilage loses the ability to adapt to repetitive stresses, loss of athletic performance may be followed by the development of chondropenia and ultimately osteoarthritis (OA).

This manual reviews the functional anatomy of articular cartilage, the pathophysiology of osteochondral injury, and the clinical evaluation and management of athletes with osteochondral injuries of the knee. A clinical algorithm is presented as a clinical tool to organize the treatment options for these patients.

ANATOMY AND BIOMECHANICS OF ARTICULAR CARTILAGE

Articular (or hyaline) cartilage is a viscoelastic material that allows variable load bearing by the knee during daily functional and athletic activities. Stress reduction to the subchondral bone and minimization of friction of the articular surface are essential in fulfilling this role. Articular cartilage provides joint surfaces with low-friction wear characteristics that are required for repetitive motion, allowing the athlete to perform consistently at the highest levels of activity and performance without symptoms elicited from the knee joint.

The functional characteristics of articular cartilage depend on its specific structural composition and organization.[2] Normal articular cartilage is composed of an extracellular matrix and chondrocytes. The extracellular matrix consists primarily of water, proteoglycans, and collagens. Type II collagen accounts for 90% to 95% of the total collagen volume, while types V, VI, IX, X, and XI comprise the remaining 5% to 10%.[3] Water content varies from 65% to 85%, depending on the load status and the presence or absence of degenerative changes. During the early phases of OA, the water content can increase to 90%.[3]

The functional organizational unit of articular cartilage is composed of 4 layers: the superficial tangential zone, the middle zone, the deep zone, and the calcified cartilage. The *tidemark* lies between the deep zone and the calcified cartilage and represents the transition from uncalcified to calcified cartilage. The subchondral bone and the calcified cartilage are continuous and are crucial supportive structures involved in load transmission. The resilience of the functional load-bearing unit is essential for durability and smooth joint motion.

PATHOPHYSIOLOGY OF ARTICULAR CARTILAGE INJURY

PROGRESSIVE LOSS OF CHONDRAL INTEGRITY

While the natural history of chondral injury of the knee is not well defined, it is apparent that a loss of articular integrity through injury, pathologic loading, and aging can cause degenerative changes over time. These changes begin as a loss of cartilage volume (chondropenia) and function, followed by development of articular cartilage defects that lead to elevated joint contact pressures and further joint degradation and, possibly, the eventual development of OA. The continuum of cartilage injury can be clinically depicted in a dose-response curve (**Figure 1**). As the athlete competes, a force (dose) is presented to the articular cartilage. If the cartilage is normal, a typical response occurs. However, as chondropenia and articular cartilage defects develop, the ultrastructural properties of articular cartilage can no longer provide an adequate response, leading to symptoms of pain, swelling, and a loss of athletic performance.

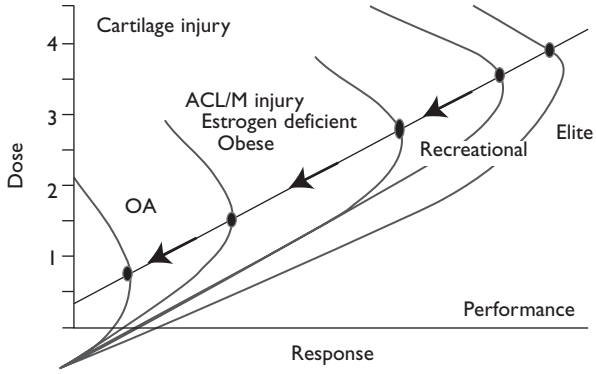


Figure 1. The loss of cartilage integrity represents a continuum with chondropenia and osteoarthritis (OA) at opposite ends of the spectrum. Clinically, as articular cartilage integrity fails and with each step down the continuum, the athlete finds he or she is unable to reach the same levels of performance (response) with the executed activity (dose). ACL = anterior cruciate ligament; M = meniscus.

A principal challenge for the clinician is the lack of accurate measurement tools to objectively identify chondropenia and to assess the pathologic progression of articular cartilage failure. To a limited degree, magnetic resonance imaging (MRI) with T1 fat suppression offers increased sensitivity for assessing cartilage volume and proteoglycan content.[4] Alternative methods, such as molecular markers that sensitively measure cartilage turnover, may also prove to be effective for detecting osteoarthritic changes in the joints at an early stage of the disease.[5] In addition, these markers may be important for the development of new disease-modifying therapies.

SPECTRUM OF ATHLETIC INJURY

Focal Cartilage Lesion

Focal cartilage lesions involve only the articular cartilage, while preserving the integrity of the underlying subchondral bone. These lesions can be partial or full thickness. Partial-thickness lesions do not penetrate the tidemark and have no healing potential. Full-thickness lesions penetrate the tidemark but do not involve the subchondral bone (**Figure 2**). Two classification schemes are available to facilitate the description and clinical tracking of chondral pathology. The Bauer and Jackson classification scheme is based on the authors' study of 167 chondral lesions of the femoral condyle, in which 6 distinct arthroscopic appearances were defined (**Figure 3**).[6] The modified International Cartilage Repair Society (ICRS) articular cartilage injury classification system focuses on the extent and depth of chondral pathology (**Table**).[7]

Symptomatic focal chondral lesions present as pain, swelling, and athletic dysfunction. Diagnosis of chondral lesions is often difficult because patients present with symptoms similar to meniscal injury. Radiographic findings are typically normal unless there is an osseous component to the injury. MRI is important in the diagnosis of chondral injury because it facilitates the diagnosis of concomitant injuries.

The natural history of focal chondral lesions remains unclear. Deeper lesions tend to progress and enlarge over time due to stress concentrations on the rim of the defect.

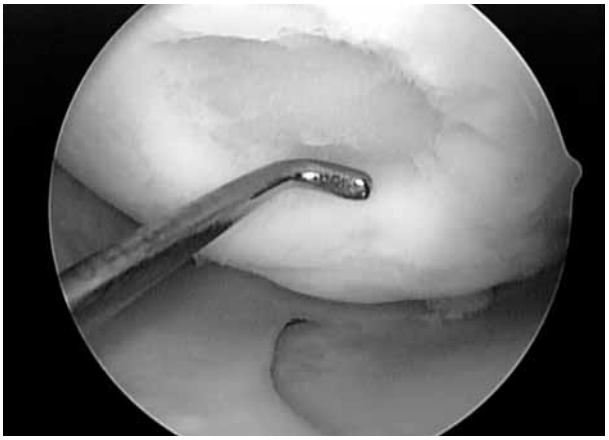


Figure 2. Full-thickness articular cartilage defect. (Adapted with permission from Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:420.)

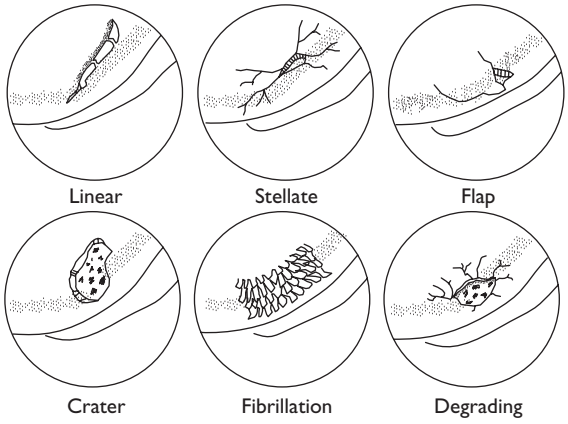


Figure 3. Bauer and Jackson classification scheme for chondral lesions of the femoral condyle. (Adapted with permission from Bauer M, Jackson RW. Chondral lesions of the femoral condyles: a system of arthroscopic classification. Arthroscopy 1988;4:97–102.)

Osteochondral Fracture

Osteochondral fractures are easier to diagnose than

Table. Modified International Cartilage Repair Society Grading System for Chondral Injury

Injury Grade	Injury Description
0	Normal cartilage
1a	Soft indentation
1b	Superficial fissures and cracks
2	Defects extending down to < 50% of cartilage depth
3a	Defects extending down to > 50% of cartilage depth
3b	Defects extending down to calcified layer
3c	Defects extending down to but not through subchondral bone
3d	Delamination
4	Severely abnormal, with penetration through subchondral plate

Data from International Cartilage Repair Society (ICRS) cartilage injury evaluation package. Articular cartilage injury classification. Available at www.cartilage.org/files/ICRS_evaluation.pdf. Accessed 29 Nov 2005.

chondral defects because the bony component can be seen radiographically (**Figure 4**). Osteochondral fractures are often the result of patellar dislocation. Nomura et al[8] evaluated chondral and osteochondral injuries during acute lateral patellar dislocation and found osteochondral fractures in 19%. Of these, 95% involved the medial facet of the patella.

The natural history of osteochondral fractures is unclear. Anatomic reduction and stable fixation allow the underlying bone to heal, but the overlying articular cartilage may degenerate over time as a response to the initial traumatic event.

Osteochondritis Dissecans

In osteochondritis dissecans (OCD), a fragment of subchondral bone and articular cartilage separates from the articular surface. The etiology of OCD is variable and may include a traumatic event, repetitive microtrauma, or a loss of subchondral vascularity. Often no clear etiology exists, suggesting the cause may be multifactorial.[9] The knee is the most commonly affected joint (75%–85% of cases); however, the capitellum of the elbow and the talar dome of the ankle can also be affected.[9,10] Within the knee, the lateral aspect of the medial femoral condyle is involved in 80% to 85% of patients, the lateral femoral condyle in 10% to 15%, and the patella in up to 5%.[11]

Two distinct forms of OCD are recognized, based on

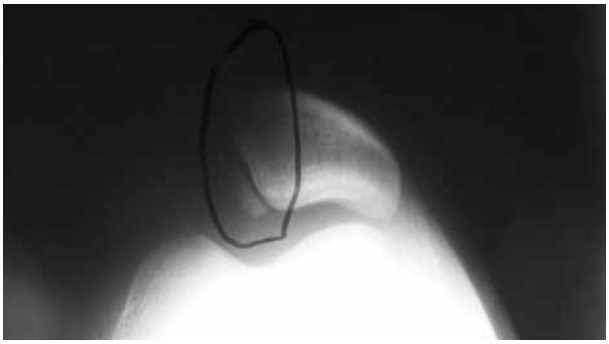


Figure 4. Radiograph of an osteochondral fracture after dislocation of the patella.

patient population affected. The juvenile form affects individuals whose physes remain open.[12] The adult form is seen in adolescents with closed physes and in adults. Because treatment for the juvenile and adult forms often differs, it is important to think of each form as a separate clinical entity. However, the orthopaedic literature often groups these 2 distinct forms together.

Clanton and DeLee[13] described 4 grades of OCD lesions: grade 1 (depressed osteochondral fracture), grade 2 (osteochondral fragment attached by an osseous bridge), grade 3 (detached, nondisplaced fragment), and grade 4 (displaced fragment [loose body]). Cahill and Berg[14] described another classification scheme based on the degree of radioisotopic uptake on scintigraphy: stage I (lesion visible on plain radiographs, but bone scan reveals normal findings), stage II (bone scan reveals increased uptake in the area of the lesion seen on plain radiographs), stage III (stage II findings plus increased isotopic uptake in the entire femoral condyle), and stage IV (stage III findings plus uptake in the adjacent tibial plateau).

Plain radiographs (**Figure 5**) are often negative in the early stages of OCD, which may prompt the clinician to consider other diagnostic modalities. The use of MRI (**Figure 6**) allows for the evaluation of the underlying subchondral bone as well as the presence or absence of fluid behind the fragment. The presence of fluid behind the fragment is suggestive of fragment instability.[15] In 1990, Nelson et al[15] used MRI to predict the grade of OCD lesions and to correlate it with arthroscopic findings; the authors were able to correctly predict the grade of lesion in 11 of 12 patients.

The natural history of OCD in the knee depends on several variables. Physis status, lesion size, and degree of fragment stability contribute to the progression or regression of disease. While we know of no prospective, randomized controlled trial comparing various treatment regimens for juvenile and adult OCD, certain facts as

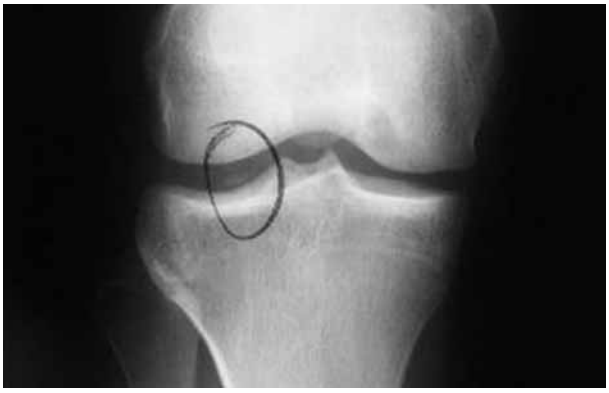


Figure 5. Radiograph demonstrating an osteochondritis dissecans lesion of the posterolateral medial femoral condyle.

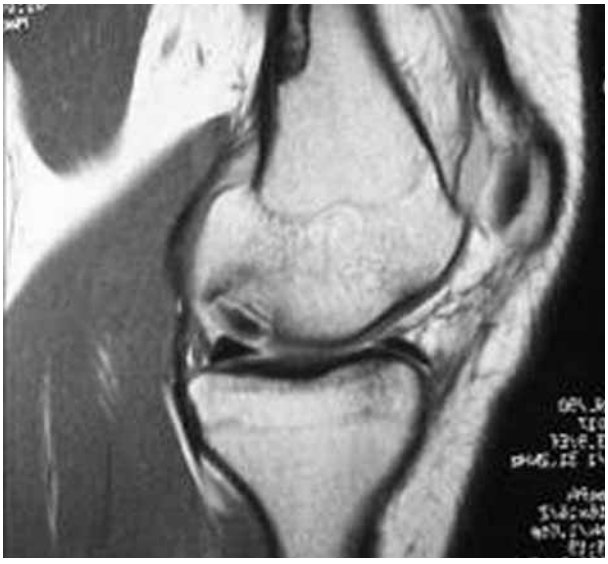


Figure 6. Magnetic resonance image of the same lesion shown in Figure 5.

well as conclusions drawn from the literature can help guide the approach to treatment. For example, it is known that defects of articular cartilage do not heal with normal articular cartilage. Mesenchymal tissue is converted into fibrocartilage, and fibrocartilage has decreased proteoglycan content compared with hyaline cartilage. Fibrocartilage is also less resilient and, therefore, continued trauma to the joint can lead to further degeneration.[16,17] Further, studies show that articular cartilage lesions in the weight-bearing surface of the femur progress.[18] Finally, symptoms and radiographic evidence of gonarthrosis approached 100% in adults with untreated OCD[19]; spontaneous healing typically occurs if the physes are open.

EVALUATION

When evaluating a patient for chondral or osteochondral injury, it is important to appreciate that the *functional unit* of articular cartilage includes alignment (limb and patellofemoral), meniscal integrity, and ligamentous stability. Malalignment, loss of meniscal integrity, or ligamentous instability will increase the load on the chondral surface and may worsen existing defects and/or prevent successful repair or restoration.

HISTORY

An accurate and thorough patient history is essential. The history should include a detailed description of the traumatic episode as well as the type, location, timing, and duration of symptoms. Patients will often complain of nonspecific symptoms including localized pain, swelling, and loss of motion. If the defect involves a detached or loose body, mechanical catching may be described.

PHYSICAL EXAMINATION

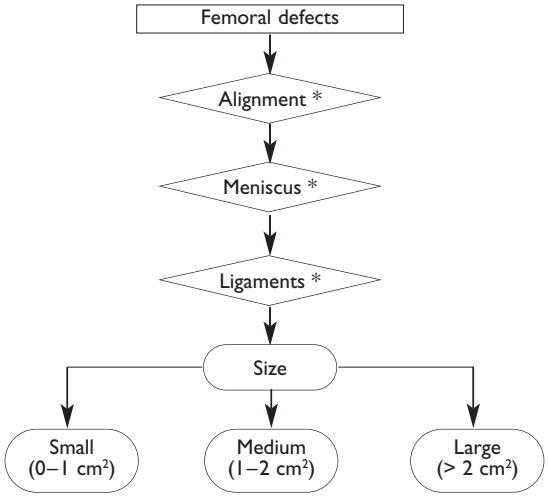
A comprehensive physical examination of the knee provides a functional assessment of articular cartilage status. Important elements to be assessed include range of motion, swelling (soft tissue, joint effusion), and joint line tenderness. Additionally, assessment for varus or valgus malalignment and defects of the anterior and posterior cruciate ligaments provides insight regarding the knee macroenvironment and possible forces transferred through an articular cartilage defect. Each element of the examination should be compared with the asymptomatic side. Examination should also include an observation of gait to evaluate for dynamic pathology as well as adaptive mechanisms used to decrease weight bearing of the joint.

Wilson[20] described a useful physical examination test for OCD of the knee. The Wilson sign is elicited by flexing the knee to 90 degrees, internally rotating the tibia, and then slowly extending the knee. A positive sign is pain at approximately 30 degrees of flexion that is relieved by external rotation of the tibia.

DIAGNOSTIC IMAGING

Plain Radiography

Since the physical findings of cartilage injury are nonspecific, plain radiographs should be used to rule out fractures, evaluate for degenerative changes, and assess alignment. The standard radiographic series includes weight-bearing anteroposterior, 45-degree



1° Treatment options	Small	Medium	Large
Microfracture	++	++	+
OCG	++	++	–
Allograft	–	–	++
ACI	–	–	++

2° Treatment options	Small	Medium	Large
Microfracture	++	+	–
OCG	++	++	–
ACI	–	++	++
Allograft	–	–	++

Figure 7. Treatment algorithm for articular cartilage defects of the femoral condyle, showing primary (1°) and secondary (2°) options. Secondary treatment choices should be considered if primary treatment fails or if other factors preclude the use of a first-line option. *Appropriate treatment is staged to avoid compromise of postoperative rehabilitation. ACI = autologous chondrocyte implantation; OCG = osteochondral grafting; – indicates treatment not recommended; + indicates acceptable treatment; ++ indicates optimal treatment. (Adapted with permission from Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:423.)

flexion posteroanterior, patellofemoral, and lateral views. Additional views include "long-leg" hip-to-ankle films taken to evaluate limb mechanical axis. Because cartilage is not visible on plain radiography, joint space width seen on weight-bearing radiographs has been used as a proxy for cartilage integrity. Despite these techniques, radiographs are unable to detect subtle changes in cartilage morphology associated with articular cartilage defects, chondropenia, and early OA.[21]

Magnetic Resonance Imaging

MRI is becoming increasingly more important in the evaluation of chondral and osteochondral injury. Yulish and colleagues[22] were the first to report on the use of MRI to assess articular cartilage and claimed MRI could reliably diagnose early and late stages of OA in the patella. However, this claim has not been reliably reproduced. Today, there remains a lack of consensus among investigators on what constitutes the normal magnetic resonance appearance of articular cartilage. Investigators have applied a variety of magnetic resonance pulse sequences toward the depiction of articular cartilage and have reached varied conclusions regarding its appearance.[23,24] On high-resolution MRIs, articular cartilage demostrates a multilaminar appearance. However, there is disagreement about the number of layers in normal articular cartilage and the histologic significance of each layer.

MRI has improved dramatically in recent years and now has the potential to replace plain radiography in assessing articular cartilage structural integrity.[21] The use of contrast enhancement through direct or indirect injection increases the ability of MRI to detect focal lesions. A recent preliminary study demonstrated good correlation between an MRI-based quantification of cartilage damage and arthroscopic findings.[25] Other investigators have explored MRI-based measurements of cartilage thickness and volume[26] and the assessment of chondropenia and OA progression. Interobserver agreement, reproducibility, and accuracy remain significant problems for MRI-based evaluation of the severity of knee OA.[27] Moreover, optimal imaging protocols have not been determined.[28] Thus, the potential of MRI as a primary outcome measurement tool for studies of OA has not been realized.

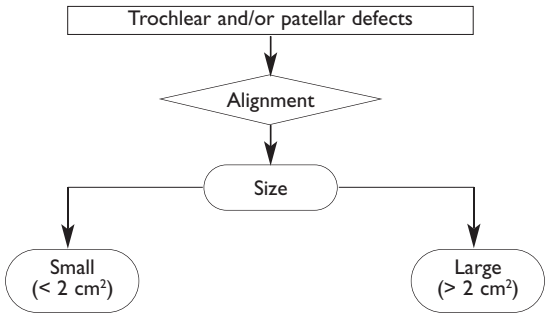
Bone Scintigraphy

Bone scintigraphy can be used as a measure of osteoblastic activity and blood flow. The degree of osseous uptake may correlate with the healing potential of an OCD fragment and serve as a useful prognostic indicator.[14]

MANAGEMENT

ASSESSMENT OF FACTORS INFLUENCING MANAGEMENT

Primary and secondary treatment options for isolated defects of the femoral condyle and for patellar-trochlear defects are summarized in **Figure 7** and **Figure 8**, respectively. The choice of treatment is influenced by several local, regional, and systemic factors that may affect the progression or degeneration of the articular cartilage defect. It is important to define and characterize these factors as the first step in managing a patient with a focal chondral lesion, osteochondral fracture, or OCD.



1° Treatment options	Small	Large
Rehabilitation	++	++
Microfracture	++	++
Lateral release	+	+

2° Treatment options	Small	Large
ACI	+	++
Fresh allograft	–	++
Lateral release	+	+
PF realignment	+	++

Figure 8. Treatment algorithm for patellofemoral articular cartilage defects, showing primary (1°) and secondary (2°) options. Secondary treatment choices should be considered if primary treatment fails or if other factors preclude the use of a first-line option. ACI = autologous chondrocyte implantation; PF = patellofemoral; – indicates treatment not recommended; + indicates acceptable treatment; ++ indicates optimal treatment (Adapted with permission from Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:424.)

In an effort to ensure uniform standards of management, the ICRS has developed a comprehensive method for classifying articular cartilage defects, which is based on an assessment of 9 variables: etiology, defect thickness, lesion size, degree of containment, location, ligamentous integrity, meniscal integrity, alignment, and relevant factors in the patient history (ie, general medical, systemic, and/or family history factors).[7] Etiology is classified as traumatic or chronic. Defect thickness is assessed according to the ICRS grading system shown in the Table; partial-thickness defects that do not penetrate the tidemark have no healing potential. Lesion size dictates treatment approach; small (< 2 cm^2) defects have different treatment options than large (> 2 cm^2) defects. A *contained* defect is surrounded by articular cartilage on all sides; as the degree of containment decreases, consequent loss of joint space is seen on radiographs (**Figure 9**). In terms of location, defects in the weight-bearing surface of the knee may be isolated (*unipolar*) or combined with a defect on the articulating surface (*bipolar*). As previously noted, ligamentous and meniscal integrity and varus/valgus alignment are critical components of the *functional unit* of articular cartilage. An unstable tibiofemoral or patellofemoral joint leads to increased articular cartilage lesions,[29] loss of only 30% of the meniscus increases joint contact pressures by more than 350%,[3] and varus or valgus malalignment increases medial or lateral compartment forces, respectively.[30]

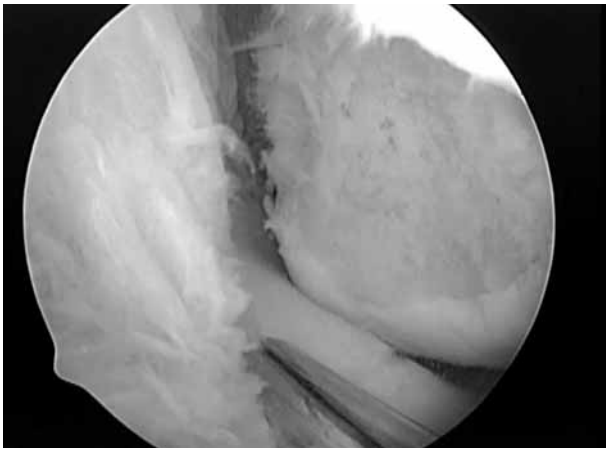


Figure 9. Uncontained full-thickness defect of the lateral femoral condyle after traumatic dislocation of the patella. (Adapted with permission from Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:422.)

TREATMENT OPTIONS FOR SPECIFIC SITUATIONS

Scopp and Mandelbaum[31] have introduced a clinical algorithm to organize management options for articular cartilage defects. The algorithm includes 10 specific "situations" defined by lesion size and depth and associated factors (ie, alignment, ligament and meniscal integrity). Each situation considers the injury category, the current surgical treatment options, and unresolved questions regarding management.

The surgical options presented should be considered only after failure of conservative therapy. Nonoperative modalities include rest (removal of athletic stress) and restoration of joint motion and strength. Limited weight bearing may be required after acute injury. Gait training should be incorporated into a physical therapy regimen. Crutches should not be discontinued until the antalgic gait has been resolved. Painful effusions may be aspirated to facilitate the maintenance of motion. Joint aspiration may also provide clinical data to confirm diagnosis. For example, cruciate ligament tears, traumatic patellar dislocations, and osteochondral fractures frequently present with a bloody effusion.

Situation 1: Meniscal Tears with Partial-Thickness Articular Cartilage Defects

Meniscal tears with partial-thickness articular cartilage defects are the most common type of articular cartilage injury encountered in orthopaedic surgical practice. The primary treatment option for this combined injury is arthroscopic chondroplasty with partial meniscectomy. While there is no easy way to determine the extent of chondroplasty required, it is important to remove any loose flaps of cartilage and edges that appear friable.

Mechanical and thermal chondroplasty may lead to progression of partial-thickness defects. Caffey et al[32] studied the effects of radiofrequency probes on human articular cartilage using 5 different systems. They concluded that when probes were held 1 mm from the chondral surface, no cellular death was seen. However, when the chondral surface was contacted, treated defects demonstrated energy penetration to the subchondral bone and cellular death of adjacent chondrocytes. No statistical differences between monopolar and bipolar devices were noted.[32]

Osteogenic protein 1 (OP-1) has been shown to stimulate synthesis of matrix by normal articular chondrocytes[33] and to possibly up-regulate matrix production in osteoarthritic chondrocytes.[34] The use of OP-1 is currently being investigated for the treatment of partial-thickness chondral defects.

Glucosamine sulfate and chondroitin sulfate are used to minimize the symptoms of OA, although their mechanism of action remains unclear. Chan et al[35] studied bovine articular cartilage explants after physiologic concentrations of chondroitin sulfate and glucosamine sulfate were administered and concluded that the substances can regulate gene expression and synthesis of nitric oxide and prostaglandin E_2, providing a plausible explanation for their purported anti-inflammatory properties.

The role of physical therapy after arthroscopic chondroplasty and partial meniscectomy for every patient is unclear, but it is helpful when strength deficits exist preoperatively. Physical therapy has also been shown to hasten the return to sport in athletes.[36,37]

Situation 2: Small (< 1 cm^2) Full-Thickness Femoral Articular Cartilage Defects without OCD

Unstable chondral fragments should be removed to prevent the development of loose bodies. To fill the defect, however, several techniques have been advocated.

The use of microfracture for small chondral defects has been well studied. With this technique, a small pick is used to penetrate the subchondral bone. This releases mesenchymal stem cells, which can form a fibrocartilage cover over the defect. Steadman et al[38] followed a case series of 72 patients (75 knees) for an average of 11 years following microfracture. The authors found that over the follow-up period, 80% of patients who were younger than age 45 years and had no ligamentous or meniscal injury demonstrated good or excellent results. Results seen with the microfracture technique have led to a question of the durability of fibrocartilage repair in active individuals. Gobbi et al[39] prospectively followed 53 athletes after microfracture (mean follow-up, 72 months) and found that 80% noted a decline in sports activity at final follow-up.

Osteochondral grafting is another technique used to fill a small articular cartilage defect. During autogenous osteochondral grafting, a core of bone and cartilage is immediately transferred to the defect. Because hyaline cartilage is transferred, no fibrocartilage forms, unless multiple plugs are transferred (the spaces between plugs fill in with fibrocartilage).[40]

Situation 3: Medium (1–2 cm^2) Full-Thickness Femoral Articular Cartilage Defects without OCD

Débridement, microfracture, and osteochondral grafting offer technically simple primary treatment options. As the defects get larger, autologous chondrocyte implantation becomes a viable treatment option. This approach is cellular-based. Chondrocytes are harvested at an index operation (knee arthroscopy) and cultured. During a second operation, the cultured chondrocytes are implanted into the articular cartilage defect, beneath a periosteal patch. Peterson et al[41] studied the biomechanics and long-term durability of this repair technique; 51 of 61 patients followed for up to 11 years were found to have good to excellent results on follow-up examinations. Second-look arthroscopies were performed in several of these patients, and indentation probe measurements found the repaired cartilage to have 90% of the stiffness characteristics of the surrounding normal articular cartilage. Biopsy samples obtained from these patients showed hyaline-like characteristics and stained positive for type II collagen.

Situation 4: Large (> 2 cm^2) Full-Thickness Femoral Articular Cartilage Defects with OCD or Avascular Necrosis

Osteochondral grafting, autologous chondrocyte implantation, and osteochondral allografts can be used to manage these large articular cartilage defects. Osteochondral allografts have 2 components (cartilage and bone). The transplanted cartilage is aneural, avascular, and immunoprivileged. The allograft bone is used as a scaffold and vehicle for the transfer of chondrocytes. In a study used to evaluate the viability of chondrocytes at time of implantation, Allen et al[42] found a significant

decrease in chondrocyte viability after 14 days of storage, but 60% to 90% chondrocyte viability has been seen on retrieval studies.

Treatment of OCD. Juvenile forms (open growth plates) that do not heal with conservative treatment and symptomatic adult forms require operative intervention. The basic tenets of surgical treatment include restoration of the congruity of the joint surfaces, enhancement of the local blood supply to the fragment or the crater, rigid fixation of unstable fragments, and protected weight-bearing with motion of the joint as soon as possible postoperatively.[18]

In situ drilling. Subchondral drilling can be used for stable OCD lesions with intact articular cartilage. In this technique, the subchondral bone is channeled in an anterograde or retrograde fashion. Image intensification can be used to facilitate the technique so that the intact cartilage surface is not violated. The goal of subchondral drilling is to promote revascularization.[18]

In situ fixation. Fixation can be accomplished with metallic pins, compression screws, bioabsorbable pins, or osteochondral grafts. If an OCD lesion is intact or partially detached, it can be fixed in situ and stabilized. If a metallic screw is used, it should be countersunk to avoid abrasion on the articulating surface. Several headless screw options exist that allow for compression across the defect. If a metallic implant is used, reoperation is required for removal. The use of autogenous osteochondral grafts to stabilize the lesion eliminates the need for metallic intra-articular fixation and promotes revascularization of the subchondral bone, while addressing any bone loss.[43]

Open reduction internal fixation of displaced fragments. When an OCD lesion is completely detached and there is an adequate bony component to the fragment, open reduction internal fixation can be attempted. The subchondral bed must be meticulously prepared to remove all fibrous tissue. The subchondral bone should then be drilled to enhance revascularization. It is vital to ensure anatomic reduction to decrease abrasive wear on the adjacent chondral surfaces.

Osteochondral autograft and allograft. As discussed, these options allow for immediate transfer of bone and hyaline cartilage to fill a defect devoid of both. The use of allograft allows management of larger bony defects without the morbidity associated with harvesting multiple grafts.[18,42]

Autologous chondrocyte implantation. This technique can be used to manage OCD defects up to 8 mm deep without bone grafting. In a study of 58 patients with OCD treated with autologous chondrocyte implantation, Peterson et al[44] observed 91% good to excellent results at a mean follow-up of 5.6 years. Average defect depth was 7.8 mm, and no bone graft was used.

Fragment excision. Excision of the OCD fragment without other treatment has been reported to be unsuccessful.[14,45] Lesions progress in size and continue to be symptomatic.

Treatment of avascular necrosis. Management of avascular necrosis parallels that of OCD. The defect left after débridement is managed depending upon lesion size and bone loss.

Situation 5: Femoral Articular Cartilage Defects with Malalignment and/or Ligamentous or Meniscal Instability

Again, it is imperative to consider articular cartilage, limb alignment, the meniscus, and the ligaments as functionally interconnected: a defect in one affects the others. Osteotomy, meniscal repair or allograft replacement, cruciate reconstruction(s), autologous chondrocyte implantation, fresh allograft, or osteochondral autograft may be needed to reestablish alignment, chondral integrity, meniscal integrity, and ligamentous integrity.[46]

Situation 6: Patellar and/or Trochlear Articular Cartilage Defects without Malalignment or Instability

Nonoperative treatment of patellofemoral pain can successfully eliminate dynamic malalignment and core weakness.[47] Weakness of hip external rotators and abductors can affect lower extremity control. These factors are known to increase tension in the anterior cruciate ligament and to contribute to patellofemoral pain and instability.[48] Rehabilitation that includes a combination of muscle strengthening, stretching, and patellofemoral taping is beneficial in creating an internal biomechanical environment that encourages maximal tissue healing.[49] Operative intervention for pain without instability or tilt has a low level of success. In a study of 22 knees, Schonholtz et al[50] found improvement in only 1 patient whose only symptom was pain.

Situation 7: Patellar and/or Trochlear Articular Cartilage Defects with Significant Malalignment or Instability (Figure 10)

Despite malalignment and patellofemoral instability, first-line treatment for patellar and/or trochlear articular cartilage defects is rehabilitation, including taping, bracing, and pelvic stabilization. There is no single best option for management of anterior knee instability. The goal is to find the most accurate and least invasive method of treatment.[49]

Instability is caused by many structural and functional

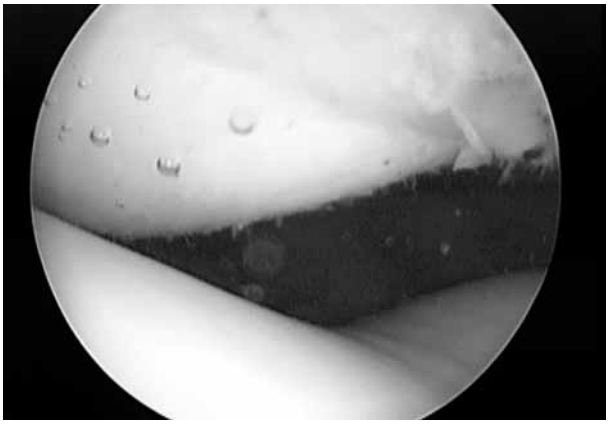


Figure 10. Arthroscopic image showing patellofemoral malalignment. This is demonstrated by contact of the patella with the ridge of the lateral femoral condyle. (Adapted with permission from Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:425.)

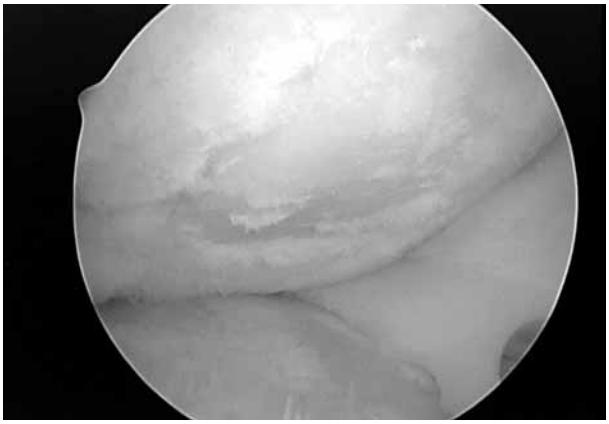


Figure 11. Bipolar lesions with full-thickness defects of the medial femoral condyle and medial tibial plateau. The diminished cartilage volume seen in the surrounding areas is demonstrative of chondropenia. (Adapted with permission from Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:425.)

factors that contribute to the malalignment and dysfunction. Surgical treatment is aimed at addressing specific pathophysiology identified by careful preoperative examination. Lateral retinacular release will provide good results when there is clinical and radiologic evidence of patellar tilt but will not consistently correct subluxation.[51] Proximal medial imbrication may be helpful following lateral release when there is injury to the medial patellofemoral ligament. Distal realignment is reserved for more profound levels of malalignment as well as articular cartilage lesions. When there is anteromedialization of the tibial tubercle, decreased contact pressures are seen in the lateral facet of the patella. There is also a shift in the contact pressures of the patella, both proximally and medially.[52] Therefore, prior to treatment it is critical to understand the location of the articular cartilage defect to avoid increasing the load upon repair.

Cartilage repair options for the patellofemoral joint parallel those for the tibiofemoral joint, with some modifications in technique as well as postoperative regimens (Figure 8). A "demand-match" approach has been described, by which patient-specific factors are matched with cartilage restoration techniques.[53] Patient-specific factors include activity level, physiologic age, and generalized laxity. Knee-specific factors include functional status of the meniscus, alignment, and ligament status. Cartilage-specific factors include procedure cost, primary repair versus salvage, lesion dimensions, lesion depth, and degree of containment. The use of marrow-stimulation techniques, cell-based techniques, or osteochondral transfer must be considered and applied according to treatment recommendations outlined in Figures 8 and 9 and discussed above.

Situation 8: Tibial Articular Cartilage Defects without Malalignment or Instability

Isolated articular cartilage defects of the tibial plateau are uncommon. Treatment is based on lesion size (Figure 8). However, access may require release of the medial collateral ligament as well as detachment of meniscal insertions.

Complex posttraumatic osteochondral defects of the tibial plateau secondary to trauma or avascular necrosis require osteochondral substitution. In 1 study, 89% of patients treated with osteochondral autografts for posttraumatic osteochondral defects of the tibial plateau demonstrated good to excellent results at 2- to 5-year follow-up.[54] Allograft tibial plateau substitution may be used in massive defects. The use of tibial plateau allograft allows for meniscal transplantation at the same time. Concomitant osteotomy must be considered when malalignment is present but should not conflict with postoperative protocols and may need to be staged.

Situation 9: Significant Chondropenia and Early OA (Figure 11)

Nonoperative management of early OA is multifactorial. Nonsteroidal anti-inflammatory medications are effective for managing minor pain and inflammation present in the early degenerative knee. Viscosupplementation, oral chondroprotective agents (glucosamine sulfate, chondroitin sulfate), physical therapy, and unloading

braces provide effective nonoperative management in the chondropenic knee.[55]

Operative options include arthroscopy and osteotomy. The use of arthroscopy in the chondropenic knee is controversial but is most effective when mechanical symptoms exist for less than 6 months and there is neutral alignment with minimal radiographic evidence of joint degeneration.[56] Tibial or femoral osteotomy may maintain the patient's active lifestyle and delay the need for arthroplasty. Unicompartmental and total knee arthroplasty each can provide reliable relief of symptoms but may not permit a return to the activities that the patient values.[55]

Situation 10: Degenerative Meniscal Tears with Late OA

Primary treatment should follow the regimen outlined in situation 9. If the nonoperative measures fail, operative interventions should be considered. The results of arthroscopy for late OA (global grade IV changes) are variable and unpredictable.[55] Indications for the use of arthroscopy, osteotomy, and arthroplasty are variable among practitioners, but each of these options should be part of the therapeutic armamentarium for OA in the aging athlete.

REFERENCES

1. Curl WW, Krome J, Gordon ES, et al. Cartilage injuries: a review of 31,516 knee arthroscopies. Arthroscopy 1997;13: 456–60.
2. Burks RT. Arthroscopy and degenerative arthritis of the knee: a review of the literature. Arthroscopy 1990;6:43–7.
3. Buckwalter JA, Einhorn TA, Simon SR. Orthopaedic basic science: biology and biomechanics of the musculoskeletal system. 2nd ed. Rosemont (IL): American Academy of Orthopaedic Surgeons; 2000.
4. Potter HG, Linklater JM, Allen AA, et al. Magnetic resonance imaging of articular cartilage in the knee: An evaluation with use of fast-spin-echo imaging. J Bone Joint Surg Am 1998;80:1276–84.
5. Verzijl N, DeGroot J, Bank RA, et al. Age-related accumulation of the advanced glycation endproduct pentosidine in human articular cartilage aggrecan: the use of pentosidine levels as a quantitative measure of protein turnover. Matrix Biol 2001;20:409–17.
6. Bauer M, Jackson RW. Chondral lesions of the femoral condyles: a system of arthroscopic classification. Arthroscopy 1988;4:97–102.
7. International Cartilage Repair Society. Cartilage injury evaluation package. Available at www.cartilage.org/files/ICRS_evaluation.pdf. Accessed 29 Nov 2005.
8. Nomura E, Inoue M, Kurimura M. Chondral and osteochondral injuries associated with acute patellar dislocation. Arthroscopy 2003;19:717–21.
9. Clanton TO, DeLee JC. Osteochondritis dissecans. History, pathophysiology and current treatment concepts. Clin Orthop Relat Res 1982;(167):50–64.
10. Aichroth P. Osteochondritis dissecans of the knee. A clinical survey. J Bone Joint Surg Br 1971;53:440–7.
11. Federico DJ, Lynch JK, Jokl P: Osteochondritis dissecans of the knee: a historical review of etiology and treatment. Arthroscopy 1990;6(3):190-197
12. Green WT, Banks HH. Osteochondritis dissecans in children. J Bone Joint Surg Am 1953;35-A:26–47.
13. Clanton TO, DeLee JC. Osteochondritis dissecans. History, pathophysiology and current treatment concepts. Clin Orthop Relat Res 1982;(167):50–64.
14. Cahill BR, Berg BC. 99m-Technetium phosphate compound joint scintigraphy in the management of juvenile osteochondritis dissecans of the femoral condyles. Am J Sports Med 1983;11:329–35.
15. Nelson DW, DiPaola J, Colville M, Schmidgall J. Osteochondritis dissecans of the talus and knee: prospective comparison of MR and arthroscopic classification. J Comput Assist Tomogr 1990;14:804–8.
16. DePalma, AF, McKeever CD, Subin DK. Process of repair of articular cartilage demonstrated by histology and autoradiography with tritiated thymidine. Clin Orthop Relat Res 1966;48:229–42.
17. Mitchell N, Shepard N. The resurfacing of adult rabbit articular cartilage by multiple perforations through the subchondral bone. J Bone Joint Surg Am 1976;58:230–3.
18. Schenck RC Jr, Goodnight JM. Osteochondritis dissecans. J Bone Joint Surg Am 1996;78:439–56.
19. Linden B. Osteochondritis dissecans of the femoral condyles: a long-term follow-up study. J Bone Joint Surg Am 1977;59:769–76.
20. Wilson JN. A diagnostic sign in osteochondritis dissecans of the knee. J Bone Joint Surg Am 1967;49:477–80.
21. Loeuille D, Olivier P, Mainard D, et al. Review: Magnetic resonance imaging of normal and osteoarthritic cartilage. Arthritis Rheum 1998;41:963–75.
22. Yulish BS, Montanez J, Goodfellow DB, et al. Chondromalacia patellae: assessment with MR imaging. Radiology 1987;164:763–6.
23. Lequesne MG, Mery C, Samson M, Gerard P. Indexes of severity for osteoarthritis of the hip and knee. Validation—value in comparison with other assessment tests [published errata appear in Scand J Rhematol 1988;17:following 241 and 1988;73:1]. Scand J Rheum Suppl 1987;65:85–9.
24. Roos EM, Roos HP, Lohmander LS, et al. Knee Injury and Osteoarthritis Outcome Score (KOOS)—development of a self-administered outcome measure. J Orthop Sports Phys Ther 1998;28:88–96.
25. Drape JL, Pessis E, Auleley GR, et al. Quantitative MR imaging evaluation of chondropathy in osteoarthritic knees. Radiology 1998;208:49–55.
26. Eckstein F, Westhoff J, Sittek H, et al. In vivo reproducibility of three-dimensional cartilage volume and thickness measurements with MR imaging. AJR Am J Roentgenol 1998;

170:593–7.
27. McNicholas MJ, Brooksbank AJ, Walker CM. Observer agreement analysis of MRI grading of knee osteoarthritis. J R Coll Surg Edinb 1999;44:31–3.
28. Waldschmidt JG, Braunstein EM, Buckwalter KA. Magnetic resonance imaging of osteoarthritis. Rheum Dis Clin North Am 1999;25:451–65.
29. Dunn WR, Lyman S, Lincoln AE, et al. The effect of anterior cruciate ligament reconstruction on the risk of knee reinjury. Am J Sports Med 2004;32:1906–14.
30. Cerejo R, Dunlop DD, Cahue S, et al. The influence of alignment on risk of knee osteoarthritis progression according to baseline stage of disease. Arthritis Rheum 2002;46:2632–6.
31. Scopp JM, Mandelbaum BR. A treatment algorithm for the management of articular cartilage defects. Orthop Clin North Am 2005;36:419–26.
32. Caffey S, McPherson E, Moore B, et al. Effects of radiofrequency energy on human articular cartilage: an analysis of 5 systems. Am J Sports Med 2005;33:1035–9.
33. Nishida Y, Knudson CB, Eger W, et al. Osteogenic protein 1 stimulates cells-associated matrix assembly by normal human articular chondrocytes: up-regulation of hyaluronan synthase, CD44, and aggrecan. Arthritis Rheum 2000;43:206–14.
34. Loeser RF, Pacione CA, Chubinskaya S. The combination of insulin-like growth factor 1 and osteogenic protein 1 promotes increased survival of and matrix synthesis by normal and osteoarthritic human articular chondrocytes. Arthritis Rheum 2003;48:2188–96.
35. Chan PS, Caron JP, Rosa GJ, Orth MW. Glucosamine and chondroitin sulfate regulate gene expression and synthesis of nitric oxide and prostaglandin E(2) in articular cartilage explants. Osteoarthritis Cartilage 2005;13:387–94.
36. St-Pierre DM. Rehabilitation following arthroscopic meniscectomy. Sports Med 1995;20:338–47.
37. Vervest AM, Maurer CA, Schambergen TG, et al. Effectiveness of physiotherapy after meniscectomy. Knee Surg Sports Traumatol Arthrosc 1999;7:360–4.
38. Steadman JR, Briggs KK, Rodrigo JJ, et al. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. Arthroscopy 2003;19: 477–84.
39. Gobbi A, Nunag P, Malinowski K. Treatment of full thickness chondral lesions of the knee with microfracture in a group of athletes. Knee Surg Sports Traumatol Arthrosc 2005;13:213–21.
40. Hangody L, Fules P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: ten years of experimental and clinical experience. J Bone Joint Surg Am 2003;85-A Suppl 2:25–32.
41. Peterson L, Brittberg M, Kiviranta I, et al. Autologous chondrocyte transplantation. Biomechanics and long-term durability. Am J Sports Med 2002;30:2–12.
42. Allen RT, Robertson CM, Pennock AT, et al. Analysis of stored osteochondral allografts at the time of surgical implantation. Am J Sports Med 2005;33:1479–84.
43. Berlet GC, Mascia A, Miniaci A. Treatment of unstable osteochondritis dissecans lesions of the knee using autogenous osteochondral grafts (mosaicplasty). Arthroscopy 1999;15:312–6.
44. Peterson L, Minas T, Brittberg M, Lindahl A. Treatment of osteochondritis dissecans of the knee with autologous chondrocyte transplantation: results at two to ten years. J Bone Joint Surg Am 2003;85-A Suppl 2:17–24.
45. Cahill B. Treatment of juvenile osteochondritis dissecans and osteochondritis dissecans of the knee. Clin Sports Med 1985;4:367–84.
46. Gillogly SD, Myers TH. Treatment of full-thickness chondral defects with autologous chondrocyte implantation. Orthop Clin North Am 2005;36:433–46.
47. Post WR. Patellofemoral pain: results of nonoperative treatment. Clin Orthop Relat Res 2005;(436):55–9.
48. Mandelbaum BR, Silvers HJ, Watanabe DS, et al. Effectiveness of a neuromuscular and proprioceptive training program in preventing anterior cruciate ligament injuries in female athletes: 2-year follow-up. Am J Sports Med 2005;33:1003–10.
49. Dye SF, Staubli HU, Biedert RM, et al. The mosaic of pathophysiology causing patellofemoral pain: therapeutic implications. Oper Tech Sports Med 1999;7:46–54.
50. Schonholtz GJ, Zahn MG, Magee CM. Lateral retinacular release of the patella. Arthroscopy 1987;3:269–72.
51. Fulkerson JP, Schutzer SF, Ramsby GR, Bernstein RA. Computerized tomography of the patellofemoral joint before and after lateral release or realignment. Arthroscopy 1987;3:19–24.
52. Fulkerson JP, Becker GJ, Meaney JA, et al. Anteromedial tibial tubercle transfer without bone graft. Am J Sports Med 1990;18:490–7.
53. Farr J. Patellofemoral articular cartilage treatment. Common patellofemoral problems. AAOS Monograph Series; 2005:85–98.
54. Ma HL, Hung SC, Wang ST, et al. Osteochondral autografts transfer for post-traumatic osteochondral defect of the knee—2 to 5 years follow-up. Injury 2004;35:1286–92.
55. Cole BJ, Harner CD. Degenerative arthritis of the knee in active patients: evaluation and management. J Am Acad Orthop Surg 1999;7:389–402.
56. Hunt SA, Jazrawi LM, Sherman OH. Arthroscopic management of osteoarthritis of the knee. J Am Acad Orthop Surg 2002;10:356–63.

NOTES

SYNVISC®
HYLAN G-F 20

genzyme
Biosurgery

68452.A Volume 2, Part 3 5/2005