Treatment of Chondral Defects in the Patellofemoral Joint

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INTRODUCTION

Anterior knee pain is a common musculoskeletal complaint seen daily in the practices of primary care physicians, rheumatologists, and orthopedic surgeons. In the past, the term "chondromalacia" was misused interchangeably with anterior knee pain or patellofemoral pain syndrome. The implication that cartilage is the source of symptoms is incorrect as the majority of patients presenting with anterior knee pain do not have cartilage defects and cartilage is aneural. The prevalence of patellofemoral cartilage defects is controversial, as it is unknown what percentage of lesions become symptomatic enough to prompt evaluation. Several studies have reported the presence of high-grade focal chondral defects in 11%-20% of knee arthroscopies. Among these defects, 11%-23% were located in the patella and 6%-15% in the trochlea.[4,11,18] A group investigating asymptomatic NBA basketball players with knee magnetic resonance imaging (MRI) found articular cartilage lesions in 47%, with patellar lesions in 35% and trochlear lesions in 25% of players; however, only approximately half of these defects were characterized as high-grade lesions.[20] These reports emphasize the importance of a thorough history and physical evaluation of the entire kinetic chain from pelvis to foot, a gait analysis, and assessment of all knee structures (tendons, ligaments, and soft tissues) before attributing a patient's symptoms solely to the presence of a chondral defect. Patellofemoral pain, as a subset of anterior knee pain, is typically multifactorial and to achieve success in treatment, each contributing factor requires management individually and in conjunction with the other factors.

The key to successful treatment in this group of patients lies not only in the correct diagnosis of a chondral defect, but more importantly, in the accurate identification of associated pathomechanical factors, such as patella alta, trochlea dysplasia, increased lateral position of the tibial tubercle to the femoral sulcus (previously assessed as a "Q" angle), and secondary soft-tissue problems, such as a weakened or hypoplastic vastus medialis muscle with a contracted lateral retinaculum. These pathomechanics lead to abnormal forces of the patellofemoral joint, which can cause injury to the articular cartilage in itself through repetitive microtrauma or exacerbate the effects of a traumatic event.

A comprehensive discussion of anterior knee pain and patellofemoral pain is beyond the scope of this article. Therefore, the focus will be on the distinct subset of patients presenting with patellofemoral symptoms who have chondral defects. The etiology of these defects is typically multifactorial, but might include focal degeneration, trauma (direct impact or repeated patellofemoral instability), and/or repetitive microtrauma with significant biomechanical abnormalities.

DIAGNOSIS

History

Patellofemoral articular defects frequently present as anterior knee pain; patients often report their pain to be located retropatellar, peripatellar, or in the instance of trochlear defects, the pain at times is located posteriorly in the popliteal area. As the articular cartilage does not have a nerve supply, the pain reported is always secondary. This secondary pain may be from synovial or capsular irrita-

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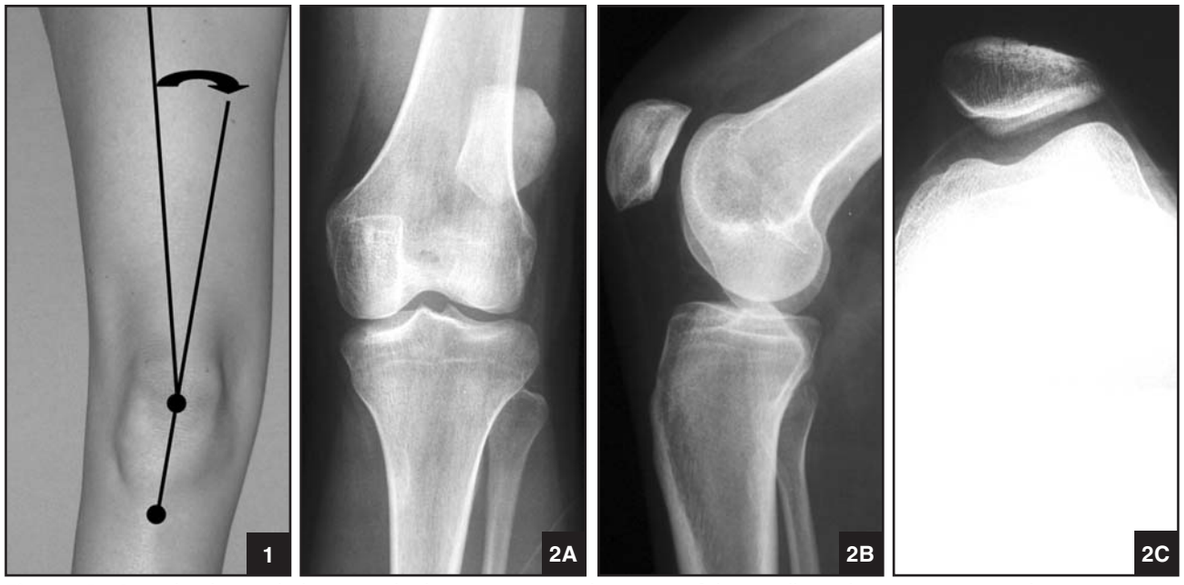


Figure 1. Q-angle: the angle between the lines connecting the center of the patella to the anterosuperior iliac spine proximally and to the tibial tubercle distally. Average Q-angles in asymptomatic patients are 14° in males and 17° in females.[1]
Figure 2. Preoperative radiographs in AP (A), lateral (B), and Merchant (C) views.

tion or due to subchondral bone overload. Thus, in light of this secondary nature of pain, other factors may also contribute, making it difficult to assign a percentage of pain to the cartilage pathology. Large defects can cause clicking or popping, giving way, and activity-related swelling. Standard patellofemoral symptoms are often reported such as increased pain with prolonged flexed knee position and stair climbing. Patients are approximately evenly split in reporting a traumatic versus a more gradual onset of symptoms; sports participation was the most common inciting event associated with the diagnosis of chondral lesions.[4] Patellar dislocation is associated with damage to the articular surface, with chondral defects of the patella seen in up to 95% of patients.[27] Patients often report extended courses of physical therapy, bracing and taping, or prior knee surgery.

Physical Examination

Gait abnormalities, such as intoeing or hip abductor weakness, are frequently seen in this patient population, as is an increase in femoral anteversion and valgus malalignment of the lower extremity. Adaptations in gait are also seen such as hip and knee external rotation and contractures of the hip abductors and iliotibial band. Traditionally, the quadriceps angle (Q-angle) has been used in the evaluation of patellofemoral symptoms (Figure 1). Many different methods of measuring this angle have been reported, and the high interobserver variability makes it of questionable usefulness.[14,15] If used, the Q-angle should be evaluated in both full extension and approximately 30° of flexion, because in some cases, a laterally subluxated patella in full extension can falsely decrease the Q-angle (the patella should be repositioned in the central sulcus before measuring the Q-angle). Quadriceps wasting, especially of the vastus medialis, is common in long-standing patellofemoral symptoms. Recently, more emphasis has been placed on core muscle weakness, especially of the hip abductors, hip extensors, and pelvic stabilizers. Weakness in this group can be demonstrated by asking the patient to single-leg stand on the affected limb, resulting in a pelvic drop on the contralateral side. In addition to poor pelvic support, dynamic internal rotation of the femur and dynamic valgus positioning of the limb can be observed. Activity-related swelling and, in particular, a joint effusion indicate more advanced disease. Palpation of the medial and lateral retinaculum can elicit pain; the lateral structures often are contracted (tested by attempting to reverse patellar tilt), while the medial soft tissues can be attenuated (such as chronic patholaxity of the medial patellofemoral ligament [MPFL]). Patellar mobility, tilt, and subluxation should be assessed and quantified medially and laterally. Catching with mobilization of the patella against the trochlea is suggestive of larger defects. Knee range of motion usually is preserved but may be inhibited by pain or large effusions in acute cases. The J-sign (the patient slowly extends the knee from full flexion, the patella subluxes laterally once it leaves the constraints of the trochlear groove near full extension) is a common finding in normal patients, but if exaggerated it may suggest patholaxity of the medial soft tissues (especially the MPFL).

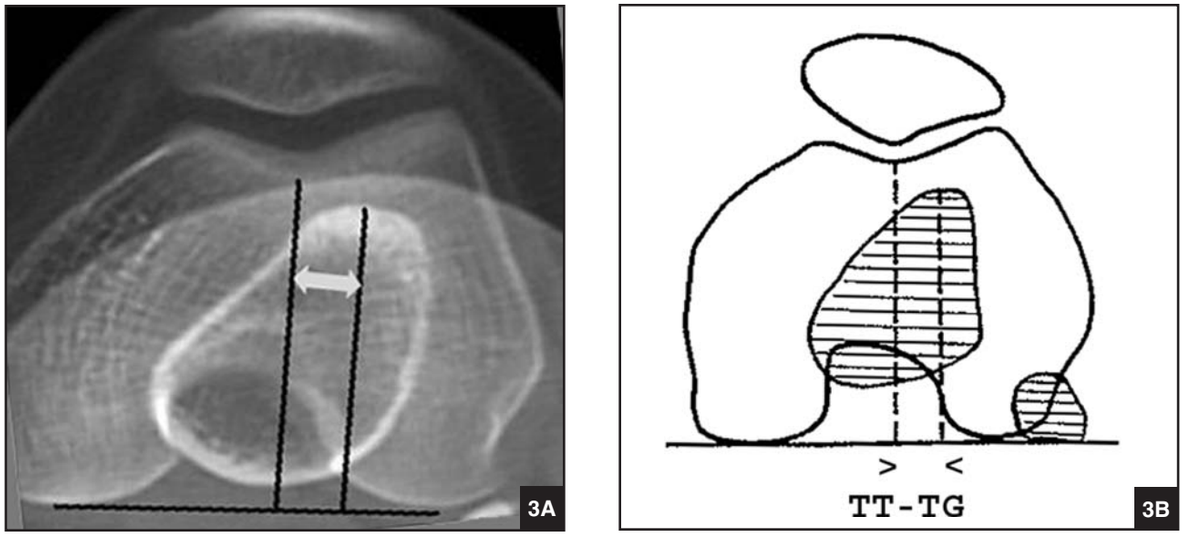


Figure 3. Tibial tubercle to trochlear groove distance (TT-TG): overlay of CT through the trochlea and tibial tubercle (A) with a schematic representation (B).

Imaging

Patient imaging routinely begins by obtaining conventional radiographs as a screening tool: standing anteroposterior (AP), 45° flexion posteroanterior (PA "Rosenberg"), flexion lateral, shallow angle axial (Merchant), and long-leg axial alignment radiographs (Figure 2). These allow assessment of degenerative changes in the tibiofemoral and patellofemoral articulations, trochlear dysplasia, patella tilt, and subluxation. It is important to bear in mind that although the standard Merchant view is useful to determine joint space narrowing or osteoarthritis of the patellofemoral articulation, it is not effective for the assessment of maltracking or trochlear dysplasia. This view is taken at 45° of flexion where the patella is normally well engaged in the trochlea, whereas maltracking usually occurs from entering the sulcus to 30° of flexion. Dejour et al[13] have shown the advantages of a true lateral radiograph in assessing trochlear dysplasia and patellar tilt not appreciated on the Merchant view.

To accurately assess patella subluxation, computed tomography (CT) of the patellofemoral joint is performed with the leg in full extension, once with the quadriceps relaxed and again with the muscle maximally contracted. Computed tomography also allows a more precise evaluation of patellar and trochlear anatomy than the Merchant view. Furthermore, superposition of two CT images, one through the patellofemoral articulation, the other through the tibial tubercle, allows calculation of the tibial tubercle to trochlear groove (TT-TG) distance: the center of the trochlear groove and the center of the tibial tubercle are marked, and the medial-to-lateral distance between the two is measured (Figure 3). A TT-TG distance of $<$15 mm is considered normal; values $>$20 mm are abnormal and should be considered for a tibial tubercle osteotomy.[5,13]

Overall, although we do not obtain CT in all cases, we have found it particularly helpful in obese patients, where an accurate clinical examination often is difficult.

Magnetic resonance imaging assessment of the articular surface has received increased attention due to newly developed high-resolution imaging protocols and also the option of enhancement by intravenous gadolinium. Although arthroscopy remains the gold standard for assessing articular injury, sensitivities and specificities approaching 90% have been reported with MRI protocols using a 1.5 Tesla magnet with appropriate orthogonal gantry tilting to the surfaces of the trochlea and appropriate sequences.[29,31,37,38] It is also possible to use MRI obtained during routine knee evaluation to measure the TT-TG distance (Schoettle et al[33] demonstrated the equivalency of CT and MRI TT-TG measurements) and the Caton-Deschamps measurement of patellar height[10] (alta, infera, normal), thus providing additional information without added cost.

TREATMENT

Conservative Management

The goal of physical therapy is to restore soft-tissue balance in the patellofemoral joint, including muscular and capsuloligamentous balance often remote from the joint. Rehabilitative exercises should include a stretching regimen to restore flexibility of the quadriceps, hamstrings, and iliotibial band, as well as patellar mobilizations as

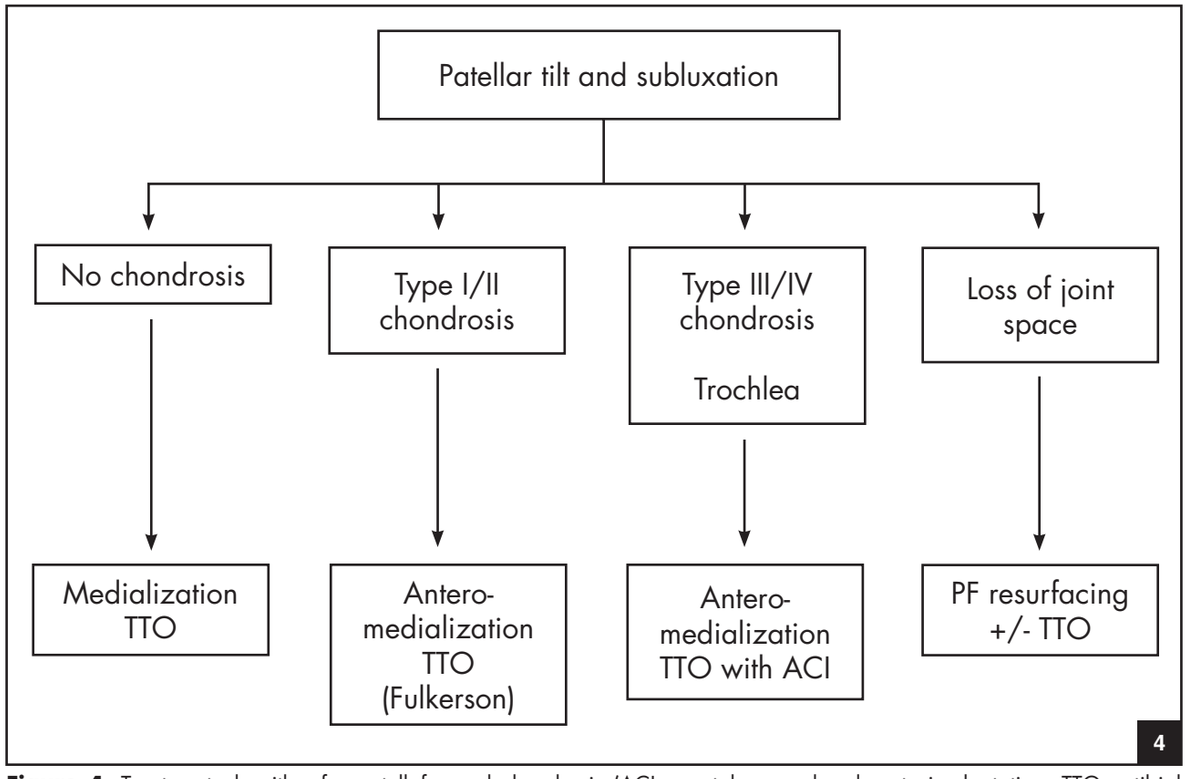


Figure 4. Treatment algorithm for patellofemoral chondrosis (ACI = autologous chondrocyte implantation, TTO = tibial tubercle osteotomy).

needed to optimize capsular structure balance (eg, reverse tilt) of the quadriceps and patellar tendon. After flexibility has been restored, a strengthening program should be instituted, emphasizing the core proximal musculature, including the hip abductors and external rotators, as most patients have previously received too much emphasis on isolated quadriceps strengthening. Gait training should focus on avoidance of an intoeing gait, which results in functional femoral anteversion. Throughout rehabilitation, it is important to protect the patellofemoral articulation by using isometric and short arc closed chain concentric and eccentric muscle strengthening, which is individually designed to avoid specific arcs of pain or loading of cartilage defects. A trial of patellar McConnell taping or patellar bracing to centralize a maltracking patella is worthwhile, especially when symptoms are limited to certain activities, such as athletic endeavors. The patient should understand the comprehensive McConnell approach, which uses the taping to allow a pain-free rehabilitation, ie, the taping is not an end in itself.

Surgical Management

When standard conservative measures have failed, surgical intervention followed by careful rehabilitation often is successful if the underlying pathomechanics can be identified and addressed surgically. Patellofemoral chondral disease represents a spectrum with differing severities of altered loading, subluxation, chondrosis, or arthrosis. This algorithm addresses the different stages of disease in a step-wise fashion of increasing severity adapted from the Fulkerson classification (Figure 4).

Patellar Tilt With or Without Mild Chondrosis (Outerbridge Grade I or II). Surgical release of contracted lateral structures has been overused in the treatment of patellofemoral pain. It is indicated for isolated patellar tilt without subluxation in contracted lateral retinacular structures and decreased patellar mobility. The goal of lateral release is to rebalance patellar tracking and unload the lateral capsular tissues, but it does not significantly alter or unload the lateral trochlea and patellar facet. Early grade I or II chondral changes can be associated with a tilt. A neutral patella may, in rare circumstances, be treated with an isolated lateral release after all rehabilitation efforts have failed, but higher grade lesions should not be treated with lateral release alone. Any partial thickness defects and chondral flaps should be debrided to decrease pain and mechanical symptoms. Notably, patients who achieve the most predictable outcome are those with low grade chondral disease, pain along the lateral patella and retinaculum, and appropriate findings on physical examination.

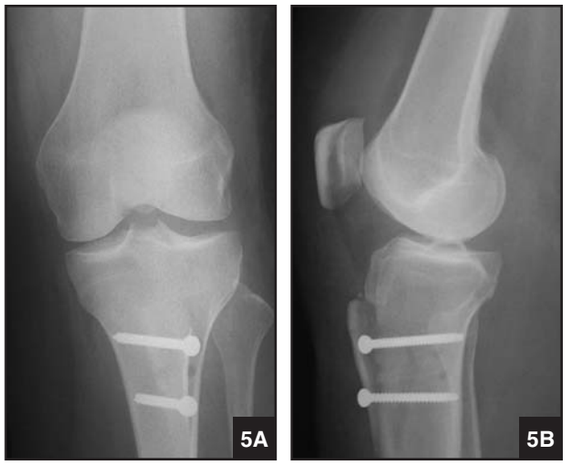


Figure 5. Postoperative AP (A) and lateral (B) radiographs after anteromedialization osteotomy of the tibial tubercle and compression screw fixation.

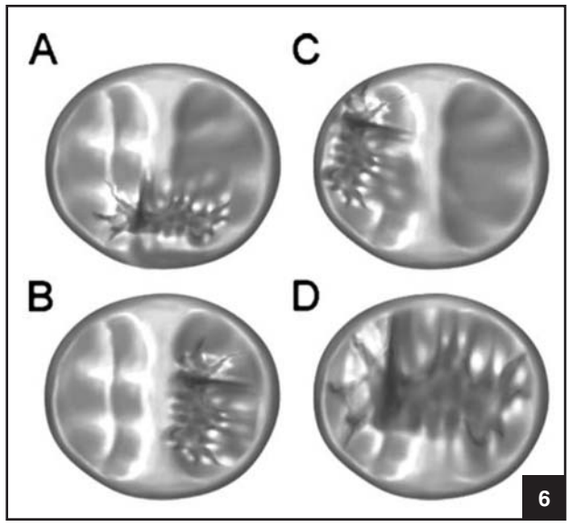


Figure 6. Types of patellar chondrosis based on location. Type I is localized on the inferior pole (A); Type II: lateral facet (B); Type III: medial facet, also frequently associated with a trochlear defect (C); Type IV: injury to the proximal pole (Type IVa) or diffuse injury (Type IVb) (D).

The release can be performed either arthroscopically or open and should be titrated to reverse tilt, and not to achieve the historical "turn up sign." The absolute proximal limit of the release is just distal to the vastus lateralis muscle (approximately the proximal pole of the patella) and should extend distally to the inferior lateral aspect of the patellar tendon. It is important not to release the tendinous portion of the vastus lateralis muscle from the superior lateral patella because of the ensuing weakness and potential medial instability that could result. The superior and inferior lateral geniculate arteries are often cut with this procedure and must be cauterized or ligated to avoid a large postoperative hemarthrosis. Typically, this is performed without a tourniquet; however, if one is used, hemostasis must be achieved after it is deflated.

Isolated lateral release is contraindicated in the presence of patellar instability (subluxation or dislocation) or significant hypermobility of the patella as it could lead to iatrogenic medial instability.

Patellar Tilt and Subluxation With or Without Mild Chondrosis (Outerbridge Grade I or II). Lateral patellar subluxation is multifactorial with components of an abnormally increased TT-TG distance and medial soft-tissue attenuation, either congenital or traumatic as the result of MPFL rupture after patellar dislocation. Over time, lateral positioning with lateral contracture results in overload of the trochlea and patella, because this altered position often decreases patellofemoral contact area. Although the force remains the same, contact stress increases, often to levels with the potential to develop chondrosis.

Surgical correction involves a rebalancing of the abnormal patellar tracking. In patients with normal TT-TG distances and attenuated or ruptured medial structures, repair or reconstruction of the MPFL can be combined with a lateral release as needed to titrate medial-to-lateral glide. Significant distal lateral chondrosis with an increased TT-TG distance, however, is better addressed with a normalizing tibial tubercle osteotomy (Figure 5). The goals of a corrective osteotomy of the tibial tubercle are to normalize the TT-TG distance and thus transfer stress from areas of chondrosis to areas of intact cartilage, and increase patellofemoral contact area by improving congruity, thereby decreasing contact stress. Several variations of tibial tubercle osteotomy have been developed over the years. The Fulkerson anteromedialization tibial tubercle osteotomy has gained popularity in the United States as a modification of the Elmslie-Trillat procedure in that it allows a more aggressive anterior translation than the latter.[30,32] This procedure transfers load from the lateral and inferior poles of the patella by anteromedialization of the tibial tubercle, but increases loads of the proximal and medial poles of the patella and the medial trochlea.[6] It has demonstrated good and excellent clinical outcomes in 87% of patients with chondral defects of the inferior patella pole (type 1) or lateral patella facet (type 2). Conversely, it has demonstrated poor clinical outcomes when the patellar chondrosis was medial (type 3) (good/excellent in 55%), proximal or diffuse (type 4) (good/excellent in 20%), or with central trochlear or patellar involvement (all poor)[28] (Figure 6). When considering the TT-TG distance as a guide for surgical realignment, patellofemoral chondrosis associated with a normal TT-TG distance should be addressed with anterior displacement of the tibial tubercle,

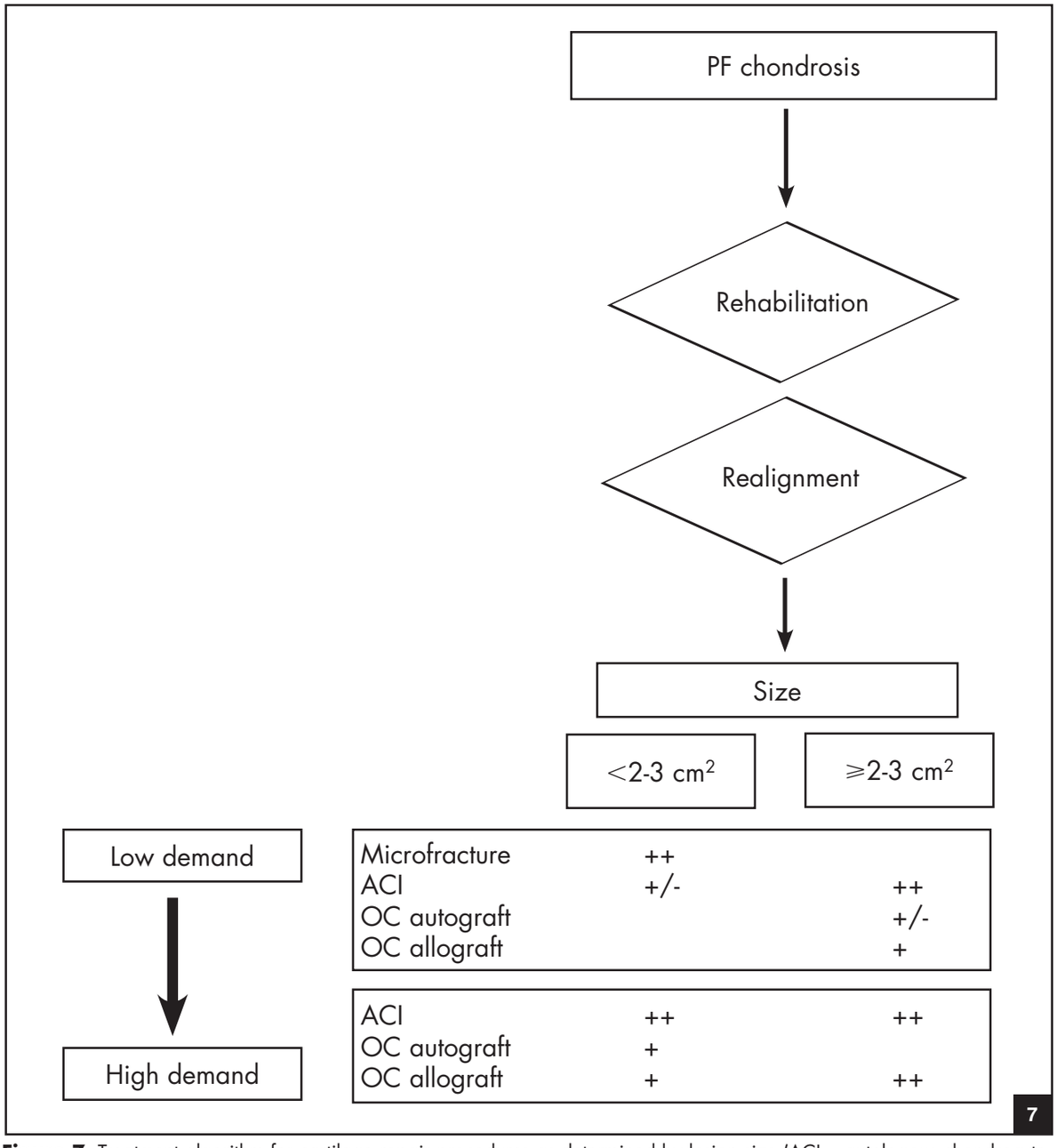
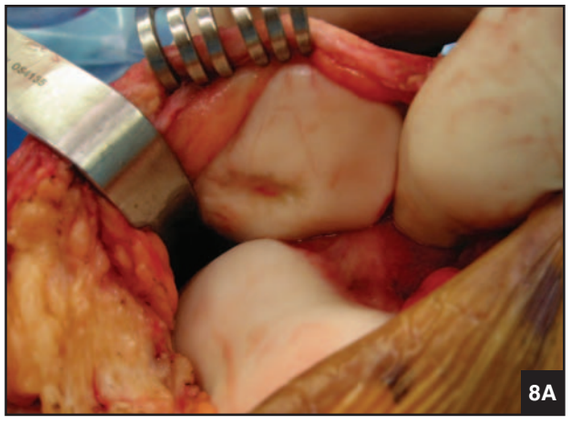


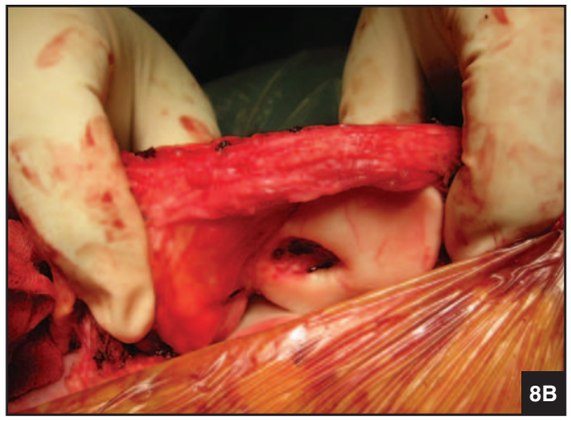
Figure 7. Treatment algorithm for cartilage repair procedures as determined by lesion size (ACI = autologous chondrocyte implantation, OC = osteochondral, PF = patellofemoral).

whereas a TT-TG distance of >20 mm should be treated with an anteromedialization osteotomy.

High-Grade Patellofemoral Chondrosis (Grade III/IV). Soft-tissue realignment and corrective osteotomy have demonstrated encouraging clinical outcomes with careful patient selection and in cases of patellar maltracking associated with chondral defects of the inferior and lateral patella. The experience with treatment of other defect locations by realignment alone, however, has been less satisfactory. High-grade defects, and those that are diffuse, central, or medial, should therefore be considered for a cartilage repair procedure in addition to surgical correction of patellofemoral malalignment and maltracking.

The role of debridement alone is limited to small





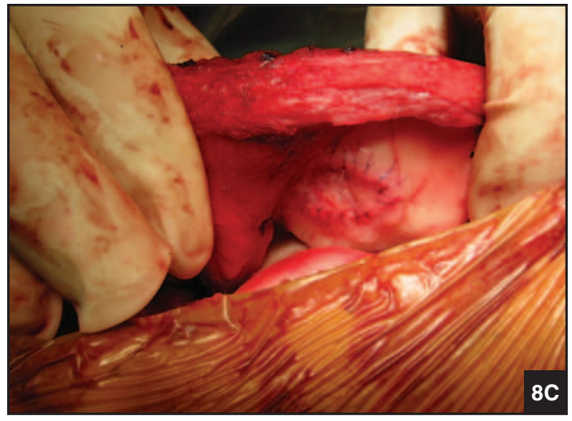


Figure 8. A patellar cartilage defect before (A) and after (B) preparation. The final result with periosteal patch coverage (C).

lesions and those that are coincidental findings whose association with the patient's symptoms has not been established. The treatment of larger lesions is mainly determined by size (Figure 7). Authors have reported good results with microfracture in lesions <2-3 cm^2,[8,21,35] whereas larger lesions and those in high-demand patients were better treated with autologous chondrocyte implantation[23] or osteochondral grafting.[17]

Microfracture

Marrow stimulation techniques, such as microfracture, induce a reparative response by perforation of the subchondral bone. Perforation of the subchondral bone results in the extravasation of blood and marrow elements with formation of a blood clot in the defect. Over time, this blood clot and the mesenchymal cells contained within differentiate into a fibrocartilaginous repair tissue that fills the defect, but may also form bone, resulting in an intralesional osteophyte. Unlike hyaline cartilage, this fibrocartilage predominantly consists of type I collagen and exhibits inferior wear characteristics.

Although not technically challenging, certain aspects of the microfracture technique are critical to obtain good results. These include the thorough debridement of damaged cartilage and soft-tissue scar from the defect, the creation of stable vertical walls of healthy surrounding cartilage, removal of the tide mark or calcified layer, and preservation of a bone bridge of at least 2-3 mm between holes to maintain the integrity of the subchondral plate. This may be performed arthroscopically for trochlear lesions, but due to the difficulty of accessing some patellar locations, it is more important to adhere to proper technique, using a miniarthrotomy if needed, rather than to compromise the result in a zeal to perform the procedure arthroscopically.

Although marrow stimulation techniques result in a repair tissue with inferior wear characteristics, treatment of smaller defects (<2-3 cm^2) results in good mid-term outcomes in approximately 80% of patients.[21,25,35]

Autologous Chondrocyte Implantation

Autologous chondrocyte implantation is a technique originally reported in 1994[9] and is aimed at treating medium to large size chondral defects by in vitro expansion of an autologous chondrocyte biopsy followed by staged reimplantation. An encompassing discussion of autologous chondrocyte implantation is beyond the scope of this article and can be found elsewhere.[24] Autologous chondrocyte implantation in its current US Food and Drug Administration-approved form is a two-stage procedure in which a cartilage biopsy is obtained arthroscopically from a nonweight-bearing area of the knee and expanded in a monolayer culture for several weeks. After successful culture expansion, the patient returns to the operating room for open reimplantation. The chondral defect is carefully debrided of cartilage remnants to create stable, vertical shoulders of surrounding cartilage and a nonbleeding bed of subchondral bone (Figures 8A and 8B). A patch of periosteum is harvested from the proximal tibia

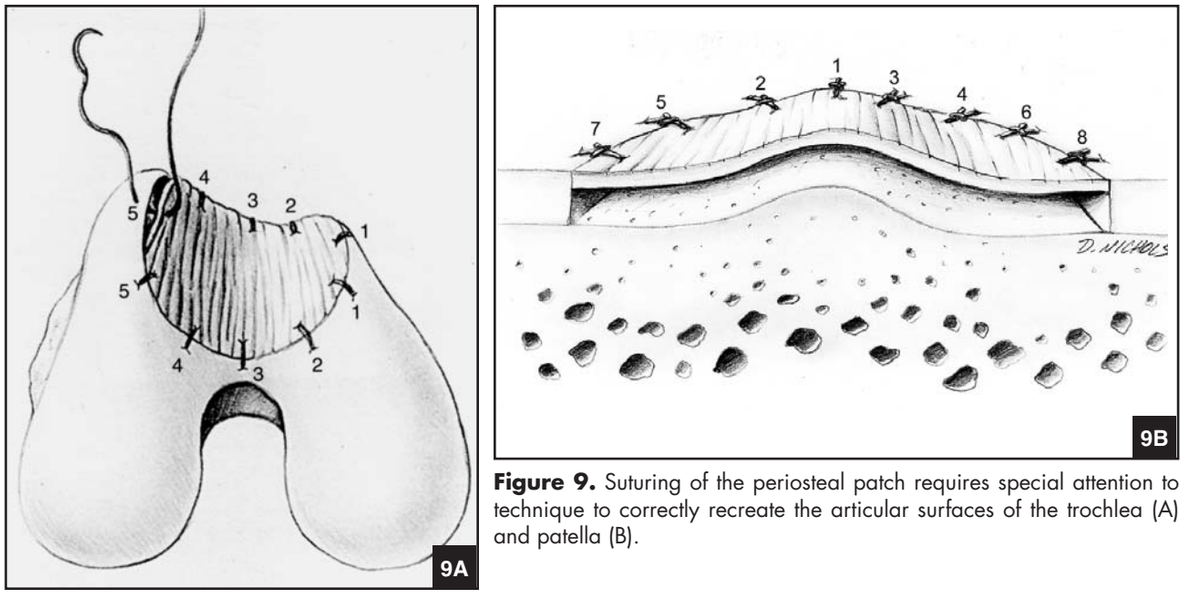


Figure 9. Suturing of the periosteal patch requires special attention to technique to correctly recreate the articular surfaces of the trochlea (A) and patella (B).

and sewn to the adjacent cartilage to cover the defect with the cambium layer facing inwards. Fibrin glue is added to the suture line to achieve a watertight seal after injection of the chondrocyte suspension into the covered defect (Figure 8C).

Repair of trochlear and patellar defects is complicated by the convexity and concavity of the articular surfaces. In trochlear defects, the concave mediolateral curvature is best reconstituted by oversizing the periosteum in this direction by several millimeters. Alternating sutures are then placed on the superior and inferior margins of the defect, adjusting tension of the periosteal patch while working from medial to lateral (Figure 9A). If suturing were to begin in the central sulcus, this aspect of the trochlea would be flattened and result in central graft overload and possibly early breakdown and resultant failure. Early failures of patellar defects were thought to have resulted in part from inadequate debridement of softened and undermined tissue. Current technique calls for the debridement of all unstable tissue, resulting in a defect whose leading and trailing margins are angled so as to not produce an abrupt interface between sutured periosteum and host cartilage. This angle may be more gradual when the articular cartilage is thick, but must be more vertical in thin cartilage to allow for secure suture fixation.

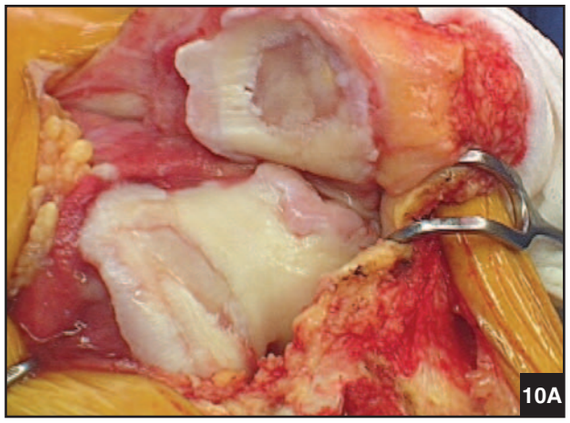
Similar to the trochlea, the contour of the patella must be reproduced by the periosteal patch. This is most easily performed by oversizing the periosteal patch in the mediolateral direction, placing the first sutures at the apex of the median ridge alternating from side to side much like "pitching a tent" (Figure 9B).

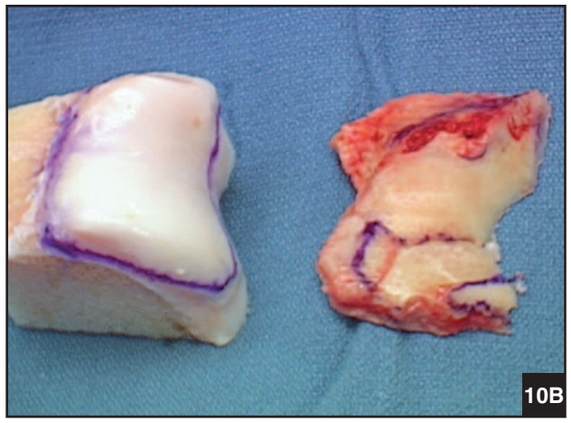
Recent results of autologous chondrocyte implantation in the patellofemoral joint have been encouraging with good and excellent results in up to 85%,[7] even in patients with large defects (average 10 cm^2) who had previously undergone an average of three surgeries.[23] These results demonstrate the importance of concomitant realignment, as early reports on patellofemoral autologous chondrocyte implantation found good outcomes in only 30% of patients without corrective osteotomy.[9]

Osteochondral Grafting

The role of osteochondral grafting techniques such as osteochondral autograft transfer system (OATS; Arthrex, Naples, Fla) or mosaicplasty in the patellofemoral joint is controversial. This technique is complicated by two issues: the difficulty of correctly matching the surface concavity and convexity of the patellofemoral articulation; for patella defects all donor plugs will have less cartilage than the surrounding patella, thus creating a mismatch in the local cartilage bone interface. The technique involves the harvest of osteochondral cylinders from nonweightbearing areas of the ipsilateral knee, preferably the distal and medial trochlea[2] with subsequent transfer to the chondral defect and press-fit fixation. Although good results have been reported in up to 80% of patients by some authors,[16] especially for the treatment of trochlear defects, others have shown failure rates that approached 100% when used for patellar defects.[7] For lesions larger than donor site availability, a macro plug can be obtained from allograft. This has the advantage of possibly matching the contour and the cartilage height, but has the disadvantages discussed in the allograft section below.

Diffuse Patellofemoral Arthritis With Joint Space Narrowing. Once marked radiographic joint space narrowing has occurred, the articular surface has sustained





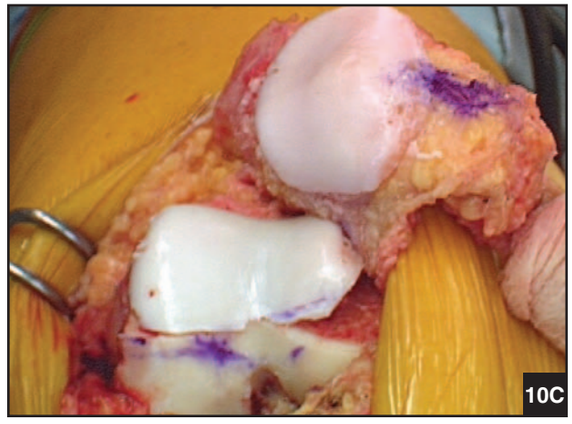


Figure 10. The patellofemoral joint is approached through a conventional arthrotomy, exposing significant damage to the articular surfaces of the trochlea and patella (A). The trochlear defect has been removed, and a similar sized transplant is obtained from an osteochondral allograft (B). Both patella and trochlea have been replaced by osteochondral transplants, which are secured by compression screw fixation (C).

damage that usually is too diffuse and advanced to be successfully treated by realignment procedures alone or the cartilage repair techniques discussed above. Instead, the entire patellofemoral joint surface has to be replaced to reduce pain and mechanical symptoms. Traditionally, this has been achieved by total knee arthroplasty (TKA)[12]; however, two techniques offer a viable alternative.

Allograft Transplantation

Mostly performed as a salvage procedure in young patients with severe arthritis, allograft transplantation uses size, side, and morphology (eg, Weiberg-type shape) matched fresh cadaver grafts to restore the patellofemoral articulation. After the knee has been exposed through a conventional arthrotomy, the chondral defect is removed along with approximately 6-8 mm of subchondral bone (Figure 10A). This may be performed with a circular reamer for incomplete patellar defects, whereas trochlear lesions often are better removed with a burr due to their irregular shape. A similarly sized and shaped graft is fashioned from the donor patella and/or trochlea, and then transplanted in the form of a large osteochondral plug. If stable fixation cannot be achieved through press-fit alone, resorbable pins or compression screws can provide additional support. Alternatively, the patella may be cut in the same manner as for patellar arthroplasty and the trochlear bone can be cut in a single plane from just proximal of the notch to trochlear entrance. The allograft is cut to match this cut surface and the shell allograft is secured with screw fixation (Figures 10B and 10C).[19,36]

Results of patellar and patellofemoral allograft replacement have demonstrated graft survival in 60%-70% of patients with follow-up of up to 10 years.[19,36]

Patellofemoral Prosthetic Arthroplasty

In patients too young for TKA, limited resurfacing of the patellofemoral articulation is possible using a variety of industry patellofemoral arthroplasty implants or by a custom-designed prosthesis, both of which have been investigated as useful interim solutions. In the custom technique, a metal trochlear inset and a standard polyethylene patellar button are implanted through a conventional arthrotomy. The trochlear component is only approximately 2-mm thick and removes little bone, allowing easy conversion to a standard TKA if necessary.

Several studies have reported good and excellent results in 80%-90% of patients with medium-term follow-up of 6-7 years[26,34] and implant survivorship of 58% at 16

years.[3] The main failure mode was progression of tibiofemoral arthritis, necessitating TKA, and uncorrected malalignment.[22] Overall, results were best for patients with isolated patellofemoral arthritis and trochlear dysplasia.

SUMMARY

Patellofemoral disease is one of the most controversial management issues in orthopedic surgery. Nonoperative management as a prerequisite first line treatment is successful in the majority of cases. However, a small subset of patients with persistent pain after adequate rehabilitation will be potential candidates for surgical intervention. Careful assessment of the underlying pathomechanics is critical for a successful outcome; these include malalignment of the extensor mechanism, trochlear dysplasia, soft-tissue imbalance, and chondral damage. As the pathology is multifactorial, the planning and treatment must be multifaceted. With careful patient selection, the options of titrated limited lateral release, restoration of MPFL function, tibial tubercle osteotomy, cartilage repair, and patellofemoral resurfacing provide improved functionality and pain relief for the young patient suffering from patellofemoral pain.

REFERENCES

1. Aglietti P, Insall JN, Cerulli G. Patellar pain and incongruence, I: measurements of incongruence. *Clin Orthop*. 1983;176:217-224.
2. Ahmad CS, Cohen ZA, Levine WN, Ateshian GA, Mow VC. Biomechanical and topographic considerations for autologous osteochondral grafting in the knee. *Am J Sports Med*. 2001;29:201-206.
3. Argenson JN, Flecher X, Parratte S, Aubaniac JM. Patellofemoral arthroplasty: an update. *Clin Orthop*. 2005;440:50-53.
4. Aroen A, Loken S, Heir S, et al. Articular cartilage lesions in 993 consecutive knee arthroscopies. *Am J Sports Med*. 2004;32:211-215.
5. Beaconsfield T, Pintore E, Maffulli N, Petri GJ. Radiological measurements in patellofemoral disorders. A review. *Clin Orthop*. 1994;308:18-28.
6. Beck PR, Thomas AL, Farr J, Lewis PB, Cole BJ. Trochlear contact pressures after anteromedialization of the tibial tubercle. *Am J Sports Med*. 2005;33:1710-1715.
7. Bentley G, Biant LC, Carrington RW, et al. A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *J Bone Joint Surg Br*. 2003;85:223-230.
8. Blevins FT, Steadman JR, Rodrigo JJ, Silliman J. Treatment of articular cartilage defects in athletes: an analysis of functional outcome and lesion appearance. *Orthopedics*. 1998;21:761-767.
9. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med*. 1994;331:889-895.
10. Caton J, Deschamps G, Chambat P, Lerat JL, Dejour H. Patella infera. Apropos of 128 cases [French]. *Rev Chir Orthop Reparatrice Appar Mot*. 1982;68:317-325.
11. Curl WW, Krome J, Gordon ES, Rushing J, Smith BP, Poehling GG. Cartilage injuries: a review of 31,516 knee arthroscopies. *Arthroscopy*. 1997;13:456-460.
12. Dalury DF. Total knee replacement for patellofemoral disease. *J Knee Surg*. 2005;18:274-277.
13. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc*. 1994;2:19-26.
14. France L, Nester C. Effect of errors in the identification of anatomical landmarks on the accuracy of Q angle values. *Clin Biomech (Bristol, Avon)*. 2001;16:710-713.
15. Greene CC, Edwards TB, Wade MR, Carson EW. Reliability of the quadriceps angle measurement. *Am J Knee Surg*. 2001;14:97-103.
16. 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;85:25-32.
17. Hangody L, Rathonyi GK, Duska Z, Vasarhelyi G, Fules P, Modis L. Autologous osteochondral mosaicplasty. Surgical technique. *J Bone Joint Surg Am*. 2004;86:65-72.
18. Hjelle K, Solheim E, Strand T, Muri R, Brittberg M. Articular cartilage defects in 1,000 knee arthroscopies. *Arthroscopy*. 2002;18:730-734.
19. Jamali AA, Emmerson BC, Chung C, Convery FR, Bugbee WD. Fresh osteochondral allografts. *Clin Orthop*. 2005;437:176-185.
20. Kaplan LD, Schurhoff MR, Selesnick H, Thorpe M, Uribe JW. Magnetic resonance imaging of the knee in asymptomatic professional basketball players. *Arthroscopy*. 2005;21:557-561.
21. Kreuz PC, Steinwachs MR, Erggelet C, et al. Results after microfracture of full-thickness chondral defects in different compartments in the knee. *Osteoarthritis Cartilage*. 2006;Epub.
22. Leadbetter WB, Ragland PS, Mont MA. The appropriate use of patellofemoral arthroplasty: an analysis of reported indications, contraindications, and failures. *Clin Orthop*. 2005;436:91-99.
23. Minas T, Bryant T. The role of autologous chondrocyte implantation in the patellofemoral joint. *Clin Orthop*. 2005;436:30-39.
24. Minas T, Peterson L. Advanced techniques in autologous chondrocyte transplantation. *Clin Sports Med*. 1999;18:13-44.
25. Mithoefer K, Williams RJ III, Warren RF, et al. The microfracture technique for the treatment of articular cartilage lesions in the knee. A prospective cohort study. *J Bone Joint Surg Am*. 2005;87:1911-1920.
26. Nicol SG, Loveridge JM, Weale AE, Ackroyd CE, Newman JH. Arthritis progression after patellofemoral joint replacement. *Knee*. 2006;13:290-295.
27. Nomura E, Inoue M, Kurimura M. Chondral and osteochondral injuries associated with acute patellar dislocation. *Arthroscopy*. 2003;19:717-721.
28. Pidoriano AJ, Weinstein RN, Buuck DA, Fulkerson JP. Correlation of patellar articular lesions with results from anteromedial tibial tubercle transfer. *Am J Sports Med*. 1997;25:533-537.
29. Potter HG, Foo LF. Magnetic resonance imaging of articular cartilage: trauma, degeneration, and repair. *Am J Sports Med*. 2006;34:661-677.
30. Preston CF, Fulkerson EW, Meislin R, Di Cesare PE. Os-

teotomy about the knee: applications, techniques, and results. *J Knee Surg*. 2005;18:258-272.
31. Recht MP, Goodwin DW, Winalski CS, White LM. MRI of articular cartilage: revisiting current status and future directions. *AJR Am J Roentgenol*. 2005;185:899-914.
32. Saleh KJ, Arendt EA, Eldridge J, Fulkerson JP, Minas T, Mulhall KJ. Symposium. Operative treatment of patellofemoral arthritis. *J Bone Joint Surg Am*. 2005;87:659-671.
33. Schoettle PB, Zanetti M, Seifert B, Pfirrmann CW, Fucentese SF, Romero J. The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. *Knee*. 2006;13:26-31.
34. Sisto DJ, Sarin VK. Custom patellofemoral arthroplasty of the knee. *J Bone Joint Surg Am*. 2006;88:1475-1480.
35. Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. *Arthroscopy*. 2003;19:477-484.
36. Torga Spak R, Teitge RA. Fresh osteochondral allografts for patellofemoral arthritis: long-term followup. *Clin Orthop*. 2006;444:193-200.
37. Winalski CS, Gupta KB. Magnetic resonance imaging of focal articular cartilage lesions. *Top Magn Reson Imaging*. 2003;14:131-144.
38. Yoshioka H, Stevens K, Hargreaves BA, et al. Magnetic resonance imaging of articular cartilage of the knee: comparison between fat-suppressed three-dimensional SPGR imaging, fat-suppressed FSE imaging, and fat-suppressed three-dimensional DEFT imaging, and correlation with arthroscopy. *J Magn Reson Imaging*. 2004;20:857-864.