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Articular cartilage defects are often seen during knee arthroscopy. If they are symptomatic, they can cause disability comparable with that associated with advanced knee osteoarthritis.

The most difficult aspect of the assessment and treatment of articular cartilage injury is the surgical decision-making process, which involves the timing of cartilage repair surgery and choosing the most appropriate procedure for the individual patient.

**Diagnosis of Articular Cartilage Defects**

Patients with symptomatic cartilage defects typically have activity-related knee pain and swelling, although larger lesions can also cause symptoms of catching or locking. Defects on the femoral condyles cause pain at or close to the joint line with impact activities, such as running or descending stairs, unlike patellofemoral defects, which cause anterior knee pain during stair-climbing, squatting, or rising from a chair. Unfortunately, there are no pathognomonic symptoms for cartilage defects, and frequently these defects coexist with meniscal tears, patellofemoral abnormalities, or early osteoarthritis.

If the patient has had prior knee surgery, reading previous operative reports can provide important clues pertaining to the condition of the articular cartilage at the time of that surgery.

**Physical Findings**

Patients with an articular cartilage defect do not have specific findings on examination. There may be a small or large knee effusion. Knee motion is usually preserved, although displaced osteochondral fragments can cause intermittent locking of the knee. Larger defects, particularly bipolar defects in the patellofemoral compartment, can cause reproducible catching or clicking on knee-motion examination and patellar manipulation. Tenderness on palpation of the femoral condyles and joint lines suggests the presence of synovial inflammation. Additional important findings are overall alignment of the knee, tracking of the patella with flexion and extension, and ligamentous laxity.

**Diagnostic Imaging**

Radiographs are important for the evaluation of malalignment and degenerative changes of the knee joint. The most commonly used radiographs include weight-bearing anteroposterior views in full extension and posteroanterior views in flexion, lateral and patellofemoral views, and a full-length hip-to-ankle anteroposterior view. Sizing radiographs are required before meniscal or osteochondral allograft transplantation, if either treatment approach is selected, and, to account for image magnification, a marker of known size is included in the radiographic view.

High-resolution magnetic resonance imaging (1.5 tesla or greater) is a reliable means with which to evaluate articular cartilage defects. The recent introduction of cartilage-specific imaging protocols has dramatically improved the quality of magnetic resonance imaging scans and allows noninvasive monitoring after cartilage repair procedures.

Computed tomography arthrography is useful for evaluating conditions...
affecting the entire osteochondral unit, such as osteochondritis dissecans, or after failed prior cartilage repair to assess changes in the subchondral bone. Both magnetic resonance imaging and computed tomography allow evaluation of the patellofemoral compartment for trochlear dysplasia, patellar subluxation, and patellar tilt. The tibial tubercle-to-trochlear groove distance can be calculated from axial images to determine the need for (antero)medialization tibial tubercle osteotomy.

**Cartilage Repair**

**Indications**

Not all articular cartilage defects are symptomatic, so careful assessment of other potential causes of knee pain is crucial. Nonoperative treatment with physical therapy, especially for patellofemoral defects, should be continued for at least three to six months in conjunction with activity modification and weight loss. Therapy should include patellofemoral strengthening as well as a four-way hip program. Older patients who are within a few years of age eligibility for knee replacement surgery should also undergo a trial of injection therapy with steroids and/or viscosupplementation. Extensive counseling about how the recovery period after cartilage repair is relatively longer than that after total knee arthroplasty is particularly important in this age group to avoid unrealistic expectations and patient disappointment.

Surgery is considered after establishing that the patient’s symptoms are consistent with a full-thickness (Grade-3 or 4) cartilage defect and after adequate nonoperative management has failed to provide acceptable pain relief. Patients must understand that rehabilitation is extensive and they will not be able to return to activities for a prolonged period after cartilage repair.

**Contraindications**

Patients who smoke\(^4\), are obese (a body mass index of >35 kg/m\(^2\)), have an inflammatory condition, or have an uncorrected articular comorbidity such as knee malalignment, meniscal deficiency, or ligamentous laxity are not good candidates for cartilage repair and usually are advised not to have this surgery. Advanced degenerative change (>50% joint space narrowing) is considered a contraindication to cartilage repair in all but very young patients with intolerable knee pain and swelling and with no other treatment options.

**Treatment Algorithm Based on Published Results of Cartilage Repair Procedures**

As indicated by multiple studies, including randomized controlled trials, treatment strategies for cartilage repair can be based primarily on the location and size of the defect, with age as a potential secondary consideration. The two most common locations for cartilage defects are the medial femoral condyle\(^3\) and the patellofemoral joint. The tibiofemoral and patellofemoral compartments function quite differently; therefore, the treatment algorithms for these two locations differ as well\(^2\) (Table I).

**Tibiofemoral Compartment**

The choice of cartilage repair procedure for an articular cartilage defect in the tibiofemoral compartment is primarily determined by the defect size. In a randomized controlled trial, Knutsen et al. showed that the overall results of microfracture and autologous chondrocyte implantation were similar\(^6\). However, cartilage defects larger than 4 cm\(^2\) treated with microfracture had significantly worse results, while the size of the defect did not affect the results of autologous chondrocyte implantation. The authors therefore concluded that larger defects should be treated with autologous chondrocyte implantation. Basad et al. investigated this issue further with a randomized controlled trial comparing matrix-associated autologous chondrocyte implantation and microfracture, specifically for defects larger than 4 cm\(^2\), and found significantly better results with autologous chondrocyte implantation\(^9\). Another recent randomized controlled trial, by Saris et al.\(^10\), demonstrated that the histological and functional outcomes of autologous chondrocyte implantation were significantly better than those of microfracture even for smaller defects (average, 2.6 cm\(^2\)). Several other studies indicated that microfracture is not efficacious in large lesions\(^11-13\).

Taken together, these studies suggest that treatment should be chosen on the basis of the size of the lesion, but whether to utilize 2, 3, or 4 cm\(^2\) as the criterion for use of a particular treatment approach is a subjective determination by the treating physician and should be individualized to the specific patient. For example, while a 3-cm\(^2\) lesion represents only a portion of the weight-bearing area of the condyle in a 6-ft, 2-in (1.9-m) man, and could be considered small, a similar size lesion in a 5-ft, 2-in (1.6-m) woman likely encompasses the entire weight-bearing area of the condyle and is comparatively large. Unfortunately there are no data to help guide this individualized decision.

**Small Lesions (<2 to 4 cm\(^2\)) in the Femoral Condyle**

Both microfracture and osteochondral autograft transfer for treatment of lesions in this location and of this size consistently produce good and excellent results in 60% to 80% of patients\(^12,18\).

The decision regarding which of these two procedures to use is based on surgeon preference and familiarity with the techniques, the patient’s functional demands, and associated bone loss. While the issue of donor site morbidity after osteochondral autograft transfer is controversial, the harvest of one to two grafts appears safe and provides sufficient material to fill a lesion of 1 to 2 cm\(^2\) size. Athletes have been shown to return to sports activity more quickly after osteochondral autograft transfer than after microfracture treatment (percentage that returns to sports, 93% versus 52%, respectively)\(^16\). Therefore, osteochondral autograft transfer is recommended for smaller lesions, lesions in high-demand athletes, and lesions with associated bone loss, while microfracture is suited for medium-size defects with little or no bone loss in lower-demand patients and they should therefore be reserved for revision situations.
More complex procedures such as autologous chondrocyte implantation and use of osteochondral allografts effectively treat lesions in this size range, but their increased morbidity and cost make them less attractive for these patients, and they should therefore be reserved for revision situations.

**Large Lesions (>2-4 cm²) in the Femoral Condyle**
The results of microfracture treatment of lesions of this size are less encouraging, and microfracture is not recommended for these larger lesions. Some have reported good results with osteochondral autograft transfer in these and even larger lesions, but donor site morbidity is a concern and is a limiting factor.

Both autologous chondrocyte implantation and osteochondral allografts have produced good and excellent results in >70% of patients, but we are not aware of any randomized controlled trials comparing the two procedures. Surgeon and patient preference are factors when deciding between osteochondral allografts and autologous chondrocyte implantation for this group of lesions. Bone loss associated with osteochondral defects can influence the decision: defects deeper than 8 to 10 mm can be treated with autologous chondrocyte implantation, but bone-grafting of the osseous defect needs to be done in a staged or concurrent fashion (sandwich autologous chondrocyte implantation). When there are multiple lesions, especially associated lesions in the patellofemoral joint, autologous chondrocyte implantation is the more flexible treatment option.

### Osteochondritis Dissecans Lesions in the Femoral Condyle
Symptomatic osteochondral defects, such as osteochondritis dissecans lesions, should be repaired whenever possible, although nonoperative treatment of stable lesions can lead to healing in an adolescent with open physes. Stable, nondisplaced lesions can be treated with antegrade or retrograde drilling in an attempt to induce bone-healing. Unstable or displaced defects are better treated with internal fixation with compressive screws. Both metal and resorbable devices can be used, but they should be seated well under the articular surface. Metal implants ideally should be removed prior to full weight-bearing.

Fragment removal alone without subsequent repair provides good short-term pain relief and therefore can be considered in specific circumstances—for example, for in-season athletes, very small defects, and patients unable or unwilling to follow the rehabilitation protocol associated with repair. Long-term follow-up studies, however, have shown high rates of osteoarthritis.

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TABLE I Treatment Algorithm for Cartilage Repair in the Tibiofemoral Compartment*

<table>
<thead>
<tr>
<th>Small Defects (&lt;2-4 cm²)</th>
<th>Large Defects (&gt;2-4 cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteochondral Autograft</td>
<td>Autologous Chondrocyte Implantation</td>
</tr>
<tr>
<td>+ Mature hyaline cartilage</td>
<td>+ No size limitation</td>
</tr>
<tr>
<td>+ Primary bone-healing</td>
<td>+ Arthroscopic procedure</td>
</tr>
<tr>
<td>+ Quicker recovery and return to sports than after microfracture</td>
<td>− Complex rehab. (continuous passive motion and touch-down weight-bearing for 6-8 wk)</td>
</tr>
<tr>
<td>− Technically difficult (mini-open)</td>
<td>− Prolonged delay before return to sports (6-9 mo)</td>
</tr>
<tr>
<td>− Donor-site morbidity with multiple plugs</td>
<td>− Arthroscopy required</td>
</tr>
</tbody>
</table>

*Plus signs indicate advantages, and minus signs indicate disadvantages.
as early as nine years after fragment removal, especially when the lesion was >2 cm². Overall, repair of osteochondritis dissecans lesions results in better outcomes than does fragment removal followed by secondary cartilage repair with osteochondral allograft transplantation. A randomized controlled trial comparing osteochondral autograft transfer with microfracture for treatment of osteochondritis dissecans revealed a better outcome with osteochondral autograft transfer (rate of excellent or good results, 83% versus 63%) at four years. The success rate has been reported to be >80% with autologous chondrocyte implantation and approximately 70% with osteochondral allograft transplantation. Specific treatment recommendations follow the same size-based algorithm discussed previously.

**Patellofemoral Compartment**

The patellofemoral compartment is a difficult location for cartilage repair, and all techniques performed here are less successful than those done on the femoral condyles. The correction of adverse factors such as abnormal tracking of the patella is crucial for success.

While microfracture, osteochondral autograft transfer, and osteochondral allograft transplantation have generally good outcomes in the femoral condyles, there is a growing consensus that they should be used cautiously in the patellofemoral compartment except in very specific cases. Kreuz et al. found only transient improvement after microfracture in the patellofemoral compartment, with worsening after eighteen to thirty-six months. The use of osteochondral autograft transfer in the patellofemoral compartment has shown varying results. Hangody and Füles reported that the results were only slightly worse than those in the femoral condyle, but Bentley et al. reported almost universal failure of osteochondral autograft transfer in the patella. Jamali et al. investigated the use of osteochondral allografts in the patellofemoral compartment and reported 60% good and excellent results.

The first study on autologous chondrocyte implantation demonstrated dismal results in the patellofemoral compartment, with only two of seven patients having a good or excellent outcome. However, with increased understanding of patellofemoral joint biomechanics and more aggressive treatment of tracking abnormalities of the patella, results have improved dramatically. Recent studies have shown successful outcomes of autologous chondrocyte implantation in the patellofemoral compartment in >80% of patients. Even though a defect in the patella is an off-label indication for autologous chondrocyte implantation, this treatment has emerged as the cartilage repair option of choice for all but the smallest defects in the patellofemoral compartment.

**Patient Age and Defect Chronicity**

The results of microfracture treatment in patients over thirty years of age are not as good as the outcomes in younger patients. In general, the same has been found with osteochondral autograft transfer and autologous chondrocyte implantation, although one study of autologous chondrocyte implantation demonstrated low failure rates among patients older than forty-five years. The patient’s age does not substantially affect the results after use of an osteochondral allograft. The effect of defect chronicity on the outcomes of cartilage repair is not well established, although repairs of acute injuries (sustained less than one year before treatment) tend to have better results.

**Surgical Procedures for the Treatment of Small and Medium-Size Cartilage Defects**

Debridement and chondroplasty (removal of degenerated tissue and creation of mechanically stable cartilage with vertical shoulders), generally performed arthroscopically, can be the definitive treatment for cartilage lesions noted as an incidental finding, or for the initial treatment for symptomatic defects prior to consideration of more invasive cartilage repair procedures. Patients recover quickly after a debride-ment and chondroplasty, and they can be fully weight-bearing. Knee pain and swelling should be absent before the patient returns to activities.

Osteochondral autograft transfer requires careful restoration of the curvature of the articular surface and may involve accessory arthroscopic portals or a mini-open approach.

After the diagnostic portion of the arthroscopic procedure has been completed, accessory portals or incisions are created to allow orientation of the instruments perpendicular to the articular surfaces in both the harvest and the recipient location. If a trans-patellar tendon approach is required, the tendon should be split in line with its fibers and repaired at the end of the procedure. Sizing rods are used to determine the size and number of required grafts (Fig. 1). Then, an appropriately sized harvester is selected and an osteochondral plug, usually approximately 10 to 15 mm in length, is obtained, with the graft harvested perpendicular to the articular surface. There are several suitable donor sites for osteochondral plug harvest in the knee. These are located in areas of less weight-bearing in the knee—for example, the medial and lateral trochlear ridge, the intercondylar notch, or the sulcus terminalis on the lateral femoral condyle, which is the transition zone between the tibiofemoral and patellofemoral weight-bearing areas. The graft length and any angulation are noted. A defect harvester of slightly smaller diameter is used to create a recipient hole in the defect area that corresponds in length, diameter, and angulation to the donor plug. Depending on the specific system used, the graft is transferred to a different device or remains in its harvesting tube. The plug is advanced within the tube so that the end is just visible (Fig. 2). The harvester is introduced into the joint and placed over the recipient hole; the graft is then slowly introduced into the defect until it is almost seated (Fig. 3). Subsequently, the harvester is removed, and the graft is seated flush with the surrounding articular surface by gentle pressure with an oversized tamp. Excessive force and use of a mallet should be avoided, since
they cause chondrocyte death.\(^{38,47,48}\) Overall, a slightly recessed graft is preferable to one that is proud, although recessing the graft by \(\geq 2\) mm also has deleterious effects on the cartilage.\(^49\)

The process is then repeated until the defect is filled. Close contact with the surrounding host bone, which is difficult to obtain with multiple plugs surrounding a central plug, is important because, without close contact with the native cancellous bone, integration is compromised and plug resorption and necrosis may follow. The donor site(s) can be left vacant or be filled—for example, with synthetic back-fill plugs, which potentially decrease the risk of postoperative hematoma.

After the surgery, the patient maintains touch-down weight-bearing for a period ranging from four weeks when there is a small defect (one or two plugs surrounded by intact cartilage) to eight weeks when the defect is larger. Continuous passive motion is optional, but if there are concerns about postoperative stiffness, it can be utilized for two to three weeks. Return to athletic activities is delayed for four to six months to allow a return of quadriceps strength and proprioception.

Microfracture is also an arthroscopic procedure, with accessory portals sometimes used to improve access to the cartilage lesion. The initial blood from the treated site is thought to contain more marrow elements than is the subsequent blood, so if multiple arthroscopic procedures are done microfracture should be the last.

With microfracture treatment, a curet is used to trim any soft and fissured cartilage along the defect rim to create vertical shoulders of mechanically stable cartilage (Fig. 4). The layer of calcified cartilage is debrided to improve the volume of regenerative tissue, but the subchondral plate should not be violated.\(^50\) After thorough debridement, multiple holes are created in the subchondral plate with a microfracture awl (Fig. 5). The procedure starts at the rim, directly adjacent to the surrounding cartilage. Ideally, microfracture holes should be spaced approximately 3 to 4 mm apart so that they do not break into each other and destabilize the subchondral plate. The awl should be kept as perpendicular to the subchondral bone as possible, and this may require accessory arthroscopic portals. After completion of the procedure, the tourniquet is deflated and the pump pressure is lowered; bleeding should be observed from all holes.

After surgery, the patient maintains touch-down weight-bearing for a period ranging from four weeks when
the defect is small (<1 cm²) and well-contained to eight weeks when it is larger and/or uncontained. Use of continuous passive motion for six weeks, six to eight hours per day, is recommended. Return to athletic activities is delayed for six to nine months to allow tissue maturation.

**Advanced Surgical Procedures to Address Complex Defects (Large, Multiple, or Osteochondral Lesions)**

Autologous chondrocyte implantation is a two-stage cell-based cartilage repair technique indicated for the treatment of medium-to-large (more than 3 to 4 cm²) full-thickness, focal cartilage defects of the femoral condyles and trochlea of the knee. Ideally, the lesion has a stable rim of intact cartilage (i.e., is contained) to support suturing of the cover membrane. Defects that are deeper than 8 to 10 mm in the subchondral bone require staged or concomitant bone-grafting.

**Arthroscopic Cartilage Biopsy**

Initially, the defect should be evaluated arthroscopically and the joint should be assessed for ligamentous instability, abnormality of patellar tracking, or meniscal deficiency. The number, size, and location of chondral defects should be noted. The opposite articular surface should be carefully evaluated for a bipolar (kissing) lesion. The quality and thickness of the surrounding articular cartilage are assessed to determine whether the lesion is contained or un-contained. If the defects are amenable to autologous chondrocyte implantation, full-thickness cartilage is harvested with a sharp gouge from the superolateral aspect of the intercondylar notch (Fig. 6) or the medial aspect of the proximal part of the trochlea. The harvested cartilage should be approximately 5 mm wide and 10 mm long and weigh 200 to 300 mg. The cartilage is placed directly in sterile transport medium and shipped for cell culturing. To obtain cultured cartilage cells, the cartilage is enzymatically digested and the approximately 200,000 to 300,000 cells contained in the cartilage sample are amplified to approximately 12 million cells per 0.4 mL of culture medium. This takes approximately six weeks, but it can be interrupted after two weeks by cryopreservation if necessary. Up to 1.2 mL (48 million cells) can be obtained through standard culture, but additional cells can be obtained with additional cell passage.

**Autologous Chondrocyte Implantation**

Adequate exposure is critical to allow proper defect preparation and suturing. A limited medial or lateral parapatellar arthrotomy is used for isolated lesions of the femoral condyles. Multiple and larger femoral lesions require a more extensile approach, and tibial plateau lesions frequently require detachment of the anterior meniscal horn insertion, with repair after grafting is complete. A bent Hohmann retractor placed in the intercondylar notch will displace the patella to the contralateral side to assist with exposure.
Defect Preparation
The defect (Fig. 7) must be cleaned of all degenerated tissue to achieve a stable rim of healthy cartilage with vertical shoulders. First, the defect is outlined with a fresh scalpel down to the subchondral plate. The degenerated cartilage is debrided with small curets, with removal of all unstable or undermined cartilage (Fig. 8), unless this removal changes a contained lesion into an uncontained one. If that is about to happen, the surgeon should leave a small rim of degenerated cartilage to sew into, rather than using bone tunnels or suture anchors. He or she should debride the calcified cartilage but not the intact subchondral plate so that bleeding is minimized. Bleeding from the subchondral bone allows a mixed stem-cell population into the chondral defect, diluting the end-differentiated chondrocytes grown in vitro. Bleeding can also compromise the mechanical stability or even rupture the cover membrane or suture line. Minor punctate bleeding is frequently encountered but can usually be controlled with thrombin or epinephrine-soaked sponges, fibrin glue, or electrocautery. After debridement, the defect is templated with aluminum foil or glove packaging paper (Fig. 9). If periosteum is used to cover the chondrocyte graft, the template should be oversized by 1 to 2 mm in all dimensions to accommodate for shrinkage of the periosteal flap after harvest.

Patch Cover
Two options are available to cover the defect. Use of a periosteal patch is the only FDA (Food and Drug Administration)-approved method, but these patches have been abandoned in Europe because of their propensity to hypertrophy, with arthroscopic debridement of areas of periosteal hypertrophy required in 20% to 50% of patients. Good outcomes and lower reoperation rates have been reported with the use of a collagen membrane. Coverage of a cartilage defect with a collagen membrane is an off-label use of the membrane and has to be discussed with the patient.

The recommended site for procurement of periosteum is the proximal-medial aspect of the tibia, just distal to the insertion of the pes anserinus. Fibers of the sartorius blend with the periosteum more proximally, potentially compromising graft quality. The periosteum is exposed, and a periosteal patch is outlined on the basis of the template. The superficial surface and orientation of the patch are marked with a pen. The periosteum is divided with use of a fresh scalpel blade and is then mobilized with a small sharp periosteal elevator. After the patch has been harvested, it should be spread out on a moist sponge to avoid desiccation and shrinkage. If a tourniquet was used, it can be deflated at this point and for the remainder of the procedure.

The cartilage defect is dried, and the periosteal patch is placed over the defect with the cambium layer facing toward the defect bed. The patch is gently unfolded and is stretched with...
nontoothed forceps; it is then trimmed to fit the defect. The patch is sutured in place with 6-0 resorbable suture on a cutting needle that was immersed in sterile mineral oil or glycerin for better handling. The suture is placed first through the patch and then through the surrounding articular cartilage, exiting approximately 3 mm from the defect edge; the patch edge is everted slightly to provide a better seal against the defect wall. The knots are tied on the patch side, seated below the level of the adjacent cartilage (Fig. 10). Interrupted sutures are placed on each side of the patch (at 3, 6, 9, and 12 o’clock), with the tension of the patch adjusted after each suture and the patch trimmed as needed so that it is neither loose enough to sag into the defect nor so tight that it cuts out of the sutures. Ideally, the patch should recreate the contour of the articular surface. The surgeon should leave an opening in the most superior aspect of the patch that is wide enough to accept an angiocatheter to inject the chondrocytes. The suture line is waterproofed with fibrin glue. The suture line is tested by slowly injecting saline solution into the covered defect with a tuberculin syringe and a plastic 18-gauge angiocatheter. Leakage is addressed by sealing with additional sutures or fibrin glue. The saline solution should be aspirated to prepare the defect for injection of the chondrocyte suspension.

With use of sterile technique, the chondrocytes are aspirated from the transport vials through a flexible, 2-in (5-cm) plastic 18-gauge angiocatheter. The angiocatheter is placed into the defect through the residual opening and, as the angiocatheter is slowly withdrawn, cells are injected until the defect is filled with the cell suspension (Fig. 11). One or two additional sutures and fibrin glue are used to close the injection site.

Rehabilitation
There are three phases of rehabilitation after autologous chondrocyte implantation. These follow the progress of tissue maturation: proliferation, transition, and remodeling. Initially, the cell graft is soft and not strongly integrated with the surrounding cartilage and underlying bone. It is vulnerable to shear and compression forces, which need to be avoided while the graft is protected. Continuous passive motion is used for six to eight hours per day for six weeks after the surgery, and the patient utilizes crutches with toe-touch weight-bearing. The exact rehabilitation protocol
depends on the defect location: patients with condylar defects are allowed a full active range of motion, while those with patellofemoral defects are limited to passive extension and active flexion. Likewise, the range of the continuous passive motion is advanced as tolerated to 90° for patients with condylar lesions but is held at 40° for those with patellofemoral defects. To decrease the risk of arthrofibrosis, the patient dangles the leg over the side of the bed at least three times per day to achieve 90° of flexion by three weeks after the surgery. During the second phase (beginning seven weeks after the surgery), the patient gradually transitions to full weight-bearing and begins closed-chain strengthening exercises. The third phase (beginning at twelve weeks) starts a slow return to activities of daily living, with progression of strengthening and proprioceptive exercises. Patients are restricted from impact activities such as running for twelve to eighteen months and from cutting sports for at least eighteen months.

**Osteochondral Allograft Transplantation**
The use of osteochondral allografts dates to the early twentieth century but was done sparingly prior to the last decade. The purpose of osteochondral allograft transplantation is to implant fully developed tissue capable of withstanding normal load transmission. The hyaline cartilage already has a mature matrix, and the intact subchondral bone remains integrated at the cartilage-bone interface. The matrix and chondrocytes have been shown to survive in long-term recovery studies. Furthermore, the allograft chondrocytes are immunoprivileged, in part as a result of protection by the avascular and dense cartilage matrix. Failure of allografts does not appear to be a result of an immune reaction to the transplanted cartilage but rather seems to be due to failure of the transplanted bone. The allograft articular cartilage is fully developed at the time of implantation and does not need to heal. The allograft bone needs to heal and acts as a scaffold that incorporates over time by creeping substitution. Successful integration into the host bone is critical for the graft’s ultimate function and load transmission. However, the transplanted bone generates an immunologic reaction primarily from remaining donor blood elements. Patients with a positive result on anti-human leukocyte antigen antibody screening after osteochondral allograft transplantation have greater surrounding bone edema and more abnormal graft marrow than do those patients with a negative result of the screening. For this reason, one should
transplant as little bone as necessary and remove as many donor marrow cells as possible through lavage prior to transplantation. The FDA began regulatory oversight of the use of cadaveric tissue in 1993. Safety guidelines established by the American Association of Tissue Banks (AATB) advocate extensive serologic, bacterial, and viral testing; donor screening; procurement and storage requirements; and graft quarantine until negative testing results are ensured. The risk of HIV transmission is estimated to be approximately one in 1.6 million, and there have been no reported cases of this route of disease transmission since the late 1980s. The FDA’s Current Good Tissue Practice rules and the guidelines of the AATB, the safety of allograft implants is maximized.

Typical indications for use of an osteochondral allograft are traumatic or degenerative chondral and osteochondral lesions of the femoral condyles of >2 cm², particularly when the accompanying bone loss is >6 mm. Osteochondral allografts can be used to revise a prior failed cartilage repair. Use in the tibial plateau or the patellofemoral joint is best suited for younger patients without arthritic changes.

Contraindications to osteochondral allograft transplantation are inflammatory arthritis (rheumatoid or any other systemic arthritis), advanced degenerative changes, diffuse corticosteroid-induced osteonecrosis, uncorrectable knee instability, or knee malalignment.

Graft Viability
Fresh refrigerated allografts are the standard for osteochondral allograft transfers because frozen and freeze-dried cartilage has insufficient viable cartilage cells. In fresh refrigerated allograft cartilage, up to 98% of the chondrocytes are viable for seven days; this decreases to 70% by twenty-eight days. This decreased viability is accompanied by diminished cell density and decreased metabolic activity. Safety testing takes fourteen to twenty days, so the window for implantation is about ten to fourteen days from the time that the tissue is released from quarantine. A delay in implantation affects chondrocyte viability and tissue metabolism but does not affect the hyaline matrix or biomechanical properties of the allograft bone, and the effect of this delay on clinical outcome is unknown.

Surgical Technique
A size-matched and side-matched allograft is preferred. Size is determined by using magnification markers on anteroposterior and lateral radiographs to measure femoral condylar and tibial width and height.

The most common location requiring an osteochondral allograft is the weight-bearing surface of the medial or lateral femoral condyle. An arthrotomy with a medial or lateral capsular incision to displace the patella is usually sufficient for smaller defects, but larger defects may require subluxation of the patella during the exposure to ensure perpendicular access to the defect.

Press-fit cylindrical osteochondral plugs are recommended for most defects. A guidewire is placed in the center of the defect, and a reamer is used to remove any remaining tissue and subchondral bone down to healthy cancellous bone (Figs. 12 and 13). As little as 3 to 5 mm of bone can be removed for purely chondral lesions, while deeper lesions or necrotic bone from osteochondritis dissecans or osteonecrosis can require up to 8 to 10 mm of bone removal. The remnants of healthy bone from reaming this area should be saved and used to fill the cavity to minimize the defect depth. The depth in all four quadrants is measured and recorded.

The osteochondral allograft is harvested from the corresponding location on the allograft hemicondyle. The graft location and orientation are marked. On a back table, a matching-size coring reamer is used to harvest the donor allograft cylinder (Fig. 14). The cancellous allograft bone is trimmed to match the depth measurements from the defect. Pulsatile lavage is used on the osseous portion of the graft to remove any remaining marrow elements. The edges are beveled to facilitate insertion. The graft is pushed in place by hand; a tamp is not used because it will injure the superficial...
chondrocytes. The knee is placed through a range of motion to assist with seating of the allograft (Fig. 15). Press-fit fixation alone is usually adequate, but supplemental fixation with pins or screws can be used if needed. Irregularly shaped lesions may require a freehand technique, and these grafts frequently require supplemental fixation. The implants for fixation should be placed below the articular surface, and ideally screws should be placed either in the intercondylar notch or on the edge of the condyles away from the articular surface.23,26

Osteochondral allograft transplantation at the trochlea and patella presents special challenges because of the complex anatomy and topography. Peripheral lesions on the medial or lateral aspect of the trochlear groove may be amenable to cylindrical plugs, but it is difficult to match the central sulcus anatomy with plugs. For more extensive lesions of the trochlea, a shell allograft can be fashioned much like a patellofemoral arthroplasty, with a trochlear graft fashioned to match the anterior femoral resection and fixed with absorbable pins and/or peripheral screws.23,26

Press-fit cylindrical plugs should be used for isolated patellar facet lesions or lesions centered on the median ridge. Extensive patellar lesions can be treated with patellar allograft resurfacing. The diffusely damaged patellar surface is resected in its entirety with an oscillating saw, much like the technique for arthroplasty. The surgeon should leave sufficient patellar bone (at least 12 to 14 mm) after the resection. The allograft patellar surface is harvested in a similar fashion, making the osteochondral portion of the allograft essentially the entire surface of the patella. The osteochondral graft is placed on the resected host bone (Fig. 16), and fixation is obtained with the use of small double-pitched screws placed from anterior to posterior along the median ridge with threads in the subchondral bone in the allograft just below the articular surface (Fig. 17). Any patellar tracking problems should be corrected during the same procedure.

The most common indication for an osteochondral tibial allograft is a tibial plateau fracture malunion or bone loss from trauma. The technique allows restoration of up to 15 mm of lost bone. Typically, the allograft plateau and attached meniscus are transplanted together. The tibial plateau of the host...
is resected as is done for an onlay unicompartmental arthroplasty. The cuts on the allograft tibial plateau are matched, and fixation is performed with screws placed off the articular surface. Fluoroscopy assists with evaluation of the alignment. The compartment should not be overstuffed or understuffed, and an off-loading osteotomy should be done if necessary. The periphery of the transferred allograft meniscus is sewn to the meniscal synovial junction.

Rehabilitation
Rehabilitation after osteochondral allograft transplantation must prevent overloading of the graft-host interface until osseous union is achieved. Touchdown weight-bearing for six to twelve weeks is recommended, with the duration depending on the location and size of the graft. Progressive range-of-motion exercises are encouraged early, although open-chain exercises are avoided by patients with a patellofemoral allograft. Pulsed ultrasound has been shown to improve graft-healing.

Failure of osteochondral allografts usually results from unsuccessful osseous integration and subsequent subchondral collapse. It may also be the result of technical problems with achieving press-fit fixation or uncorrected coexisting joint pathology such as malalignment or instability that continues to overload the graft. Allografts of the tibial plateau and those used in compromised (osteonecrotic) bone have higher failure rates. Graft failure with collapse or fragmentation requires revision with repeat allografting or unicompartmental or total joint arthroplasty. As would be expected, patients with a unipolar lesion and a normal mechanical axis have the best outcomes, whereas transplants for patellofemoral disease, osteonecrosis, or arthrosis of both the femur and the tibia have less consistent results.

Articular Comorbidities
Malalignment
Malalignment of the knee frequently accompanies articular cartilage defects, and restoration of a neutral biomechanical environment is the single most important factor contributing to the success of any cartilage repair procedure. A medial opening-wedge high tibial osteotomy for varus malalignment and a lateral opening-wedge distal femoral osteotomy for valgus malalignment are the most common methods for correcting malalignment. Overcorrection with a proximal tibial osteotomy, while beneficial for advanced degenerative joint disease, is not well-tolerated by younger patients, who typically have an isolated articular cartilage defect. Therefore, correction to neutral alignment with the mechanical axis falling centrally between the tibial spines is recommended. Overcorrection by shifting the axis more into the contralateral compartment is indicated only if there
are arthritic changes in the joint. Generally, the necessary amount of correction is calculated on the basis of preoperative standing anteroposterior radiographs from the hip to the ankle, with lines drawn from the hip and ankle joints to the center of the tibial plateau (or to the contralateral tibial spine should mild overcorrection be desired). The angular difference between the two lines is the necessary angle of correction. This angle should be transformed into the amount of opening required during the osteotomy, since angles are difficult to measure intraoperatively, while the opening of the osteotomy can be easily assessed with a ruler.

During opening-wedge osteotomies, there is a tendency to change sagittal alignment. The surgeon should take care that the surgical correction does not inadvertently increase posterior tibial slope\(^{6,77}\). The normal posterior slope of the tibia ranges from 3° to 10°; an increase in the posterior slope promotes anterior translation of the tibia on the femur and will worsen the effect of anterior cruciate ligament deficiency or, conversely, diminish posterior cruciate ligament deficiency.

### Indications

Patients with a cartilage defect of the femoral condyle and a mechanical axis outside the neutral zone bordered by the tibial spines (Fig. 18) should have an osteotomy as a part of the cartilage repair treatment\(^ {76-80}\).

An osteotomy is contraindicated if there is articular cartilage degeneration in the contralateral or patellofemoral compartment, <90° of knee flexion, a knee flexion contracture of >20°, nonconcordant pain (knee pain outside the involved medial or lateral compartment), inflammatory arthritis, or ongoing infection\(^ {80,81}\). Patients who have a body-mass index of greater than 30 to 35 kg/m\(^2\), who continue to smoke, or who fail or are unable to comply with restricted-weight-bearing precautions during the postoperative period have higher failure rates, and these are relative contraindications to an osteotomy\(^ {81-85}\).

### Surgical Technique

**Medial Opening-Wedge High Tibial Osteotomy**

The posteromedial aspect of the tibia is exposed with subperiosteal dissection; a blunt retractor is placed behind the posterior cortex at the site of the osteotomy to protect the neurovascular structures, and another retractor is placed.
anteriorly to protect the patellar tendon. When a high tibial osteotomy is combined with open intra-articular procedures such as osteochondral allografting or autologous chondrocyte implantation, separate incisions can be utilized. The osteotomy incision is made directly in line with the posteromedial cortex of the tibia, and the longitudinal arthrotomy incision is made 5 to 7 cm anterior to it. Using two incisions decreases the length of an anterior single incision for the arthrotomy and allows its placement in the ideal location for all present and any future intra-articular procedures. Should osteotomy implants have to be removed at a later date, the posteromedial incision makes it possible to avoid opening the front of the knee.

An oblique osteotomy of the tibia is performed just proximal to the tibial tubercle, directed toward the superior tip of the fibular head (Fig. 19). An oblique osteotomy does not substantially affect patellar height, whereas a transverse osteotomy is more likely to create mild-to-moderate patella baja. The surgeon should leave at least 1.5 to 2 cm of lateral cortex below the joint line so that the lateral compartment is not violated and so that fixation on the lateral side is possible if the lateral cortex is inadvertently disrupted. Aiming the osteotome at the fibular head minimizes the risk of the osteotomy inadvertently extending up into the lateral compartment. Disruption of the lateral cortex causes a 58% reduction in axial stiffness, a 68% reduction in torsional stiffness of the tibia, and micromotion at the osteotomy site, all of which increase the chances of delayed union, nonunion, and loss of correction. However, the application of a staple or small periarticular plate to the disrupted lateral cortex restores the axial and torsional stiffness. It is much easier to deal with this pitfall of medial opening-wedge high tibial osteotomy intraoperatively than to later revise the osteotomy because of loss of correction and/or nonunion.

To avoid increasing the posterior slope, lateral fluoroscopic views are used to place guidewires to align the cutting jig or platform parallel to the anatomic posterior slope. With use of a combination of an oscillating saw and osteotomes, the osteotomy is continued to between 75% and 80% of the way across the tibia. Thin and/or flexible osteotomes assist in ensuring that the cortex is completely cut anteriorly and posteriorly. A 3.5-mm drill bit is used to make two or three small drill holes in the lateral cortex to “stress relieve” the bone during opening. The medial side is gradually opened with use of wedged-shaped tamps, wedged osteotomes, or lamina spreaders put in place anteriorly and posteriorly. Using the lamina spreaders allows one or two clicks at a time followed by a period of allowing stress relaxation within the bone. When the approximate opening is obtained within the expected range of correction, long-limb alignment is checked. The leg is axially loaded in full extension, and the mechanical axis is determined with fluoroscopy by placing an alignment rod or the electrocautery cord at the center of the femoral head and the center of the ankle. Any bump or
osteotomy is a modification of the technique described by Puddu et al.\textsuperscript{96}. This modification better controls sagittal and rotational alignment during the distal femoral osteotomy. There are multiple variables that affect rotational and sagittal alignment in the distal part of the femur. One variable is the anatomic obliquity of the distal part of the femur. An osteotomy aimed at the distal medial cortex creates a medial hinge that is less stable than a flat cortical hinge. In addition, this unstable medial hinge is exposed to deforming forces of the quadriceps, gastrocnemius, hamstrings, iliobial band, and adductors. To eliminate this unstable medial cortical hinge, a modified technique was developed that utilizes definitive distal fixation and provisional proximal fixation with a “sliding-screw” technique, all prior to completion of the osteotomy and before correction of coronal alignment is attempted.

The sliding screw technique is performed with lateral exposure in line with the distal part of the femur, splitting of the iliobial band and retraction of the vastus lateralis and distal quadriceps anteriorly, and placement of a retractor posterior to and directly on bone to protect the neurovascular structures.

The planned trajectory in the coronal plane starts just proximal to the metaphyseal flare of the lateral femoral condyle and aims at the distalmost aspect of the medial cortex. A fixation plate is temporarily approximated on the lateral aspect of the femur to get an idea of plate fit and the starting point for the osteotomy. Aiming the osteotomy toward a more proximal end point on the medial side increases the risk of medial hinge disruption, since the thicker cortical bone in this area is more susceptible to fracture. Conversely, a more distal medial hinge, located at the metaphyseal-diaphyseal junction, has thinner cortical bone that is more viscoelastic and therefore less likely to break when the osteotomy is wedged open. After the correct coronal trajectory for the oblique osteotomy is marked with a Kirschner wire (Fig. 21), the lateral view is used to place another Kirschner wire through a flat surface cutting guide so that the osteotomy will be perpendicular to the shaft of the femur.

The osteotomy is started with an oscillating saw blade traversing about three-quarters of the way across the femur while the anterior and posterior soft tissues are protected. The osteotomy is not opened, and the femur therefore remains stable. A locking T-plate (Synthes, West Chester, Pennsylvania) is then placed on the lateral aspect of the femur while ensuring appropriate placement with fluoroscopy. Distal fixation with two locked cancellous 6.5-mm screws keeps the plate in position. Provisional proximal fixation is then obtained with one unicortical 4.5-mm screw that is not fully tightened in the distal end of the sliding oblong hole of the plate. Two or three 3.2-mm-diameter drill holes in line with the osteotomy are made in the medial cortex to stress relieve the osseous hinge. Osteotomes and two lamina spreaders or wedges are then used anterior and posterior to the plate to gradually open the osteotomy without breaking the medial hinge. The sliding hole allows approximately 10 mm of opening before it has to be repositioned, should more opening be required. The

other build-up that had been placed under the patient’s hip for positioning should be removed so that a true recreation of weight-bearing is obtained. Final correction is adjusted by further opening or closing the osteotomy to create the degree of desired correction as determined by the mechanical axis through the knee. The preferred type of internal fixation is placed (Fig. 20). Plate position should be as posterior on the tibia as possible to avoid increasing the tibial slope. It has been shown that anterior and central plate position as well as larger correction angles increase posterior tibial slope\textsuperscript{77,94}. Strong plates and locking screws are recommended\textsuperscript{78,91,92}. The gap is filled with bone graft. Allograft wedges and cancellous chips, hydroxyapatite, tricalcium phosphate, and tricortical iliac crest and cancellous plug autografts are all options that have been used successfully\textsuperscript{72,35-95}. However, the fixation method may be more important than the specific type of bone graft\textsuperscript{96,98}.

Lateral Opening-Wedge Distal Femoral Osteotomy

One of the authors’ (S.D.G.) preferred technique for distal femoral osteotomy is a modification of the technique described by Puddu et al.\textsuperscript{96}. This modification better controls sagittal and rotational alignment during the distal femoral osteotomy. There are multiple variables that affect rotational and sagittal alignment in the distal part of the femur. One variable is the anatomic obliquity of the distal part of the femur. An osteotomy aimed at the distal medial cortex creates a medial hinge that is less stable than a flat cortical hinge. In addition, this unstable medial hinge is exposed to deforming forces of the quadriceps, gastrocnemius, hamstrings, iliobial band, and adductors. To eliminate this unstable medial cortical hinge, a modified technique was developed that utilizes definitive distal fixation and provisional proximal fixation with a “sliding-screw” technique, all prior to completion of the osteotomy and before correction of coronal alignment is attempted.

The sliding screw technique is performed with lateral exposure in line with the distal part of the femur, splitting of the iliobial band and retraction of the vastus lateralis and distal quadriceps anteriorly, and placement of a retractor posterior to and directly on bone to protect the neurovascular structures.

The planned trajectory in the coronal plane starts just proximal to the metaphyseal flare of the lateral femoral condyle and aims at the distalmost aspect of the medial cortex. A fixation plate is temporarily approximated on the lateral aspect of the femur to get an idea of plate fit and the starting point for the osteotomy. Aiming the osteotomy toward a more proximal end point on the medial side increases the risk of medial hinge disruption, since the thicker cortical bone in this area is more susceptible to fracture. Conversely, a more distal medial hinge, located at the metaphyseal-diaphyseal junction, has thinner cortical bone that is more viscoelastic and therefore less likely to break when the osteotomy is wedged open. After the correct coronal trajectory for the oblique osteotomy is marked with a Kirschner wire (Fig. 21), the lateral view is used to place another Kirschner wire through a flat surface cutting guide so that the osteotomy will be perpendicular to the shaft of the femur.

The osteotomy is started with an oscillating saw blade traversing about three-quarters of the way across the femur while the anterior and posterior soft tissues are protected. The osteotomy is not opened, and the femur therefore remains stable. A locking T-plate (Synthes, West Chester, Pennsylvania) is then placed on the lateral aspect of the femur while ensuring appropriate placement with fluoroscopy. Distal fixation with two locked cancellous 6.5-mm screws keeps the plate in position. Provisional proximal fixation is then obtained with one unicortical 4.5-mm screw that is not fully tightened in the distal end of the sliding oblong hole of the plate. Two or three 3.2-mm-diameter drill holes in line with the osteotomy are made in the medial cortex to stress relieve the osseous hinge. Osteotomes and two lamina spreaders or wedges are then used anterior and posterior to the plate to gradually open the osteotomy without breaking the medial hinge. The sliding hole allows approximately 10 mm of opening before it has to be repositioned, should more opening be required. The
mechanical axis is then checked. Once neutral alignment is achieved, fixation is completed with three or four locked bicortical screws proximally and two or three locked unicortical cancellous screws distally (Fig. 22). When fixation is secured, bone graft or allograft wedges are used to fill any gap in the bone.

Rehabilitation
The fixation is strong enough to allow an unrestricted range of motion and weight-bearing as tolerated. The rehabilitation program is, therefore, determined by the type of concomitant cartilage repair that has been done.

Patellar Maltracking
Patellofemoral pain and/or instability are a subset cause of anterior knee pain, as are chondral lesions. The management of patients with patellofemoral pain is complicated and the subject of many excellent books and papers. Articular cartilage is aneural and pain can arise from other sources, so it is imperative that all sources of pain are identified and a comprehensive treatment plan is established before patellofemoral cartilage repair surgery is performed. Initially, an extensive physical therapy program will be successful for most patients. A smaller number of patients with patellofemoral pain will need surgery, and both autologous chondrocyte implantation and osteochondral allograft transplantation have been reported to provide good outcomes for patients with a monopolar cartilage lesion, but autologous chondrocyte implantation provides more positive outcomes than does allografting for those with bipolar lesions. The goal of patellofemoral surgery combined with cartilage restoration is to optimize the environment for the cartilage implant to heal. Patellofemoral pathology is typically multifactorial but can be divided into three primary areas of concern: (1) tibial tuberosity location, (2) medial soft tissues (specifically, the medial patellofemoral ligament), and (3) lateral soft tissues. Often it is best to make minor adjustments to each of these sites rather than to attempt to "solve the problem" by only addressing one issue.

Tibial Tuberosity Position
The tibial tuberosity is the distal attachment of the extensor mechanism of the knee and is key to alignment and patellar tracking. The lateral position of the tibial tuberosity relative to the trochlear groove (an average tibial tubercle-to-trochlear groove distance of approximately 13 mm) places a lateral force...
vector on the patella during knee motion that is balanced by medial soft-tissue restraints. An excessively lateral position of the tibial tuberosity (a tibial tubercle-to-trochlear groove distance of 20 mm) results in high lateral force vectors that may lead to elevated stress in the patellofemoral compartment and/or contribute to the potential for lateral patellar instability. Restoration of patellofemoral cartilage without addressing other patellofemoral abnormalities generally yields poor results, but using the same cartilage restoration technique with attention to patellofemoral stresses has improved outcomes. The tibial tuberosity position may be normal; excessively lateral; or in the case of prior tuberosity surgery, excessively medial, posterior, and/or distal. In addition, even with a normal tuberosity position, the patella may be too distal because of prior trauma (patella baja) or may be congenitally too proximal (patella alta). The treatment goal is to optimize force and the contact area between the patella and femur. The tuberosity may be moved medially, laterally, proximally, and/or distally to normalize the position of the patella relative to the trochlear groove. Moving the tibial tubercle incorrectly results in a poor outcome. In addition to normalization of the tuberosity position, anterior displacement of the patella, which has been shown to decrease patellofemoral forces in both Fuji film and Tekscan studies, may be performed. When the tuberosity position is excessively lateral, anterior displacement of the tibial tuberosity may be combined with medial displacement. If the tibial tubercle-to-trochlear groove distance is normal, straight anterior displacement may be performed to decrease patellofemoral stress; it should be noted that there is concomitant rotation of patellar contact areas proximally. Each surgical procedure for patellofemoral cartilage restoration must be carefully customized to the patient’s specific chondral pathology.

Medial Soft Tissues (Specifically, the Medial Patellofemoral Ligament)

Proximally, the main medial soft-tissue dynamic restraint is the vastus medialis muscle, and the static restraints are the patellar ligament, the medial patellotibial ligament, the medial patellomeniscal ligament, and the medial joint capsule. Although in vitro studies vary in terms of the reported magnitude of restraint to lateral displacement forces by each of these tissues, all investigators have agreed that >50% is derived from the medial patellofemoral ligament and lesser contributions from the medial patellomeniscal ligament and the medial patellotibial ligament. Lateral patellar instability (patellar dislocation) results in injury to the medial soft-tissue restraints (the medial patellofemoral ligament) and is often associated with patellofemoral chondral injury. The chondral injury is often distal and medial; therefore, the medial soft-tissue restraints need to be normalized so as to not overload the medial patellofemoral compartment, in addition to preventing pathological lateral displacement. Historically, nonanatomic medial repairs have resulted in a high incidence of arthrosis, and thus the current recommendation is to focus on the medial patellofemoral ligament. Elias and Cosgarea showed that, regardless of the specific technique (see Appendix), it is important to properly reestablish the correct anatomic attachment sites, which allow the patella to be checkreined against lateral displacement forces. Under normal circumstances, there are low loads in the meniscal patellofemoral ligament and from approximately 30° of flexion to higher degrees of flexion the ligament should become progressively more lax.

Lateral Soft Tissues

Proximally, the main lateral soft-tissue dynamic restraint is the vastus lateralis muscle and the static restraints are the two layers of the lateral retinaculum: the superficial oblique layer confluent from the iliobial band and the deep transverse capsular layer, which contains the lateral patellofemoral ligament. Patellofemoral cartilage restoration is commonly performed in patients with lateral patellar instability, who have a chronic static lateral position (without current instability) of the patella relative to the trochlear groove (subluxation) and/or patellar lateral tilt, but not all of these patients benefit from a lateral release, especially in isolation. In fact, although somewhat counterintuitive, if the medial patellofemoral ligament is pathologically lax, the lateral retinaculum provides restraint to lateral displacement forces. Thus, a lateral release may increase lateral patellar instability. Furthermore, an overzealous lateral release may lead to the development of iatrogenic medial instability. While empirically it may appear that releasing the lateral retinaculum decreases patellofemoral stress, in vitro studies have failed to demonstrate this benefit. When patellar tilt is seen on computed tomography or magnetic resonance imaging, a limited lateral release is appropriate to balance soft tissues. The lateral release only extends proximally to allow reversal of tilt and never into the vastus lateralis. An alternative to lateral release is lateral lengthening as described by Biedert. By step-cutting the superficial and deep layers of the lateral retinaculum, it is possible to lengthen the retinaculum 1 to 2 cm while maintaining...
the lateral checkrein, thus avoiding medial subluxation. Additionally, this provides closure of the joint.

Meniscal Deficiency

Meniscal tears are the most commonly treated lesions of the knee, and many patients with articular cartilage damage have meniscal tears. While meniscal repair is possible in some cases, the vast majority of meniscal tears are treated with partial meniscectomy. In these knees treated with partial meniscectomy, the typical posterior horn pathology often results in segmental loss. To the untrained eye, this may seem to represent only one-third of the meniscal volume. However, biomechanical studies have shown that disruption of the circumferential hoop bundles at any location results in biomechanical loss of that entire hoop. For example, both the loss of a posterior 2-cm segment to a remnant rim of <3 mm and a radial posterior horn root tear to within 3 mm of the periphery have the same biomechanical effect as removal of an entire bucket-handle tear with a remnant of 3 mm. Sequential segmental removal of meniscal tissue in a laboratory setting resulted in a linear loss of articular cartilage contact area and a resultant increase in peak loads and stress in that compartment. On the basis of this in vitro study, most cartilage restoration algorithms dictate treatment of the meniscal remnant as if it is functional if it is 5 mm or wider and as if it is biomechanically absent if it is <3 mm anywhere along the circumferential hoop fibers. The effect of meniscal tissue loss is evident sooner in the lateral compartment, but over time the medial compartment articular cartilage begins to break down in a large percentage of patients. These detrimental effects on native cartilage are also thought to be detrimental to cartilage restoration. Meniscal transplantation decreases the stresses in a compartment with meniscal loss. The potentially positive protective effects of in situ meniscal transplantation on the results of articular cartilage repair are suggested by the long-term case study of humans by Verdonk et al. and by a study of a sheep model by Szomor et al. The negative effect of meniscal loss and the potentially positive effects of meniscal transplantation are the basis for adding meniscal transplantation to articular cartilage restoration surgery when the meniscus is biomechanically absent. Separate prospective case-series studies by Farr et al. and Rue et al. demonstrated the safety, feasibility, and efficacy of performing the two techniques concomitantly.

Meniscal allograft transplantation was performed initially to treat patients with chronic rotational instability of the knee who had both ligamentous and meniscal deficiencies. A table in the Appendix provides a summary of other studies supporting that approach. Allograft size and position are critical, and a 10% mismatch in size has major negative consequences with regard to the contact area. The correctly sized meniscal allograft needs to be ordered. Standard anteroposterior and lateral radiographs are corrected for magnification, and then anteroposterior and mediolateral meniscal dimensions are calculated. A typical meniscal anteroposterior distance is between 35 and 45 mm. A 10% "allowable mismatch" error range is, therefore, 3.5 to 4.5 mm. Currently, meniscal transplant availability allows rejection of graft-size approximations that differ from the calculated measurements by more than 2 to 3 mm. However, even a properly sized meniscus placed in a nonanatomic position will not reestablish proper contact areas within the compartment, as noted by authors of in vitro studies. The meniscal transplant horns are positioned to the patient's native attachment sites, but this does not guarantee proper placement. Allowing the surgeon to "place the anterior horn to effect proper tension" will often not place the horn attachment in the original attachment site and should be discouraged. The decision to use a bone bridge with a slot technique or bone plugs with a socket technique is determined by surgeon preference, but in vitro biomechanical testing suggests that soft-tissue anchoring alone is inadequate.

Overview

Cartilage defects in the knee are common, but not all are symptomatic. If pain persists despite an appropriate nonoperative treatment regimen, cartilage repair is indicated. Multiple procedures are available, which should be seen as complementary, rather than competitive, allowing treatment of the entire spectrum of lesions from small focal lesions on the femoral condyles to large bipolar or multiple lesions in the patellofemoral joint. Normalization of the biomechanical joint environment through osteotomy or meniscal transplantation is crucial to the success of any cartilage repair technique.

Appendix

Tables showing techniques for addressing deficiency of the medial patellofemoral ligament and outcomes of meniscal allograft transplants are available with the electronic version of this article on our web site at jbis.org (go to the article citation and click on "Supporting Data").
as well as other lectures presented at the Academy's Annual Meeting, will be available in
February 2011 in Instructional Course Lectures, Volume 60. The complete volume can be ordered online at www.aaos.org, or by calling 800-626-6726 (8 A.M.-5 P.M., Central time).

References


