Selected Instructional Course Lectures

The American Academy of Orthopaedic Surgeons

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Articular cartilage is vulnerable to traumatic injury and subsequent degeneration. These changes are likely related to the limited capacity for cartilage repair, poor vascular supply, and deficiency in terms of the ability of an undifferentiated cell population to respond to the insult. While the natural history of isolated chondral and osteochondral defects is not predictable, clinical experience suggests that, when left untreated, these lesions do not heal and may progress to symptomatic degeneration of the joint. Therefore, early surgical intervention for symptomatic lesions is often suggested in an effort to restore normal joint congruity and pressure distribution and prevent further injury. Treatment recommendations are made after an evaluation of symptomatic lesions and should be tailored to the specifics of each case.

The goals of surgical treatment are to provide pain relief and improve joint function, thus allowing patients to comfortably perform activities of daily living and potentially maintain or return to higher levels of activity. Multiple algorithms have been described in an effort to simplify the treatment of cartilage lesions. These are useful tools with which to organize thoughts. In general, surgical options can be grouped into three categories: palliative (arthroscopic débridement and lavage), reparative (marrow stimulation techniques), and restorative (osteochondral grafting and autologous chondrocyte implantation). All of these techniques have been reported to improve the clinical status as compared with the preoperative state. Thus, the appropriate treatment for any given cartilage lesion is patient-specific. The size and location of the lesion, the physical demands of the patient, and the treatment history all are important preoperative considerations. In addition, the surgeon must consider what subsequent treatment options are available if the current treatment fails to relieve the symptoms. A realistic and comprehensive understanding of the patient’s goals is critical to any decision regarding how to treat a symptomatic chondral defect. In keeping with these principles, the treatment algorithm consists of a graduated surgical plan. The least destructive and least invasive treatment option necessary to alleviate the symptoms and restore joint function is performed first. The more extensive treatments are reserved for potential salvage operations later. If the symptoms persist despite conservative treatment, subsequent treatments are not impeded by previous management.

**Decision-Making**

When treating articular cartilage lesions in the knee, the surgeon should focus on patient-specific and defect-specific variables and avoid “linear thinking.” The clinical presentation should correlate with the underlying pathoanatomy. For example, a patient with known classic osteochondritis dissecans of the medial femoral condyle who reports bilateral anterior knee pain with stair-climbing should be evaluated initially with a presumptive diagnosis of patellofemoral pain before ascribing the symptoms to the osteochondritis dissecans lesion. Because the natural history of cartilage lesions is not known and the surgical treatments are neither benign nor associated with a predictable outcome (particularly with regard to the prevention of arthritis), surgical decision-making must be taken quite seriously.

Understanding and addressing the patient’s specific concerns and goals are critical to achieving a successful outcome from the patient’s perspective. More spe-
specifically, patients often express concerns about whether it is safe to remain active despite symptoms and whether a delay in surgical intervention precludes certain treatment options because of disease progression. In addition, knowledge of the specific marginal improvements that a procedure should provide gives the patient a reasonable expectation regarding the outcome. Unfortunately, the lack of understanding of the natural history of these defects makes it difficult to advise patients, and it is best to carry out careful discussions on a case-by-case basis.

Patient age, body mass index, symptom type (weight-bearing pain, non-weight-bearing pain, swelling, mechanical symptoms, giving-way, and aggravation of symptoms related to walking on level ground as opposed to stair-climbing), occupation and/or family commitments, risk-aversion (desire to avoid subsequent surgical procedures), responsiveness and rehabilitation after previous surgical treatments, and the patient’s specific concerns related to his or her problem are all important preoperative considerations. While chronologic age is often cited as a relative indication or contraindication to cartilage repair, it is really physiologic age that determines the patient’s eligibility for a non-arthroplasty solution. Typically, patients who become symptomatic in the fourth or fifth decade of life have concomitant chondral and subchondral disease involving apposing articular surfaces that precludes a biologic treatment option. In addition, the results of partial and total knee arthroplasty are predictably gratifying and satisfy most patients, even those who are relatively young. Finally, one must carefully search for associated pathological conditions, such as malalignment, ligament insufficiency, and concomitant meniscal deficiency, that may contribute to treatment failure and should be corrected before or during the surgery to treat the chondral lesion.

Defect-specific variables include defect location, number, size, depth, and geometry; the condition of the subchondral bone and surrounding cartilage; and the degree of containment. The condition of the apposing surface, which is often overlooked, is also an important variable. Even minor areas of early degeneration make achieving a satisfactory clinical outcome challenging. Specific management of each of these defect-specific variables increases the likelihood of a good clinical outcome.

**Fig. 1**

Treatment algorithm for focal chondral lesions. Before treatment, it is important to assess the presence of correctable lesions. Surgical treatment should be considered for trochlear and patellar lesions only after use of rehabilitation programs has failed. The treatment decision is guided by the size and location of the defect, the patient’s demands, and whether it is first or second-line treatment. ACL = anterior cruciate ligament, PCL = posterior cruciate ligament, MFX = microfracture, OATS = osteochondral autograft transplantation, ACI = autologous chondrocyte implantation, OCA = osteochondral allograft, AMZ = anteromedialization, ++ = best treatment option, and + = possible option depending on patient’s characteristics.

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**ACL** = anterior cruciate ligament, **PCL** = posterior cruciate ligament, **MFX** = microfracture, **OATS** = osteochondral autograft transplantation, **ACI** = autologous chondrocyte implantation, **OCA** = osteochondral allograft, **AMZ** = anteromedialization, ++ = best treatment option, and + = possible option depending on patient’s characteristics.
Malalignment, ligament insufficiency, and concomitant meniscal deficiency are assessed and, when necessary, are treated with a concomitant or staged osteotomy (high tibial, distal femoral, or tibial tuberosity), ligament reconstruction, and perhaps a meniscal allograft transplantation. Patellofemoral lesions are often treated with a simultaneous realignment procedure such as anteromedialization of the tibial tuberosity. Anteromedialization is more successful for lateral patellofemoral lesions than it is for lesions located along the medial aspect of the patellofemoral joint. Medial patellofemoral lesions are treated with a more vertically oriented anteromedialization. The treatment algorithm for chondral lesions is guided by the lesion size and location and the patient activity level (Fig. 1).

Primary repair is done for any chondral injury that is amenable to fixation. Any acute osteochondral fragment or in situ and unstable osteochondritis dissecans lesion is repaired primarily. It is particularly critical to fix large fragments (>1 cm²) from the weight-bearing portion of the femoral condyles. The basic principles for primary repair include elevation of the unstable fragment, debridement of the fibrous base, microfracture if necessary to gain access to the subchondral blood supply to promote healing, bone-grafting of areas of cystic changes or bone loss, and rigid fixation of the fragment under compression. Headless-compression screw fixation, with subsequent screw removal in younger patients after eight weeks of non-weight-bearing, is often used. Continuous passive motion for up to six hours each day is recommended. Because fragments can settle over time, even headless screws can become prominent and damage the apposing surface. In addition, performing a second-look arthroscopy to evaluate the defect helps the surgeon to judge the success of the procedure and to provide accurate advice to the patient.

Patients with lesions that cannot be repaired primarily may benefit from another type of treatment (palliative, reparative, or regenerative). Marrow stimulation techniques are typically a first-line treatment. These techniques are often used for smaller lesions (<2 cm²), or in patients with larger lesions (>3 cm²) and modest physical or physiologic demand levels. Small lesions in high-demand patients or those for whom marrow stimulation has failed can be treated with one or two 10-mm osteochondral autografts harvested from the lateral femoral trochlea just proximal to the sulcus terminalis. Larger lesions (>2.5 cm²) are typically more amenable to osteochondral allografting or autologous chondrocyte implantation. Autologous chondrocyte implantation is advised for younger patients with shallow lesions, especially of the patellofemoral joint. This method does not violate the subchondral bone and minimizes the impact on future treatment such as osteochondral allograft transplantation. Larger, deeper lesions with bone loss typically require an osteochondral allograft.

Treatment is also guided by the location of the lesion. For example, osteochondral allografts are used for femoral condyle lesions because they allow accurate anatomic reconstruction. Lesions of the patellofemoral joint are often treated with autologous chondrocyte implantation because the lesions are small and the varying anatomic concavity and convexity make structural grafts too difficult to fit in place. The tibia remains a difficult articular surface to treat. Small tibial lesions that are found when the femoral articular cartilage is being restored are commonly treated with marrow stimulation techniques. Other options include the utilization of osteochondral autografts placed in a retrograde manner with use of a cannulated reamer system (Arthrex, Naples, Florida). The use of osteochondral allografts with an intact meniscus and...
concomitant realignment has been reported for the treatment of larger lesions of the tibial plateau, especially after fracture and the development of secondary arthritis, with graft survival rates of up to 65% at fifteen years.

**Surgical Options**

**Marrow Stimulation Technique (Microfracture)**

The microfracture marrow stimulation technique is carried out with a surgical awl to penetrate the subchondral bone. The violation of the subchondral plate promotes bleeding and the local migration of stem cells and other anabolic factors that support the formation of a “superclot.” It is believed that the pluripotent nature of these stem cells allows the formation of reparative fibrocartilage tissue (Fig. 2).

Critical to the success of this technique is the creation of vertical walls of stable articular cartilage to create a “well-shouldered” lesion. This improves the local mechanical environment during healing by reducing shear and compression. All unstable cartilage is removed when the lesion site is prepared. The calcified cartilage layer is carefully débrided, and surgical awls are used to penetrate the subchondral bone (Fig. 3). The holes are placed perpendicular to the bone surface, 2 to 3 mm apart, and confluence is avoided. Postoperative rehabilitation is guided by the location of the lesion, but typically it involves up to six weeks of non-weight-bearing and the use of a continuous-passive-motion machine for six hours per day. Patients with a lesion in the patellofemoral joint wear a brace with a flexion stop of 30° to limit patellofemoral contact; weight-bearing is permitted.

The best outcomes of this technique are seen in younger patients with small traumatic lesions. After two and five years of follow-up, Knutsen et al. found no difference between the outcomes of microfracture and those of autologous chondrocyte implantation for femoral condyle lesions, but patients with smaller lesions treated with microfracture did better than those with...
larger lesions. Similarly, Gudas et al. observed that, among patients with lesions exceeding 2 cm$^2$ in the central part of the medial femoral condyle, those treated with microfracture had lower clinical outcome scores than did those treated with an osteochondral autograft transplantation (Table I). Location also plays a role in the success of marrow stimulation techniques, with better results seen after the treatment of femoral condyle lesions.

**Osteochondral Autograft Transplantation**

Osteochondral autograft transplantation is the transfer of one or more cylindrical osteochondral autografts into the cartilage defect, providing a congruent hyaline-cartilage-covered surface (Fig. 4). The autografts are harvested from the non-weight-bearing periphery of the femoral trochlea or the margin of the intercondylar notch. With a combination of different graft sizes, 90% to 100% of the defect can be filled. This technique is limited by the amount of donor tissue available in the knee, and donor site morbidity increases as more tissue is harvested. Osteochondral autograft transplantation is best for small lesions (<2 cm$^2$), but good clinical results have been reported with lesions between 2 and 4 cm$^2$. The use of “mega” osteochondral autograft transplants (“mega-OATS”) from the posterior part of the femoral condyle for large osteochondral lesions (>4 cm$^2$) has had good clinical results at 5.5 years postoperatively.

Osteochondral autograft transplantation can be done through a small arthrotomy or entirely arthroscopically. To harvest donor grafts perpendicular to the surface, we prefer to obtain the donor plugs through a small lateral arthrotomy for the lateral edge of the patella can interfere with an arthroscopic harvest. The plugs are then implanted arthroscopically. There are many available

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**TABLE I** Demographic Data and Outcomes in Studies Comparing Microfracture with Other Cartilage Restoration Procedures

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Group 1</th>
<th>Group 2</th>
<th>No. of Patients</th>
<th>Mean Age (yr)</th>
<th>Mean Lesion Size (cm$^2$)</th>
<th>Lesion Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saris et al.</td>
<td>Autolog. chondrocyte implant.</td>
<td>Microfract.</td>
<td>118</td>
<td>33.9</td>
<td>Range, 2.4-2.6</td>
<td>Med. and lat. fem. condyles</td>
</tr>
<tr>
<td>Knutsen et al.</td>
<td>Autolog. chondrocyte implant.</td>
<td>Microfract.</td>
<td>80</td>
<td>Not reported</td>
<td>Not reported</td>
<td>89% med. fem. condyle; 11% lat. fem. condyle</td>
</tr>
<tr>
<td>Gudas et al.</td>
<td>Osteochondral autograft transplant.</td>
<td>Microfract.</td>
<td>60</td>
<td>24.3</td>
<td>2.8</td>
<td>84% med. fem. condyle; 16% lat. fem. condyle</td>
</tr>
<tr>
<td>Knutsen et al.</td>
<td>Autolog. chondrocyte implant.</td>
<td>Microfract.</td>
<td>80</td>
<td>32.2</td>
<td>4.8</td>
<td>89% med. fem. condyle; 11% lat. fem. condyle</td>
</tr>
</tbody>
</table>

*HSS = Hospital for Special Surgery, and SF-36 = Short Form-36.*

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Fig. 5

Osteochondral autograft transplantation. A: Identification of the lesion on the medial femoral condyle. B: A sizer is used to determine the number and size of the autografts. In this case, the lesion measured 8 mm in diameter. C: An 8-mm plug was harvested. D: The donor-plug position should be flush with the surrounding articular cartilage.
commercial systems that provide a series of donor and recipient harvesting tubes to create a press-fit implant of up to 10 mm in diameter. A sizing guide is used to determine the number and size of grafts that are needed. A properly sized graft harvester with a collared pin is introduced perpendicular to the donor site (Fig. 5) to a depth of approximately 12 to 15 mm. The recipient socket is created to a depth that is 2 mm less than the length of the donor graft. It is important to maintain a perpendicular relationship between the donor and the articular surface to create well-defined vertical walls in the recipient socket, as this facilitates congruent plug placement (Fig. 5, C). The donor plug is placed over the recipient site and gently advanced into the defect, where it is often left slightly proud. The chondrocytes can be damaged during impact; therefore, it is critical to avoid high loads when inserting the graft.$^11$ The final plug position should be flush with the surrounding articular cartilage (Fig. 5, D). Postoperatively, patients are protected from weight-bearing for six weeks and use a continuous-passive-motion machine six hours per day.

Hangody and Kárpati$^14$ evaluated the survival of the transplanted hyaline cartilage. The graft undergoes osseous incorporation to the subchondral bone while the transplanted cartilage integrates with the adjacent host articular cartilage with fibrocartilage. Recently, Hangody et al.$^{15}$ evaluated clinical outcomes at a mean of fourteen years after 1097 osteochondral autograft transplantation procedures. Encouraging results in this large multicenter series support the use of this technique for the treatment of small and medium focal chondral and osteochondral defects of the knee. The osteochondral autograft transplantation procedure has been compared with other cartilage restoration procedures (Table II).

### Osteochondral Allograft Transplantation

Osteochondral allograft transplantation provides an option for treatment of larger lesions ($>2.5$ cm$^2$) or those with substantial bone loss. It is normally a second-line treatment option, but can be a first-line treatment for high-demand patients with large lesions.

Osteochondral allograft transplantation can be used to resurface large, deep defects with mature hyaline articular cartilage while also filling any underlying osseous defect. Tissue matching and immunosuppression are not necessary because the transplanted chondrocytes are isolated by the cartilage matrix and not exposed to the host immune surveillance.$^{15}$ The allografts can be “fresh” or frozen. Fresh grafts are normally maintained at 4°C in standard or enriched culture medium for no more than twenty-eight days, which allows chondrocytes to survive after transplantation. Frozen allografts are maintained at −40°C for years. The fresh allografts elicit a minimal immune response, the chondrocytes survive, and the bone is successfully revascularized.$^{16,18}$

Allograft transplantation can be done arthroscopically; however, it is more often performed through a small arthrotomy. The allograft is slowly warmed from 4°C to 37°C by placing it in normal saline solution at room temperature. The slow warming minimizes damage to the graft.$^{19}$ The lesion is sized with a template, and a correspondingly sized reamer is used to convert the defect to a circular recipient socket with a uniform depth of 6 to 8 mm (Fig. 6). This bone depth facilitates graft implantation and limits the amount of immunogenic donor bone that is implanted. A sterile marking pen is used to mark the 12 o’clock position of the lesion to orient the donor plug appropriately. An instrumentation
A system is used to size and harvest a cylindrical plug from the allograft (Fig. 7). The donor graft is drilled through its entire depth with a harvester under irrigation with normal saline solution. The graft is extracted, and a ruler is used to measure and mark the four quadrants of the graft at the depth of the previously measured recipient sites. Before insertion, pulsatile lavage (approximately 2 L) is used to remove the residual blood and bone-marrow elements from the allograft to reduce the risk of disease transmission and graft immunogenicity. The graft is then press-fit into the socket by

**TABLE II Demographic Data and Outcomes in Studies of Osteochondral Autograft Transplantation**

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Group 1</th>
<th>Group 2 (or 2 and 3)</th>
<th>No. of Patients</th>
<th>Mean Age (yr)</th>
<th>Mean Lesion Size (cm²)</th>
<th>Lesion Location</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hangody et al.</td>
<td>Osteochondral autograft transplant.</td>
<td>—</td>
<td>1097</td>
<td>36</td>
<td>Not reported</td>
<td>798 fem. condyle, 147 patellofemoral, 31 tibia, 98 talus, 8 capitellum, 3 hum. head, 11 fem. head</td>
</tr>
<tr>
<td>Marcacci et al.</td>
<td>Osteochondral autograft transplant.</td>
<td>—</td>
<td>30</td>
<td>29.3</td>
<td>&lt;2.5</td>
<td>Med. and lat. fem. condyles</td>
</tr>
<tr>
<td>Gobbi et al.</td>
<td>Osteochondral autograft transplant.</td>
<td>Microfract.; chondroplasty</td>
<td>32</td>
<td>Osteochondral autograft transplant.: 27; microfract.: 24; chondroplasty: 32</td>
<td>Osteochondral autograft transplant.: 4; microfract.: 4.5; chondroplasty: 3.7</td>
<td>Talus</td>
</tr>
<tr>
<td>Dozin et al.</td>
<td>Débrid. then autolog. chondrocyte implant.</td>
<td>Débrid. then osteochondral autograft transplant.</td>
<td>47</td>
<td>Autolog. chondrocyte implant.: 29; osteochondral autograft transplant.: 27</td>
<td>Autolog. chondrocyte implant.: 1.97; osteochondral autograft transplant.: 1.88</td>
<td>Autolog. chondrocyte implant.: 73% fem. condyle, 27% patella; osteochondral autograft transplant.: 68% fem. condyle; 32% patella</td>
</tr>
<tr>
<td>Bentley et al.</td>
<td>Osteochondral autograft transplant.</td>
<td>Autolog. chondrocyte implant.</td>
<td>100</td>
<td>31.3</td>
<td>4.66</td>
<td>53% med. fem. condyle; 18% lat. fem. condyle; 25% patella; 3% trochlea; 1% tibial plateau</td>
</tr>
</tbody>
</table>

*HSS = Hospital for Special Surgery.

![Fig. 6](http://jbjs.org/)

**Fig. 6**
Osteochondral allograft transplantation. A: The procedure is typically performed through a small arthrotomy to expose the lesion. B: A reamer is used to convert the defect to a circular recipient socket with a uniform depth of 6 to 8 mm.
hand after careful alignment of the four quadrants to the recipient site (Fig. 8). If the implanted allograft is particularly large, fixation may be augmented with bioabsorbable or metal compression screws.

Postoperatively, weight-bearing is limited to toe-touch for the first six weeks. Patients with a patellofemoral graft are allowed to bear weight as tolerated in extension and generally are limited to 45° of flexion during the first four weeks. Continuous passive motion is used immediately after the surgery. A return to normal activities of daily living and light sports activity is considered at eight to twelve months.

Subjective improvement can be expected in 75% to 85% of patients after osteochondral allograft implantation for properly selected chondral lesions\(^4,20\) (Table III).

**Autologous Chondrocyte Implantation**

Autologous chondrocyte implantation is ideal for symptomatic, unipolar, well-contained chondral or osteochondral defects measuring between 2 and 10 cm\(^2\) with bone loss of less than 6 to 8 mm. It is typically a second-line treatment after at least arthroscopic debridement has been performed.

The first stage of autologous chondrocyte implantation is an arthroscopic evaluation of the size and depth of the focal chondral lesion and a cartilage biopsy. The total volume of the biopsied material should be approximately 200 to 300 mg. The second stage is implantation of the cells. This is done usually no sooner than six weeks after the biopsy. At the time of implantation, the defect is prepared by removing any existing fibrocartilage down to the underlying calcified layer. Vertical walls are created at the periphery of the lesion with use of a combination of a number-15 blade and sharp ring curets. After debridement, the tourniquet, if used, should be deflated, and complete hemostasis should be obtained. The use of cotton pledges soaked with epinephrine may help to obtain hemostasis (Fig. 9).

Next, a periosteal patch is harvested from the proximal-medial part of the tibia, just distal to the pes anserinus insertion, through a separate incision. The patch should be at least 2 mm larger than the defect. The patch edges are detached with a number-15 scalpel blade and elevated with a sharp, curved periosteal elevator, beginning distally. Synthetic collagen-membrane substitutes are commercially available (Chondro-Gide; Geistlich Biomaterials, Wolhusen, Switzerland) and can be used as a substitute for the periosteal patch. The

<table>
<thead>
<tr>
<th>TABLE II (continued)</th>
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<tbody>
<tr>
<td>Mean Duration of Follow-up</td>
</tr>
<tr>
<td>14 yr</td>
</tr>
<tr>
<td>Range, 2-7 yr</td>
</tr>
<tr>
<td>54 mo</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>19 mo</td>
</tr>
</tbody>
</table>
use of these scaffolds not only reduces operating time but also has been shown to avoid typical problems related to the periosteum.

The patch or scaffold is then sewn to the cartilage. When periosteum is used, the cambium layer is placed toward the lesion. With the Chondro-Gide scaffold, the porous surface should be placed toward the lesion with the smooth side facing out. Sutures (6-0 Vicryl [polyglactin]) are

Fig. 7
Osteochondral allograft transplantation. A: Fresh donor femoral condyle. B: The condyle is trimmed to create a flat surface to place on the workstation. This cut is made parallel to the potential harvest site. C: Condyle securely fixed to the workstation. D: Graft template placed on the condyle to match the bottom of the recipient site.

Fig. 8
Osteochondral allograft transplantation. A: After removal of the plug, depth-measurement markings are made on the graft to match the measurements of the recipient socket in four quadrants. B: Matching of the donor plug. The depth of bone should be limited to 8 to 10 mm to facilitate graft implantation and limit the amount of immunogenic donor bone that is implanted. C: The graft is press-fit into the socket by hand after careful alignment of the four quadrants to the recipient site. The graft is flush with the recipient articular surface.
<table>
<thead>
<tr>
<th>Author(s)</th>
<th>Type of Osteochondral Allograft</th>
<th>Mean Age (yr)</th>
<th>Type of Study</th>
<th>No. of Patients</th>
<th>Lesion Location</th>
<th>Mean Duration of Follow-up</th>
<th>Outcome*</th>
<th>Additional Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gross et al.</td>
<td>Failed fresh</td>
<td>47</td>
<td>Histological</td>
<td>69</td>
<td>Knee (exact location not specified)</td>
<td>&lt;1, 2-5, &gt;5 yr</td>
<td>Cartilage: viable chondrocytes, normal matrix and structure in middle and deep layers. Bone: creeping substitution</td>
<td></td>
</tr>
<tr>
<td>Davidson et al.</td>
<td>Fresh</td>
<td>32</td>
<td>Clinical, histological, MRI</td>
<td>8 (10 knees)</td>
<td>6 med. fem. condyle, 2 trochlea, 2 med. fem. condyle and trochlea</td>
<td>40 mo</td>
<td>Clinical: improvements in SF-36, IKDC, Tegner, Lysholm scores (p &lt; 0.05). Histological: cellular density and viability similar in host and donor cartilage. MRI: improvement in Outerbridge score</td>
<td>Prevention of short-term degenerative changes</td>
</tr>
<tr>
<td>McCulloch et al.</td>
<td>Fresh</td>
<td>35</td>
<td>Clinical, radiographic</td>
<td>25</td>
<td>Fem. condyle</td>
<td>3 yr</td>
<td>Clinical: improvements in Lysholm, IKDC, KOOS, SF-12 scores (p &lt; 0.05); 84% of patients satisfied. Radiographic: 88% had graft incorporation</td>
<td>Patients with uncorrected malalignment did worse; clinical results did not deteriorate with increasing age of graft</td>
</tr>
<tr>
<td>Jamali et al.</td>
<td>Fresh patellofem.</td>
<td>28</td>
<td>Clinical, radiographic</td>
<td>18</td>
<td>Patellofem.</td>
<td>94 mo</td>
<td>Clinical: 5 failures; good results in 60%. Radiographic: no signs of patellofemoral arthrosis in 10 of 12</td>
<td>No patellofemoral bone alignment proc. performed</td>
</tr>
<tr>
<td>Gross et al.</td>
<td>Fresh</td>
<td>27</td>
<td>Clinical outcome</td>
<td>60</td>
<td>30 med. fem. condyle; 30 lat. fem. condyle</td>
<td>10 yr</td>
<td>Survival: 95% at 5 yr, 85% at 10 yr, 65% at 15 yr</td>
<td>Comorbidities should be assessed and corrected in same procedure</td>
</tr>
</tbody>
</table>

*IKDC = International Knee Documentation Committee, KOOS = Knee Injury and Osteoarthritis Outcome Score, and SF-12 = Short Form-12.
first passed into the patch approximately 2 mm from the edge and then passed through the cartilage at a depth of 2 to 3 mm below the cartilage surface. Sutures should be placed approximately 4 mm apart, and a gap should be maintained in the upper edge to allow chondrocyte implantation (Fig. 10). The edges of the patch are sealed with fibrin glue, and a watertight test is performed with an 18-gauge angiocatheter. The chondrocytes are then delivered through the opening with use of an angiocatheter. After the cells have been implanted, the opening gap is closed with suture and fibrin glue (Fig. 9, C).

Postoperatively, patients with a femoral condyle lesion are kept non-weight-bearing and use a continuous-passive-motion machine. Patients with a patellofemoral lesion are permitted full weight-bearing with the knee in extension. Continuous passive motion for six to eight hours per day at one cycle per minute is used for six weeks after the surgery. A return to normal activities of daily living and sports activities is allowed six months after the surgery.

### TABLE IV Demographic Data and Clinical Outcomes in Studies of Autologous Chondrocyte Implantation

<table>
<thead>
<tr>
<th>Author(s)</th>
<th>No. of Patients</th>
<th>Mean Age (yr)</th>
<th>Mean Lesion Size (cm²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Zaslav et al. 34</td>
<td>126 with autologous chondrocyte implant. after other failed cartilage proc. (multicenter study)</td>
<td>34.5</td>
<td>4.63</td>
</tr>
<tr>
<td>Rosenberger et al. 35</td>
<td>56; 50% with concomitant osteotomies</td>
<td>48.6 (range, 45-60); all &gt;45</td>
<td>4.7 (range, 1-15.0)</td>
</tr>
<tr>
<td>Mandelbaum et al. 36</td>
<td>40</td>
<td>Range, 16-48</td>
<td>4.5</td>
</tr>
<tr>
<td>Kreuz et al. 37</td>
<td>118 with isolated chondral lesion</td>
<td>35 (range, 18-50)</td>
<td></td>
</tr>
<tr>
<td>Knutsen et al. 7</td>
<td>40 with autologous chondrocyte implant., 40 with microfract.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steinwachs and Kreuz 38</td>
<td>63</td>
<td>34</td>
<td></td>
</tr>
</tbody>
</table>

*VAS = visual analog scale, SF-36 = Short Form-36, and ICRS = International Cartilage Repair Society.

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**Fig. 9**

Autologous chondrocyte implantation. A: A chondral lesion in the patella. B: Preparation of the defect. C: After the chondrocytes are delivered, the gap is closed with suture and fibrin glue.
It is estimated that autologous chondrocyte implantation has been performed in >10,000 patients worldwide. The procedure has better results when it is done for lesions in the femoral condyle or in patients with a patellofemoral lesion who are undergoing a concomitant realignment procedure. There have been several studies comparing autologous chondrocyte implantation with other biologic reconstructive procedures (Table IV).

### Overview

Articular cartilage defects of the knee are common. Treatment options range from palliative (débridement) to reparative (marrow stimulation) to restorative (osteochondral grafting and autologous chondrocyte implantation). All of these techniques improve the clinical status compared with the preoperative state. Decision-making is done case by case and is guided by the patient’s physical and physiologic demand level, previous failed treatment, and the location and size of the defect.

It is critical that the surgeon also consider what subsequent treatment options might be necessary should the first-line treatment fail to relieve the symptoms.

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### TABLE IV (continued)

<table>
<thead>
<tr>
<th>Lesion Location</th>
<th>Mean Duration of Follow-up</th>
<th>Outcome*</th>
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</thead>
<tbody>
<tr>
<td>102 (67%) med. fem. condyle; 27 (18%) lat. fem. condyle; 24 (16%) trochlea</td>
<td>48 mo</td>
<td>76% were treatment successes; no difference between results of marrow stimulating proc. and débrid. at prim. op.; mean improvements in Cincinnati, VAS, and SF-36 scores from baseline to all time points (p &lt; 0.001)</td>
</tr>
<tr>
<td></td>
<td>4.7 yr (range, 2-11)</td>
<td>8 failures (14%); additional arthroscopic proc. required in 24 patients (43%) for periosteal-related problems and adhesions; 88% of these patients had lasting improvement, 78% felt improved, and 81% would again choose autologous chondrocyte implant. as a treatment option</td>
</tr>
<tr>
<td>Trochlea</td>
<td>59 ± 18 mo</td>
<td>Significant improvement in Cincinnati score, overall condition (3.1 points preop. to 6.4 points postop.), pain (2.6 to 6.2 points), swelling (3.9 to 6.3 points); no failed implant</td>
</tr>
<tr>
<td>78 fem. condyle; 17 trochlea; 23 patella</td>
<td>Clinical and MRI eval. at 6, 18, and 36 mo</td>
<td>Patients with regular (1-3 times/wk) or competitive (4-7 times/wk) sports involvement had significantly better ICRS and Cincinnati scores than patients with no or rare sports involvement (p &lt; 0.01); correlation between sports activity levels and clinical scores significant (increasing from 6 to 18 mo, 18 to 36 mo postop.)</td>
</tr>
<tr>
<td>89% med. fem. condyle; 11% lat. fem. condyle</td>
<td>2 yr</td>
<td>77% good clinical results in both groups; no significant difference between groups; younger patients did better in each group</td>
</tr>
<tr>
<td>Fem. condyle, trochlea, patella</td>
<td>6, 18, and 36 mo</td>
<td>Evaluation of autologous chondrocyte implant. with type I/III collagen membrane; significant improvement in ICRS and modified Cincinnati scores (p &lt; 0.01); graft hypertrophy can be avoided by using a collagen membrane</td>
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</tbody>
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**Fig. 10**

Autologous chondrocyte implantation. A: Injection of the chondrocytes under the upper edge of the patch. The cells should be injected slowly. B: A periosteal patch with the cambium layer facing down into the defect is carefully sutured onto the top of the defect. Chondrocytes are injected into the contained defect.
References


