# The Management of Chondral Defects in the Hip

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## Introduction

The results following treatment of focal cartilage lesions in the hip were generally poor prior to the description of the surgical hip dislocation by Ganz and colleagues [1]. These were confounded by the risk of avascular necrosis associated with the surgical approach and surgeries that were frequently performed for avascular necrosis [2, 3].

To date, most of the literature, experience, and techniques for treating focal cartilage defects have been for lesions in the knee [4, 5]. Thus, the recommendations in this chapter are based on the current understanding of hip biomechanics in combination with what is known about cartilage biology from the experience in the knee. These recommendations have the potential to change as the understanding of hip biomechanics and focal cartilage lesions in the hip improves.

The joint preservation techniques described here are for the treatment of focal, full-thickness cartilage lesions, not generalized osteoarthritis. The treatment goals for these patients are: resolution of pain, restoration of function, and return to activity. Although it has yet to be definitively proven, early treatment of a focal cartilage lesion may also help to prevent the progression of cartilage degeneration and osteoarthrosis. These techniques are contraindicated if the patient is unable or unwilling to comply with postoperative rehabilitation and weight-bearing protocols, if arthritis is due to a systemic inflammatory disorder, or if the arthritis is significantly advanced, involving the majority of both the femur and acetabulum. In these cases the patient may be a better candidate for joint replacement surgery.

## Basic Science

One of the principles of cartilage biomechanics is that mechanical stress on the cartilage causes chondrocyte death and damage to the extracellular matrix [6]. In a focal cartilage defect, there is increased strain and shear stress at the rim of the lesion, with a change in the fluid mechanics around the rim [7]. Thus, the lesion has the potential to degenerate further due to the increased stress on the chondrocytes on the rim. In addition, there appears to be a threshold effect to lesion size. Below a certain size, the surrounding tissue can absorb the increased load caused by the cartilage defect (Fig. 18.1). Above this size, the rim stress around the lesion increases and is detrimental to the surrounding cartilage. In a cadaveric knee model, the threshold size of the lesion was 10 mm and the meniscus was able to absorb the load created by smaller defects [8]. In similar finite element studies, the threshold size of a cartilage lesion became smaller if the meniscus was removed, because the meniscus could not absorb the increased load (Fig. 18.1) [9].

## Cartilage Science in the Hip

The cartilage in the hip is thinner than in the knee [10], with an average cartilage thickness that varies from 1.08 to 2.4 mm on the femoral head [10, 11] and from 1.24 to 2.25 mm on the acetabulum [10, 11]. There does appear to be some correlation of cartilage thickness to the size and weight of the person, with taller and heavier people having thicker cartilage [10].

Studies comparing the cartilage thickness between joints of the same cadaveric specimen have lead to the hypothesis that congruent joints like the hip and ankle have thinner cartilage than incongruent joints—namely, the knee [10, 12].

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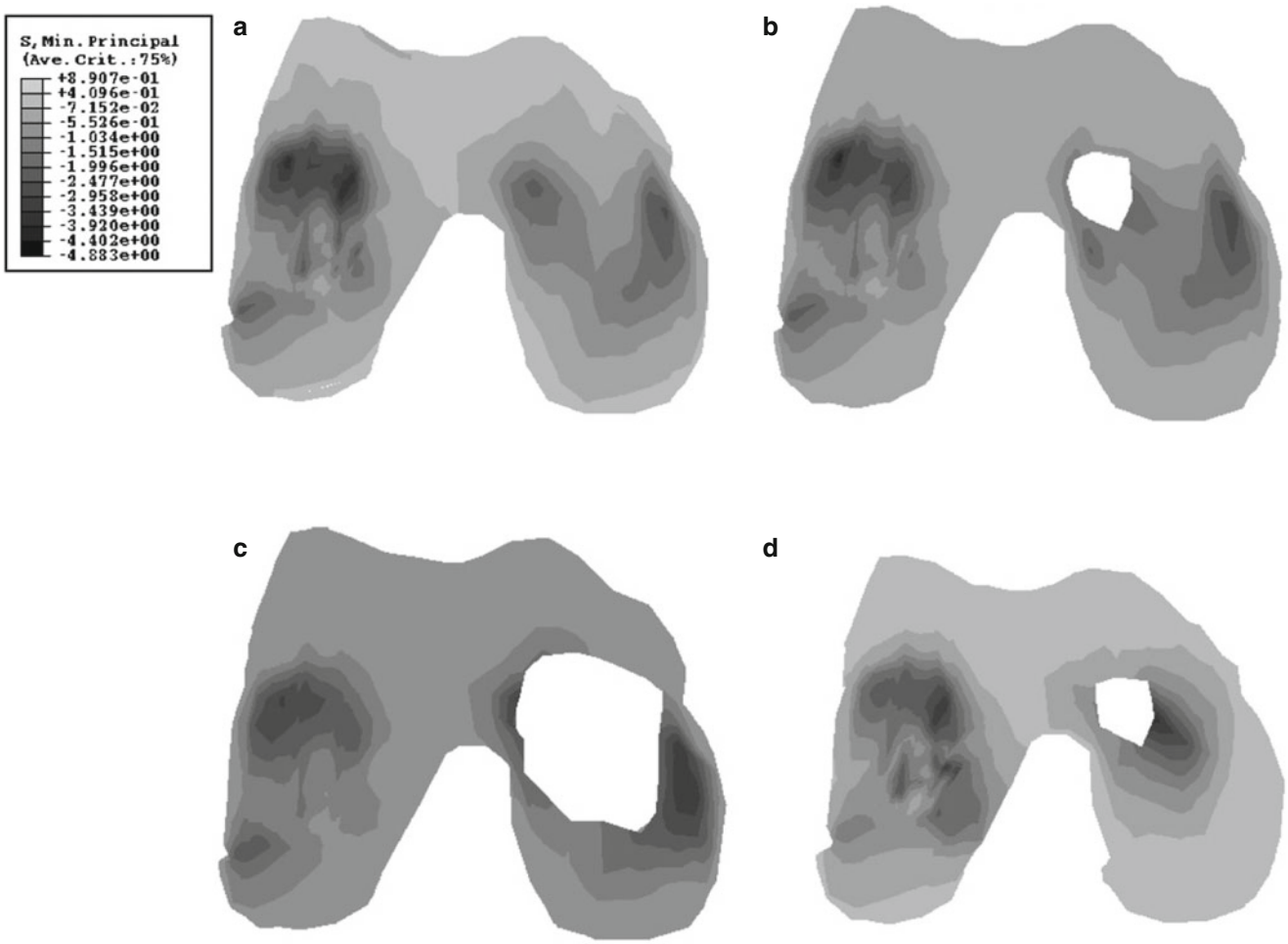



**Fig. 18.1** Both lesion size and menisectomy influence the stress on cartilage surrounding a focal defect. In this finite element model of the knee, the threshold size of the lesion was 0.78 $cm^2$ (**b**). Even though the lesion is located in a high-load area, the stress on the surrounding cartilage is the same as in the normal knee (**a**). A larger lesion (**c**, 3.14 $cm^2$) causes significantly increased stress in the adjacent cartilage. A menisectomy (**d**) also decreases the threshold size because the meniscus is unable to take up the increased mechanical load from the defect (Adapted from Peña [9], Reprinted with permission)

The rationale for this is that in a congruent joint the cartilage deforms only a small amount but that the much larger area of contact between the opposing cartilage surfaces is able to distribute the load and maintain an appropriate level of stress. For an incongruent joint with thicker cartilage, the greater degree of cartilage deformation increases the contact area between joint surfaces to decrease the stress to an acceptable level [12].

Although the hip is generally considered to be a congruent joint, it is actually slightly incongruent. The slight incongruency of the femoral head and acetabulum enables the formation of a pressurized fluid layer between the cartilage surfaces, resulting in more efficient load bearing [13]. Contact between the acetabulum and femur is first established at the labrum and chondrolabral junction at the periphery of the joint. The load is then transmitted to the pressurized fluid layer [13, 14].

The area of contact between the acetabular and femoral cartilage varies with the load and phase of the gait cycle [14]. Based on this finding, the cartilage surfaces in the hip can be described by four different types of contact:

- Habitual contact: Surfaces that make contact at the lowest loads. This occurs at the anterior and posterior portions of the acetabulum and femoral head.
- Position-dependent contact: Contact depends on the position of the hip, but can occur at low loads. This occurs at the anterior and posterior aspects of the inferior femoral head.
- Load-dependent contact: Contact occurs at higher loads, not at low loads, and is independent of hip position. The contact between the acetabular dome and femoral head is load-dependent.
- Habitual non-contact: No contact at any position or load. This is true of the periphery, perifoveal, and inframedial portions of the femoral head.

The cartilage in the hip is stiffer and less permeable than cartilage in the knee [11]. The stiffness varies somewhat in the hip such that the cartilage at the inferior aspect of the femoral head is the softest, while the stiffest cartilage is at the superomedial and posteromedial femoral head [11]. Interestingly, there is some stiffness mismatch between the cartilage that is in contact during particular motions. When seated, the anterior acetabulum and inferior femoral head cartilage contact each other; however, cartilage in the anterior acetabulum is stiffer than the corresponding cartilage on the inferior femoral head [11]. It is not yet known if this contributes to the patterns of degenerative arthrosis seen in the hip.

Studies of the labrum and finite element modeling of the cartilage and labrum have provided further insights. Finite element studies and a cadaveric model suggest that the labral seal is important for maintenance of the pressurized fluid layer between the acetabulum and femur [15–17]. Thus, loss of the labral seal increases the cartilage load and the potential for degenerative changes. Correspondingly, an MRI study of cartilage strain found that strain decreased after labral repair as compared to labral resection [18].

Because the hip is more constrained than the knee, the combination of size and location of a cartilage defect may be important. For example, the stress on the adjacent cartilage may be different if the entire rim of the defect is within the contact area and is loaded as compared to a partially loaded defect with an area of focal stress on the rim [7]. Thus, the effect of lesion size may be different in the hip as compared to the knee. Because cartilage contact first occurs at the periphery of the hip, the threshold size may be different for lesions near the labrum or the adjacent area on the femoral head. In addition, if the labrum is not intact or functioning normally, the congruency of the joint and the fluid layer change. An abnormal labrum may decrease the threshold size of a cartilage defect. On some MR arthrograms performed for patients with FAI, the femoral head was observed to settle into an anterosuperior acetabular cartilage defect [19]. During open surgical dislocation, the defect was found to be substantial, ranging from one-third to one-half of the cartilage width. All of these cartilage defects were associated with labral lesions. This suggests that, in addition to the threshold size of a cartilage defect, there is also an interaction between defect size, location, and associated labral lesions.

In the hip, the percent of the involved cartilage surface area is likely more important than the absolute size of the lesion. Thus, a cartilage lesion in a female patient with a smaller femoral head may be much worse than the same size lesion in a male with a larger femoral head, because the defect takes up a larger portion of the femoral head surface area. Another important and unanswered question is the influence of the concavity or convexity of the surface. For example, would a focal cartilage defect on the acetabulum be less likely to progress than one of the same size on the femur? The acetabulum is a concave surface and the edges of a lesion face relatively inward as compared to convex surface of the femur where the edges of a cartilage lesion would be relatively outward facing. In addition, the quality of the subchondral bone is different between the acetabulum (relatively harder) and the femoral head (relatively softer). The relevance of this to the likelihood of defect progression or for potential therapy has not yet been investigated.

## Clinical Evaluation

### General Considerations

The treatment goals for a patient with a focal cartilage lesion are resolution of pain, restoration of function, and return to activity. There are some general considerations and important factors that influence the treatment protocol, the specific technique used for the cartilage lesion, and the overall prognosis.

Three different classification schemes are commonly used for cartilage lesions in the hip (Table 18.1). The Outerbridge classification was originally described for lesions of the patellofemoral joint [20], but is widely used for cartilage lesions in other joints as well. The Outerbridge grade helps to characterize lesions that have a better prognosis (Grade I or II lesions) as compared to lesions with a poorer prognosis (Grade III or IV lesions) [21]. The ICRS grading system is similar to the Outerbridge system and is part of the overall evaluation in patients undergoing cartilage repair [22]. The Beck classification of cartilage damage was originally described for patients undergoing surgical dislocation for FAI, but is also useful for grading of hip cartilage lesions due to other causes [19].

The location of the lesion is important. Clinically, acetabular defects may have a better prognosis than femoral head lesions. On long-term follow up of hip arthroscopy patients, lesions on the femoral head had worse prognoses than acetabular lesions [21]. In theory, lesions on the non-weightbearing portion of the femoral head could be treated conservatively, similar to the treatment for a Pipkin I femoral head fracture; however, there is no discussion of this in the literature.

For more advanced cartilage restoration techniques, the diameter of the femoral head, the size of the lesion, and the quality of the adjacent subchondral bone are important factors that may dictate treatment options. Finally, any associated bony pathology should also be addressed, either concomitantly or in a staged manner to prevent further damage to the reconstructed area. Thus, this may include treatment of associated osteochondral fractures due to an acute dislocation or subluxation event; arthroscopic or open management of FAI; osteotomy for acetabular dysplasia, Perthes or rotational malalignment; or recognition of avascular necrosis and potential for femoral head collapse.

**Table 18.1** Cartilage classification systems

| Grade | Criteria | |
|---|---|---|
| *Outerbridge classification* [20] | | |
| I | Cartilage swelling or softening | |
| II | Cartilage fragmentation or fissuring, <0.5 in diameter | |
| III | Cartilage fragmentation or fissuring, >0.5 in diameter | |
| IV | Cartilage erosion to bone | |
| **Stage** | **Description** | **Criteria** |
| *ICRS classification* [22] | | |
| 0 | Normal | Normal cartilage |
| 1 | Nearly normal | Superficial lesions; soft indentation, superficial fissures or cracks |
| 2 | Abnormal | Lesions extending down to <50 % of cartilage depth |
| 3 | Severely abnormal | Defects extending >50 % of cartilage depth as well as to the calcified cartilage, but not through subchondral bone. Includes cartilage blistering. |
| 4 | Severely abnormal | Cartilage defect that extends into the subchondral bone |
| *Beck Classification* [19] | | |
| 0 | Normal | Macroscopically sound cartilage |
| 1 | Malacia | Roughening of surface, fibrillation |
| 2 | Pitting malacia | Roughening, partial thinning and full-thickness defects or deep fissuring to bone |
| 3 | Debonding | Loss of fixation to the subchondral bone, macroscopically sound cartilage, carpet phenomenon |
| 4 | Cleavage | Loss of fixation to the subchondral bone, frayed edges, thinning of the cartilage |
| 5 | Defect | Full-thickness defect |

## History and Physical Exam

The treatment plan should be individualized for each patient and developed from a collective assessment of the injury mechanism, symptoms, physical exam, radiographic findings, and the result of an intraarticular diagnostic injection with local anaesthetics. The time course of symptom onset is important. Symptoms that began with a relatively minor trauma or the insidious onset of pain generally occur in combination with underlying bony pathology. For patients with symptoms directly attributable to an acute trauma, the damage may be related to an acute dislocation or subluxation event. Focal cartilage damage can also occur after a direct lateral impact to the greater trochanter during a fall [23]. Because there is often little soft tissue to absorb the force of the fall, the force is transmitted to the central joint surface. Characteristically, the resulting cartilage lesions are focal defects in either the medial femoral head or in weightbearing portion of the acetabulum just above the fossa [23].

The quality of symptoms is less specific for cartilage pathology. Nonetheless, mechanical symptoms including sharp groin or buttock pain, stiffness, clicking, popping, or catching may suggest a loose cartilage flap or fragment [23, 24]. Labral tears can also present with similar mechanical symptoms, and labral tears and cartilage lesions often occur concurrently.

No single examination maneuver is specific for chondral pathology. Pain with weightbearing or specific examination maneuvers may depend on the location of the lesion. Pain that is provoked by logrolling the hip is generally indicative of associated synovitis or a synovial effusion.

## Radiographic Findings

We routinely order AP pelvis and cross-table lateral x-rays for all young patients with hip pain. These should be closely scrutinized for dysplasia, FAI, joint incongruity, and arthritic changes.

Patients also undergo magnetic resonance imaging with intraarticular contrast (MR arthrogram). On high quality MR arthrography, the cartilage defect can be seen directly and any associated labral tears can be evaluated. In general, MR arthrography for a suspected hip labral tear or cartilage lesion should be performed with either a 1.5 Tesla (T) or a 3 T magnet, and a small field of view coil. Sequences should include coronal, sagittal, axial, and radial images. Although MR arthrography is useful for evaluating the labrum, it is somewhat less effective for evaluation of the articular cartilage [24, 25]. The size of a cartilage lesion should be measured, and the quality of the underlying bone evaluated, including the presence of bone marrow edema or cystic changes. Edema in bone adjacent to a cartilage lesion may be indicative of a recent trauma [23] or of local overload [24]. Delayed gadolinium-enhanced MRI of cartilage (dGEMRIC) is one means of assessing the biochemical integrity of cartilage. DGEMRIC is most often used for cartilage biology research, but is not routinely used clinically [26].

### Diagnostic Injection

The response to an intraarticular injection often helps with surgical decision-making, particularly when the imaging or examination findings are equivocal. Diagnostic injection is sensitive and specific for intraarticular pathology, with 90 % accuracy for determining whether the pathology is intra or extraarticular [27].

## Treatment Options and Results

The indications for treatment of focal cartilage defects in the hip include: acute trauma with an unstable cartilage fragment, loose bodies visible on pre-operative imaging, or continued pain despite conservative management with a defect visible on preoperative imaging and a positive response to diagnostic injection. The timing of surgery is dependent on the type of lesion, the age of the patient, and the type and duration of previous interventions. Often the choice of arthroscopic or open management is dependent on the location of the lesion and associated bone and labral pathology. We perform arthroscopy with the patient supine. For open treatment of cartilage lesions in the hip, we perform a surgical hip dislocation as it allows for wide intraarticular access, treatment of FAI and other bony pathology, and preserves the blood supply to the femoral head. This has been described extensively in multiple publications [1, 19, 28].

### Non-operative Management

For patients with the insidious onset of pain and a stable cartilage lesion on MR arthrography, an initial course of nonoperative management is appropriate. This generally entails some combination of activity modification, physical therapy, medical management, and injections, all of which are well-covered in other chapters of this book.

### Operative Management

#### Direct or Primary Cartilage Repair

Direct or primary cartilage repair is indicated for an acute unstable osteochondral fragment or an unstable osteochondritis dissecans lesion. This requires a surgical dislocation to access the joint. The femoral head is gently dislocated to minimize further damage to the unstable fragment. The unstable fragment is then elevated and any fibrous or cystic tissue at the base is then debrided. Sclerotic bone at the base of the lesion should be microfractured or drilled. Areas of cystic change or bone loss should be bone grafted with cancellous autograft from the stable portion of the trochanter. The fragment is then fixed back to the donor site rigidly and under compression with a headless compression screw [5].

#### Arthroscopic Debridement and Chondroplasty

Arthroscopic debridement of a cartilage defect is considered to be a palliative therapy. Nonetheless, debridement of loose bodies or flaps of cartilage can be quite effective for relieving symptoms and allowing patients to return to activity [23]. It is not unusual for patients undergoing hip arthroscopy for treatment of FAI or labral pathology to have a concomitant and undetected cartilage lesion. Furthermore, if preoperative imaging was inconclusive but suspicious for labral or cartilage lesions, arthroscopy is considered definitive for the diagnosis. Arthroscopic debridement may also be indicated for patients with dysplasia undergoing acetabular reorientation and who have mechanical symptoms [29].

#### Microfracture

Microfracture is one type of bone marrow stimulation technique. All bone marrow stimulation techniques involve perforation of the subchondral plate with either a microfracture awl or drill to promote bleeding from the bone marrow into the lesion [30–33]. This results in the migration of mesenchymal stem cells and formation of a "superclot" in the lesion. The ultimate goal is complete filling of the defect with reparative fibrocartilage because the best functional results correlate with the degree of defect filling [34, 35]. The stability of the superclot contributes to the success of the procedure [36, 37]. Thus, the walls surrounding the lesion should be vertical and consist of normal, stable cartilage. This decreases shear and compression forces on the clot and protects it during healing [5]. The advantage of microfracture and other bone marrow stimulation techniques is that they are technically straightforward, low cost, and can be performed arthroscopically. The disadvantage is that fibrocartilage contains less type II collagen and has different biomechanical properties than hyaline cartilage. This may make the reparative cartilage less durable over the long-term [36]. In addition, the overall concentration of mesenchymal stem cells in the bone marrow is low and the chondrogenic potential declines with age [38].

*Technique*. Microfracture is indicated for full thickness lesions in patients undergoing concomitant open or arthroscopic management for FAI or dysplasia [29]. It can be performed arthroscopically for acetabular rim lesions in the contact area as well as for accessible lesions on the femoral head. Microfracture is contraindicated for large or extensive lesions, bipolar or kissing lesions, and for patients who are unwilling to undergo the postoperative rehabilitation.

Once the lesion has been identified, unstable cartilage should be debrided to re-establish stable vertical cartilage walls of the lesion. The calcified cartilage layer at the base of the lesion should be removed with a sharp ring curette so that

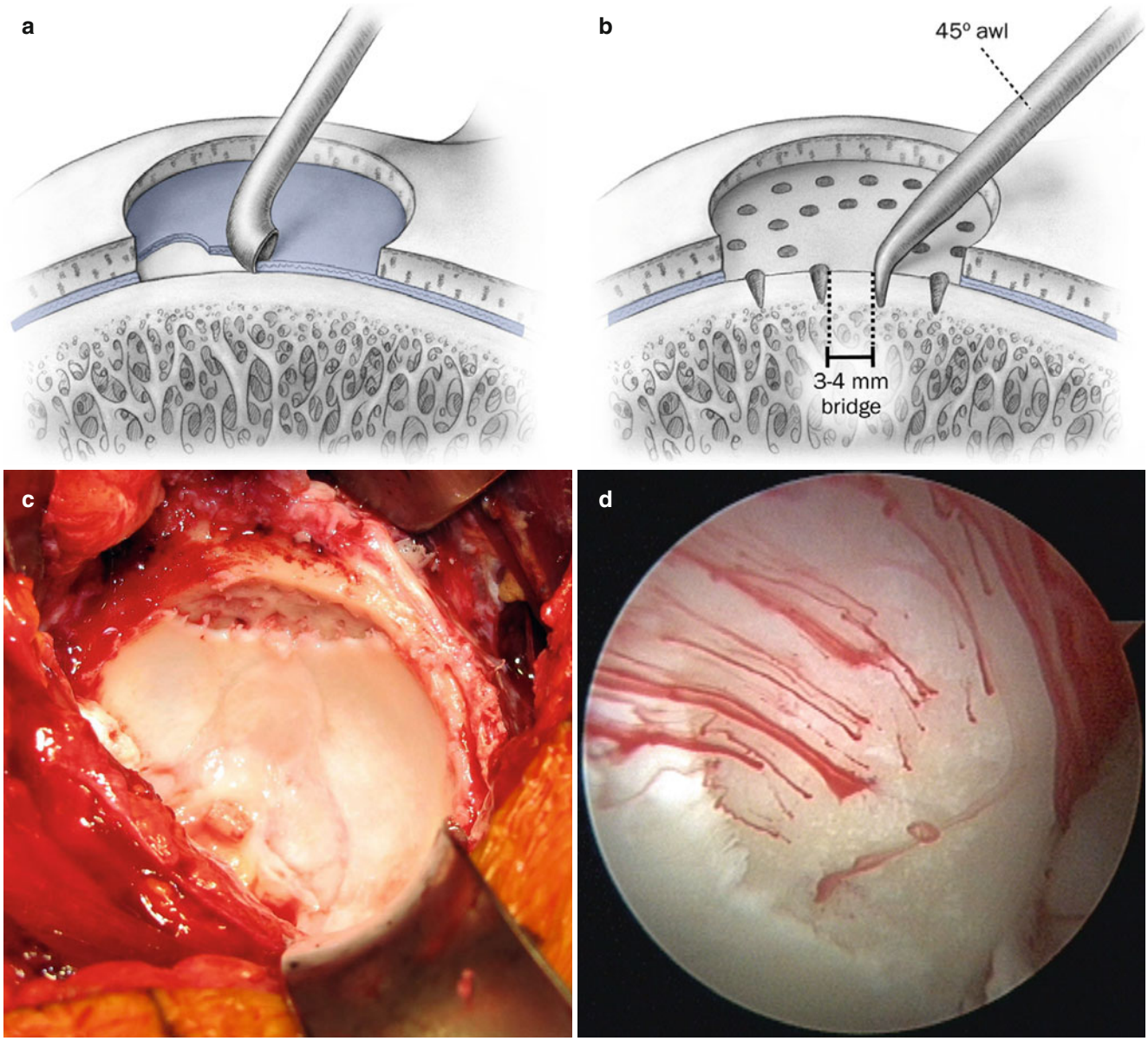



**Fig. 18.2** Microfracture technique. (**a**) Preparation of the defect includes creating stable vertical walls and removing the calcified cartilage layer with a ring curette. (**b**) Microfracture awls are then used to perforate the subchondral surface. Holes should be spaced 2–3 mm apart and be perpendicular to the surface. (**c**) Microfracture of the acetabulum performed during a surgical hip dislocation. (**d**) Arthroscopic image of an acetabularmicrofracture. The blood and fat droplets coming from the surface indicate that the holes communicate with the bone marrow and that the microfracture is of adequate depth (**a** and **b** reprinted with permission from Mithoefer et al. [39])

the subchondral bone is visible (Fig. 18.2a). This is important for clot adhesion and the overall success of the procedure. Microfracture awls are then used to penetrate the subchondral surface (Fig. 18.2b, c) [39]. A 1.1 mm wire can also be used to drill the subchondral surface. Holes should be perpendicular to the surface and spaced 2–3 mm apart. Extreme care should be taken to avoid confluence of the holes and destabilization of the subchondral plate. Following arthroscopic microfracture, the pressure in the joint should be decreased so that blood and fatty droplets can be seen coming from the surface (Fig. 18.2d). This is indicative of communication with the bone marrow and appropriate depth of the microfracture [4].

Patients begin CPM on postoperative day 1 for 6–8 h per day [31, 39]. This promotes clot healing, joint nutrition, and decreases adhesion formation. To protect the healing lesion, patients should be non-weight bearing for 6–8 weeks postoperatively [31, 39].

Results for microfracture in the hip have generally been reported as part of combined therapy for FAI [40–42]. In a cohort of FAI patients with concomitant pathology, patients who had microfracture did better than patients who had simple

debridement [41]. For larger lesions, the greatest improvement was seen by 8 weeks but was maintained at 12 month follow up [41]. In one series of second-look arthroscopy after microfracture, there was fibrocartilage fill of most or all of the defects [43]. Nonetheless, patients with more extensive lesions still progress to total hip arthroplasty [21, 40, 41, 43], indicating that the technique is limited by the size and extent of the lesion. These are similar to the results reported for microfracture of lesions in the knee, where the best outcomes are seen in younger patients with small traumatic lesions [31, 35]. Age and lower body mass are both independent predictors of improvement [31, 34, 35], with good to excellent results reported in 67 % of patients, fair results for 25 % of patients, and poor results in 8 %. In comparison to other procedures, the results of microfracture may deteriorate over time [34, 35]. Finally, osteophyte formation rather than fibrocartilage fill has been observed in 25–50 % of cases [34], decreasing durability and patient satisfaction with the procedure.

## Second-Generation Bone Marrow Stimulation (AMIC)

A second-generation bone marrow stimulation technique has been developed, encompassing concepts from both microfracture and autologous chondrocyte implantation (ACI). This technique has been dubbed autologous matrix-induced chondrogenesis (AMIC). Essentially, AMIC consists of microfracture with the subsequent application of a collagen I/III membrane over the lesion to protect the clot and facilitate chondrocyte differentiation from mesenchymal stem cells [37, 44]. It is frequently compared to ACI and matrix-associated chondrocyte implantation (MACI), but is relatively less expensive and can be performed in a single surgery.

AMIC is indicated for symptomatic full-thickness cartilage lesions and osteochondral lesions in weightbearing regions of both the acetabulum and femoral head. Because the membrane protects the microfracture clot, it should be possible to successfully perform AMIC for lesions that are too large to undergo routine microfracture.

*Technique*. A surgical hip dislocation is performed and the cartilage lesion is evaluated. If the lesion is appropriate for AMIC, we prepare the lesion as described above for microfracture. Instead of microfracture awls, we use a 1.1 mm Kirschner wire to penetrate the subchondral plate (Fig. 18.3b). If there is subchondral bone loss, an autogenous cancellous bone graft from the stable portion of the trochanteric osteotomy is used to fill the defect, such that the bone is even with the surrounding subchondral bone. An aluminum foil template is used to determine the size and shape of the membrane (Fig. 18.3c). Then, autologous or commercially available fibrin glue is used to fill the defect and entrap the clot. The membrane is sized according to the template and sewn into the lesion with 6–0 vicryl suture. Sutures should be placed about 4 mm apart, taking care to place the knots on the "patch" side of the lesion (Fig. 18.3d). The membrane should be slightly below the joint surface to prevent shearing once the hip is reduced. Fibrin glue is used to seal and smooth the edges of the membrane.

Postoperatively, patients are treated the same as patients who have had a microfracture: CPM, partial weightbearing to 10 kg and limited flexion to 70° for 6–8 weeks.

*Results*. We have performed AMIC for six patients with follow up ranging from 7 to 24 months. Pain scores subjectively improved for all patients, with no complications and three patients reporting complete resolution of their pain. MRIs performed 6 months postoperatively show resolution of pre-operative bone marrow edema and cystic changes, and no progression of degenerative changes (Fig. 18.4). Similar results have been reported for patients who underwent AMIC in the knee, with improved clinical outcome scores and postoperative MRIs showing healing of the lesion and resolution of pre-operative bone marrow edema [44].

## Autologous Chondrocyte Implantation and Matrix-Associated Chondrocyte Implantation

The basic principle behind both autologous chondrocyte implantation (ACI) and matrix-associated chondrocyte implantation or transplantation (MACI, MACT) is implantation of cultured autologous chondrocytes into a cartilage defect [45]. MACI and MACT are subsequent-generation techniques where chondrocytes are delivered on absorbable scaffolds to support the cells during healing. The theoretical advantage of both ACI and MACI is that they have the potential to restore hyaline cartilage in the defect. However, both ACI and MACI are two-stage procedures, requiring an initial arthroscopy for chondrocyte harvest. Similar to AMIC, ACI and MACI also require application of a synthetic collagen membrane to cover the defect. This can be technically challenging. ACI is classically performed with a periosteal patch covering the implanted cells [45], although most physicians now use a synthetic collagen membrane as it minimizes surgical time and decreases complications related to the periosteum [5].

*Technique*. ACI or MACI is indicated for symptomatic, unipolar, well-contained defects measuring 2–10 $cm^2$ with no more than 6–8 mm of subchondral bone loss. An initial arthroscopy is performed to evaluate the size and depth of the lesion and to obtain a cartilage biopsy for culture. The second stage is performed 6 weeks later. A surgical dislocation is performed to access the cartilage lesion. Calcified cartilage is debrided from the base of the lesion with a ring curette. The lesion should then be carefully debrided back to stable vertical walls with a 15 blade and ring curettes. Complete hemostasis must be obtained as bleeding can affect chondrocyte viability. This can be facilitated with epinephrine-soaked pledgets. The synthetic collagen membrane can then be sewn or glued into the walls of the lesion, depending on

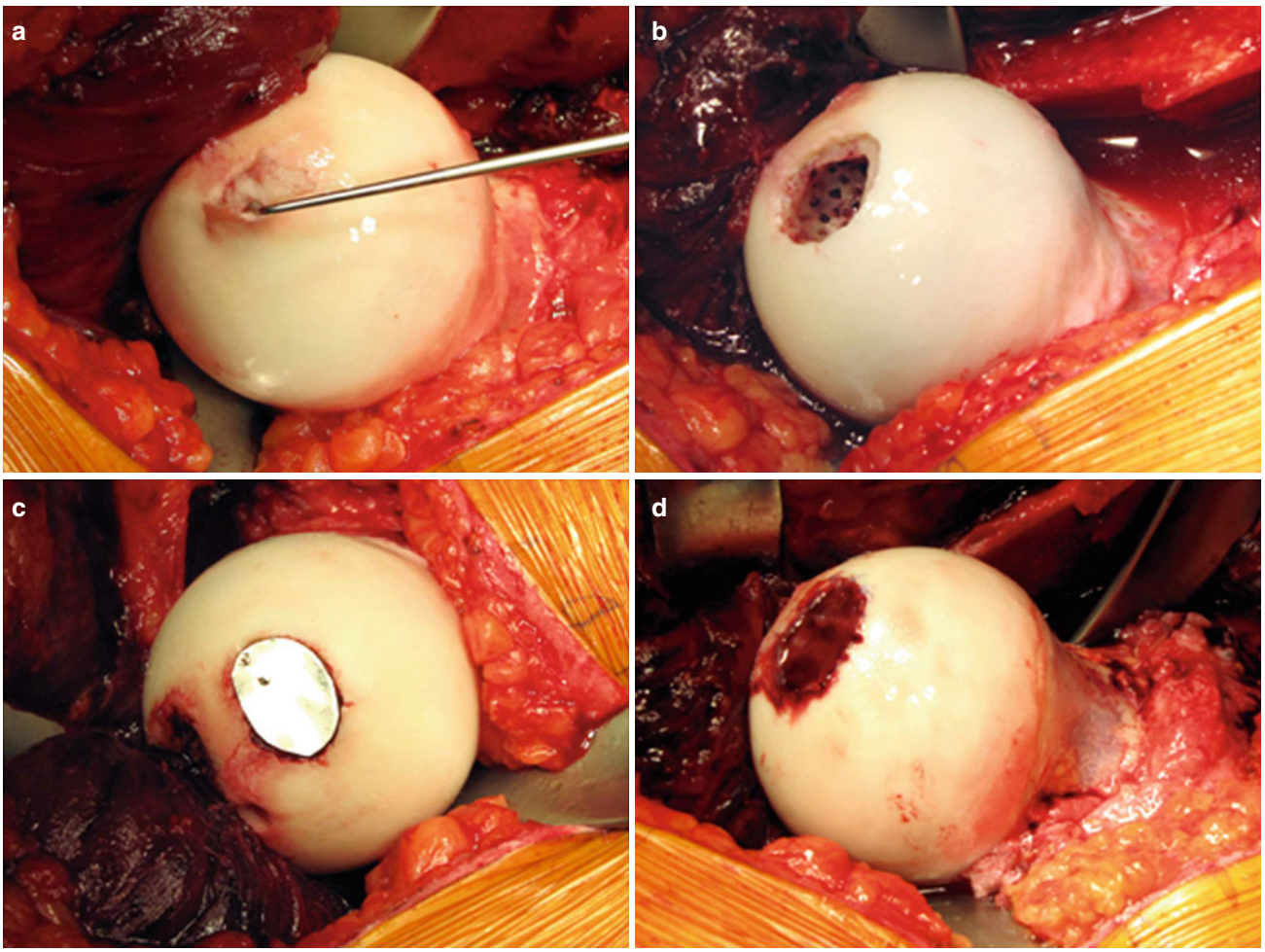
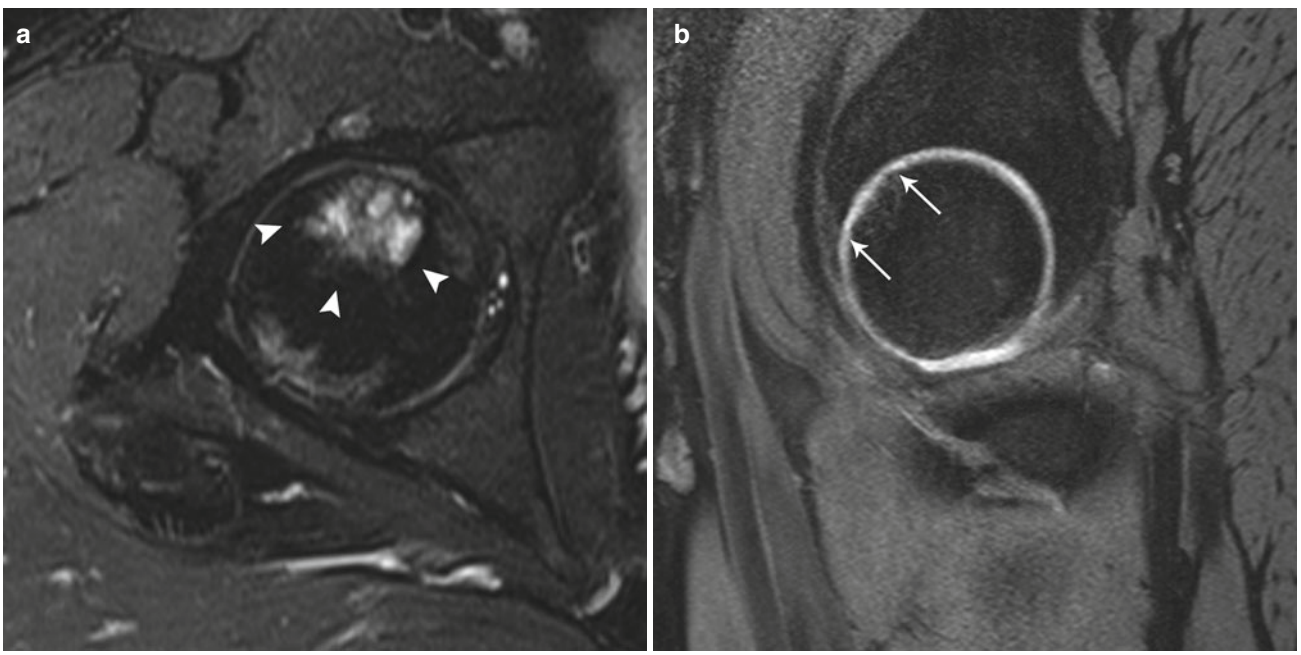



**Fig. 18.3** Autologous matrix-induced chondrogenesis. (**a**) Chondral defect in the femoral head. (**b**) Preparation of the defect includes removal of all degenerative and unstable cartilage, unstable or necrotic subchondral bone, and drilling at the base of the defect with a 1.1 mm K-wire. (**c**) The size of the defect is templated with a sterile aluminum foil so that the collagen membrane can be cut to fit. (**d**) Finally, the collagen membrane is sewn into place with 6–0 vicryl suture. Note that the knots are on the "patch side" of the defect



whether ACI or MACI is being performed. When performing ACI, the membrane is sewn in as described above for AMIC with the exception that a gap is left on one side of the lesion for chondrocyte implantation. Fibrin glue is then used to seal the patch and water-tightness is tested with an 18 gauge angiocatheter. The water is removed and the chondrocytes are delivered through the opening in the membrane with the angiocatheter. The gap is then sutured and sealed with fibrin. Postoperatively, patients remain non-weightbearing for 6–8 weeks with CPM and hip flexion limited to 70°.

*Results*. The results for ACI in the hip are limited to one case report with short term follow up [46]. There are more results for ACI and MACI in the knee. Most of these are case series, reporting 75–85 % good results [4, 5, 47]. Patients who have undergone ACI with a periosteal patch may require additional procedures for problems related to the periosteum, including adhesions and periosteal hypertrophy [48]. Although ACI is often used after failed microfracture or debridement, the results of ACI after microfracture are worse than for patients who previously only had debridement of the lesion [49, 50]. Mid-term results for MACI in the knee have been reported, with high patient satisfaction scores for pain relief (98 %) [51]. Graft hypertrophy and associated mechanical symptoms were observed in 10–20 % of patients, although these symptoms improved following arthroscopic debridement [51].

## Osteochondral Autograft Transplantation (OATS)

Osteochondral autograft transplantation (OATS or mosaicplasty) involves transplanting healthy mature cartilage from a nonweightbearing part of the hip or knee to a focal defect. The graft undergoes osseous integration with the subchondral bone and the cartilage integrates with the adjacent host cartilage via fibrocartilage [5, 52]. The advantage of OATS is that it involves the transplant of mature hyaline cartilage in a single-stage procedure. Disadvantages of OATS include morbidity at the donor site, limited graft availability, and potential dead space between grafts [4] (Fig. 18.5).

*Technique* (Fig. 18.2). OATS is indicated for small to medium-sized focal lesions on the femoral head and acetabulum. The size of the lesion that can be treated is generally limited by the amount of donor cartilage [52]. Although OATS can be performed arthroscopically in the knee, a surgical hip dislocation is required for appropriate access to the femoral head and acetabulum. A commercially available system is used for both graft harvest as well as preparation of the recipient site. A sizing guide is used to determine the number of grafts needed to fill the defect. The graft can be taken from either the non-weightbearing portion of the femoral head or lateral femoral condyle in the knee. When harvesting the

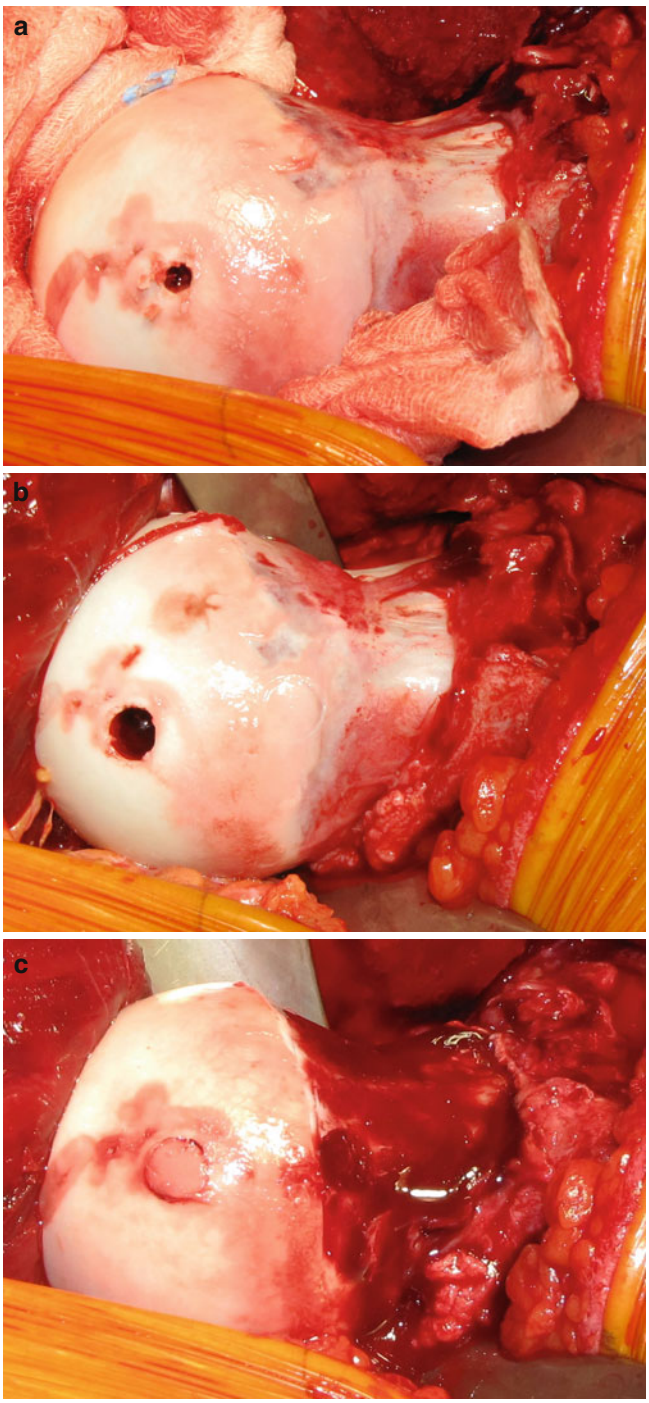



**Fig. 18.5** Osteochondral autograft transplantation (OATS or mosaicplasty). (**a**) A chondral defect with a large cyst was present in the femoral head. The lesion has been debrided and the cyst was curetted as part of the lesion preparation. (**b**) The sizing guide was then used to prepare the defect for the autograft, taking care to create vertical walls of the recipient site. (**c**) The femoral head after placement of the graft and osteoplasty for a cam deformity at the head-neck junction. The donor site can be seen just lateral to the femoral head cartilage and was part of the cam deformity

**Fig. 18.4** (**a**) Pre-operative axial T2 MRI from the patient in Fig. 18.5. Note the cystic changes within the lesion (*arrowheads*). (**b**) Sagittal T2 MRI obtained 6 months postoperatively. The joint space has been maintained, the cartilage surface appears regular, and the subchondral bone appears normal. *Arrows* indicate the extent of the bone graft and cartilage repair

graft and preparing the recipient site, it is important to create well-defined vertical walls perpendicular to the cartilage surface (Fig. 18.2b). This enables congruent plug placement [5]. The goal is to create a press-fit implant flush with the adjacent cartilage surface because elevated grafts increase contact pressure in the graft surface [53]. Chondrocytes can be damaged from the force of impaction, so the graft should be inserted carefully [54]. Postoperatively, the rehabilitation protocol is the same as for other cartilage procedures: patients are non-weightbearing for 6–8 weeks with CPM beginning on postoperative day 1.

*Results*. There have been a few case series published for patients undergoing OATS in the hip for various indications. Authors generally report good results in short-term (2 years) follow up [55–58]. In a larger series of patients treated for Perthes disease, four patients underwent OATS for osteochondral defects with anecdotally good results [28]. One exception to the otherwise good results was a series of OATS performed for avascular necrosis with 4 out of 5 patients having a poor result and progressing to hip arthroplasty [3]. Reliably good results have been reported for OATS in the knee by several investigators, with long-term results being published by the developer of the technique [4, 59]. The result appears to be durable and, for larger lesions in particular, the results of OATS are significantly better than those for microfracture [4, 5, 59].

### Osteochondral Allograft

Osteochondral allograft transplantation involves transplantation of intact viable cartilage and the underlying subchondral bone into a cartilage defect [60]. Because cartilage is relatively immunoprivileged with an avascular extracellular matrix, the host immune reaction to the transplant is limited [60]. As part of the healing process, the allograft bone becomes necrotic and is subsequently absorbed. During the healing process, however, the allograft provides a scaffold for bony ingrowth and supports the articular surface [61]. As compared to OATS, osteochondral allograft can be used for larger defects because it is not limited by donor site morbidity [60]. Disadvantages to osteochondral allograft include graft availability, cost, risk of rejection, and the possibility of incomplete incorporation or disease transmission. In addition, it can be technically demanding to size the allograft to the recipient site [4].

*Technique*. Osteochondral allografting is indicated for treatment of larger lesions or for lesions with substantial associated bone loss. It is performed through a surgical dislocation. Fresh allograft should be used in all cases as freezing decreases chondrocyte viability [62]. The graft should be slowly warmed from 4° to 37° in room temperature normal saline. Similar to OATS, commercially available kits are helpful for sizing and orienting both the graft and the recipient site. In many cases, press-fit fixation is sufficient for graft stability. When necessary, however, headless compression screws may also be used for fixation. Like other cartilage restoration procedures, patients remain non-weightbearing for 6–8 weeks post-operatively with CPM beginning on post-operative day 1.

*Results*. A few case reports have been published for osteochondral allografting in the hip. The results are mixed and appear to be technique-dependent. Short-term (2 years) follow up after fresh osteochondral allograft to either the acetabulum or femoral head was promising, with patients having near-normal Harris Hip scores postoperatively [63, 64]. In contrast, a patient who had a fresh-frozen osteochondral allograft for a severe fracture-dislocation had progressive degenerative changes and full-thickness cartilage loss 4 years post-operatively [65]. In a much older series published prior to the description of the surgical hip dislocation, the results of osteochondral allograft transplants for avascular necrosis were mixed [2]. In the knee, 75–85 % of appropriately selected patients subjectively improved after osteochondral allograft transplantation [5]. Increased failure rates have been observed in bipolar lesions, patients with ligamentous instability, and in worker's compensation patients [4]. Overall survival rates of osteochondral allografts are 75–95 % at 5 years, but decrease to 63–73 % at 15 years [4].

## Summary and Conclusions

The successful treatment of focal cartilage defects in the hip is relatively new and has been facilitated by advancements in open and arthroscopic surgical techniques. Some, but not all, of the cartilage basic science and treatments developed for the knee are applicable in the hip. A better understanding of the cartilage biomechanics specific to the hip as well as more biomechanical and animal models of hip cartilage lesions would help to advance these treatments. In addition, as all of the current clinical literature consists of case series and small case reports, more prospectively collected data and longer follow up is necessary. To obtain sufficient numbers of patients, some of these may need to be multi-center studies. Nonetheless, the recent experience in treating these lesions is encouraging and appears to be of significant benefit to young and active adults with cartilage defects.

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