Current Concepts

Glenohumeral Joint Preservation: Current Options for Managing Articular Cartilage Lesions in Young, Active Patients

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Abstract: This is a review of joint-preservation techniques for the shoulder. Whereas the management of diffuse articular cartilage loss in the glenohumeral joints of elderly and less active patients by total shoulder arthroplasty is well accepted, significant controversy persists in selecting and refining successful operative techniques to repair symptomatic glenohumeral cartilage lesions in the shoulders of young, active patients. The principal causes of focal and diffuse articular cartilage damage in the glenohumeral joint, including previous surgery, trauma, acute or recurrent dislocation, osteonecrosis, infection, chondrolysis, osteochondritis dissecans, inflammatory arthritides, rotator cuff arthropathy, and osteoarthritis, are discussed. Focal cartilage lesions of the glenohumeral joint are often difficult to diagnose and require a refined and focused physical examination as well as carefully selected imaging studies. This review offers a concise guide to surgical decision making and up-to-date summaries of the current techniques available to treat both focal chondral defects and more massive structural osteochondral defects. These techniques include microfracture, osteoarticular transplantation (OATS [Osteochondral Autograft Transfer System]; Arthrex, Naples, FL), autologous chondrocyte implantation, bulk allograft reconstruction, and biologic resurfacing. As new approaches to glenohumeral cartilage repair and shoulder joint preservation evolve, there continues to be a heightened need for collaborative research and well-designed outcomes analysis to facilitate successful patient care.

With an increasingly active population and with improvements in medical technology and surgical technique, there is both a need and an opportunity for joint-preserving surgery for the glenohumeral joint. Focal cartilage lesions of the glenoid and humerus have historically been underdiagnosed by clinical examination and by imaging techniques, predisposing them to incidental discovery at the time of surgery. The incidence of symptomatic Outerbridge grade II to IV lesions of the shoulder has been reported to be as high as 5% to 17% in patients with rotator cuff tears[1] or overhead athletes.[2]

Cartilage injury in the shoulder may be caused by or associated with a myriad of factors that include previous surgery, trauma, acute or recurrent dislocation, osteonecrosis, infection, chondrolysis, osteochondritis dissecans (OCD), inflammatory arthritides, rotator cuff arthropathy, and osteoarthritis. Though symptomatic osteoarthritis of the shoulder can be successfully treated with shoulder arthroplasty, focal cartilage lesions in the younger, active patient population demand alternative treatment strategies that preserve the joint. Survival rates of total shoulder arthroplasty in younger patients are not as good as in older, less active patients, with survivorship being reported as low as 61% at 10 years in a series of 33 patients with a mean age

From the Steadman Hawkins Research Foundation, Vail, Colorado, U.S.A.

F.E., S.B., P.J.M. have received support from Arthrex, Naples, FL, exceeding US $500 related to this research.

Received August 27, 2009; accepted October 21, 2009.

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0749-8063/10/2605-9498$36.00/0

doi:10.1016/j.arthro.2009.10.017

of 46 years at the time of surgery.[3] This review will explore joint-preserving techniques for cartilage lesions of the glenohumeral joint that can be used when treating young, active patients.

ANATOMY

Recent investigations have deepened our understanding of the dimensions of the chondral surfaces of the glenohumeral articulation. At the center of the humeral head, the hyaline cartilage on the articular surface is 1.2 to 1.3 mm deep,[4] thinning at the periphery to 1.0 mm or slightly less. On the glenoid surface, hyaline cartilage is thicker at the periphery than in the center. Bone density in adults is also greater beneath the cartilage at the center of the glenoid, which has been attributed to the higher loads that are seen in this region.[5] The glenohumeral articular surface is congruent because of this varying thickness of the cartilage.[6]

INCIDENCE

The true incidence of focal glenohumeral chondral defects is unknown, because most lesions are discovered in the setting of treatment for concomitant injury. Arthroscopic evaluation of the glenohumeral cartilage surface in 200 shoulders (195 patients) with full-thickness rotator cuff tears showed a 13% prevalence of articular cartilage pathology.[1] Of the patients, 17 had minor lesions (8.5%) and only 9 (4.5%) had major full-thickness lesions that measured more than 150 mm^2. The prevalence of high-grade articular cartilage injury in high-level overhead athletes was 17% in throwers, all located near the insertion of the supraspinatus tendon.[2] Ellman et al.[7] found that 6% of patients (mean age, 51 years) who underwent arthroscopic treatment for impingement symptoms had articular cartilage lesions, which ranged in size from 15 mm in diameter to comprising 50% of the humeral chondral surface. Other authors have reported a 29% prevalence of humeral head lesions and 15% prevalence of glenoid cartilage lesions in patients who underwent arthroscopy for subacromial impingement.[8] For patients who underwent arthroscopic shoulder surgery at our institution between 1993 and 2008, there was a 12.4% prevalence (280 of 2,251) of grade III and IV lesions on 1 or both glenohumeral surfaces (unpublished data, P.J.M., September 2009).

ETIOLOGY OF CARTILAGE LESIONS

The causes of glenohumeral chondral injury may be considered in a systematic fashion. Injury patterns can be broadly considered in relation to acute or recurrent instability, high-impact trauma, post-traumatic changes of osteonecrosis or OCD, and complications or sequelae of surgery (Table 1).

Table 1. *Etiology of Cartilage Lesions*

Previous surgery
Penetrating or high-impact trauma
Acute or recurrent dislocation
Osteonecrosis
Infection
Chondrolysis
OCD
Inflammatory arthritides
Rotator cuff arthropathy
Osteoarthritis

Instability

There is a well-documented association between acute and recurrent shoulder dislocation and cartilage lesions in the glenohumeral joint. In 1934 Hermodsson[9] was the first to describe the ubiquitous compression fracture of the posterolateral humeral head that occurs with traumatic anterior instability, but it was Hill and Sachs for whom the lesion was named.[10] Studies have shown a high incidence of 47% to 100% of cartilage compression fractures or shear injury to the chondral surface in first-time anterior dislocations and recurrent dislocations of the shoulder.[11-13]

Another cause of glenoid articular cartilage defects may be glenolabral articular disruption (GLAD) lesions.[14,15] This lesion consists of an anterior-inferior labral tear associated with an injury to the glenoid articular cartilage (Fig 1) and is usually caused by a forced adduction injury to the shoulder from an abducted and externally rotated position. They may be difficult to diagnose clinically, although magnetic resonance imaging (MRI) will usually show them.

Chronic instability has been clearly linked to early glenohumeral osteoarthritis.[16,17] The time period of shoulder instability from first dislocation to surgical anterior stabilization, glenoid rim impaction fracture, Hill-Sachs lesion, age at the time of first dislocation, recurrent instability, presence of a rotator cuff tear, and clearly, any cartilage injury with an index episode of dislocation have all been independently linked to early glenohumeral osteoarthritis (Table 2).[16,18,19] Marx et al.[18] showed that patients with a history of previous shoulder dislocation had a 19 times greater risk of shoulder arthritis developing than patients without such a history. We have shown older age and

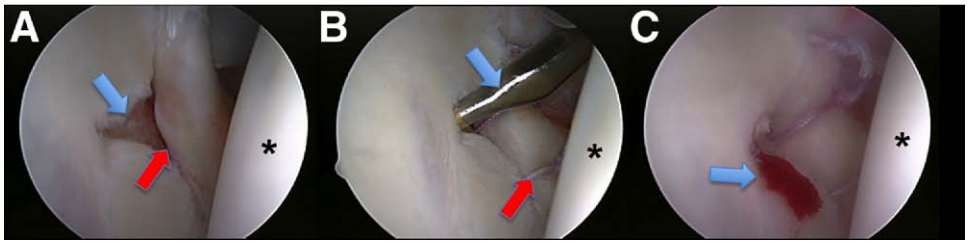


FIGURE 1. Right shoulder viewing from posterior. Asterisk, humeral head. (A) GLAD lesion in right anterior inferior glenoid (blue arrow) with associated labral tear (red arrow). (B) Microfracture of GLAD lesion (blue arrow, awl) after debridement and labral repair (red arrow). (C) Early thrombus formation at chondral defect side (blue arrow).

time from injury to surgery to be independent predictors of osteoarthritis, but no correlation between direction of instability and osteoarthritis was found.[20] We detected no difference between acute and chronic instability with regard to the prevalence and grade of cartilage injury. In contrast, Brophy and Marx[21] showed that chondral damage was associated with shoulder instability that worsened over time, although the relation has yet to be clearly defined.

Open Bankart reconstructions, as well as nonanatomic reconstructions such as the Bristow, Magnuson-Stack, and Putti-Platt procedures, have well-documented associations with degenerative chondral lesions.[21-24] In addition to the trauma of the dislocations, chondral injury may occur by excessive shear forces from nonanatomic repairs on articular cartilage. A single high-impact trauma to the shoulder may also cause chondral injury.[25] In high-energy injuries the subchondral bone is also at risk.[26-28] The underlying mechanisms relate to high compressive/shear forces during the impact with disruption of the extracellular matrix and decreases in chondrocyte metabolism.[25]

Active patients and athletes are at higher risk for traumatic cartilage lesions.[26,27] In overhead athletes most lesions are located near the insertion of the supraspinatus tendon, superior to the site of a classic Hill-Sachs lesion. These lesions can be caused by impingement between the humeral head and the posterosuperior glenoid rim.[2] Thus microinstability or shear on the cartilage plays a role in these settings.

Osteonecrosis of the humeral head may result in both focal and diffuse cartilage lesions of the humeral head. Symptomatic osteonecrosis of the humeral head is not uncommon, second in location only to the femoral head, and it affects patients at younger ages more than other degenerative conditions of the glenohumeral joint. Given the diminished weight-bearing role of the shoulder, however, many cases are not detected until advanced stages occur. The articular surface of the humeral head in contact with the glenoid in midrange levels of motion usually collapses first. It is critical to avoid loaded overhead activity to preserve the surrounding cartilage during nonoperative or postoperative rehabilitation.

Classification follows a modified Ficat scheme, as described by Cruess.[29] If detected in early stages, osteonecrosis of the humeral head may be surgically managed by core decompression and bone grafting.[30] Arthroscopic debridement of unstable, focal cartilage flaps and lesions has also shown good short-term outcomes at 28 months,[20] with significant improvement in pain and function in 54 of 61 patients with grade IV cartilage damage. Other authors have described good or excellent results in 80% of patients at 30 months' follow-up.[31]

Osteochondritis dissecans

OCD is rare in the shoulder and is more commonly seen in the femoral condyles or the talus. The disease is attributed to fragmentation of the subchondral plate resulting in destabilized chondral flaps and lesions. The precise etiology of OCD remains unclear, but it likely results from repetitive microtrauma or isolated major trauma.[32] Most lesions have been described in young male patients in the anterosuperior aspect of the humeral head.[32-35]

TABLE 2. *Predisposing Factors for Early Glenohumeral Osteoarthritis in Chronic Instability*

Cartilage injury with index episode of dislocation
Glenoid rim impaction fracture
Hill-Sachs lesion
Age at time of first dislocation
Recurrent instability
Presence of rotator cuff tear
Time period from first dislocation to anterior stabilization

Treatment options for OCD lesions include debridement, removal of loose flaps or bodies, microfracture, bone grafting with or without autologous cartilage implants (autologous chondrocyte implantation [ACI]), and transfer of osteochondral plugs.[35,36] The optimal approach to surgical management remains controversial.

Cartilage injury associated with rotator cuff tears

Several studies identify a correlation between rotator cuff pathology and articular cartilage damage in the glenohumeral joint.[1,2,35] Focal cartilage lesions have been identified in 13% of shoulders in a large series of full-thickness tears.[1] Neer et al.[37] described the concept of rotator cuff arthropathy, which results from the combination of massive rotator cuff tears and destructive glenohumeral wear patterns. According to Neer et al., the process is multifactorial. Both mechanical factors, such as impingement and instability of the shoulder, and metabolic considerations, such as malnutrition from inactivity and synovial fluid extravasation in massive rotator cuff tears, have been proposed. Both atrophy and degeneration of the articular cartilage and osteoporosis of the subchondral bone of the humeral head may result. A genetic predisposition for increased calcium crystal deposition has also been shown to hasten the onset of arthritis in the setting of massive cuff tears.[38]

Iatrogenic injury

Iatrogenic damage to the articular cartilage can be subclassified into chondrolysis (chemical, thermal, idiopathic), infection, and mechanical factors. The development of glenohumeral chondrolysis after shoulder arthroscopy is devastating, and some of the more recent reports in the literature have highlighted some of the potential causes.[39] Several case reports have shown extensive glenohumeral chondrolysis after seemingly routine shoulder arthroscopy, thermal capsulorrhaphy, or rotator cuff repairs.[40-42] Little had been known about the underlying cause of this rare but devastating complication. Ablative thermal energy and intra-articular pain pumps are clearly implicated as causative factors. Thermal ablation in the setting of little or no flow may increase arthroscopic fluid temperatures above 50°C to 55°C and result in cartilage cell death.[43] Routine local anesthetics (e.g., bupivacaine and lidocaine), which have been used for years as single injections in and around joints, have been shown to be cytotoxic to chondrocytes in several studies.[44,45] There are likely to be dose- and time-dependent effects with these medications, and it is currently our strong recommendation that intra-articular administration through pain pumps be avoided.

Postoperative glenohumeral sepsis can also be devastating to articular cartilage. The outcomes of surgery for septic arthritis are related to timely management, the presence of irreparable rotator cuff tears, and the extent of cartilage loss.[46]

The risk of cartilage damage due to intra-articular corticosteroid injections remains controversial. Beneficial results from suppression of local joint inflammation are mostly transient. We recommend judicious and limited use of intra-articular steroids. Animal studies have shown that corticosteroids alter tendon structure and weaken collagen, but there are few if any clinical data to support disease progression or chondral injury.[47]

Finally, mechanical factors, such as malpositioned or proud anchors after labral repair or perforating screws after osteosynthesis of the humeral head, may result in rapid destruction of the chondral surfaces.[48]

CLINICAL EVALUATION

Symptomatic cartilage lesions rarely appear in isolation, and thus concomitant conditions must be carefully and systematically addressed. The clinical presentation of focal chondral lesions may closely resemble findings of impingement syndromes.[7] A precise history and clinical examination, especially in young and active patients, remain pivotal in arriving at an accurate diagnosis.

Focal cartilage lesions should be considered in the setting of

- Previous trauma or shoulder surgery
- Recent or recurrent shoulder dislocations or subluxations
- Mechanical symptoms (clicking or catching)
- Discomfort, pain, or interrupted sleep
- Weakness or loss of range of motion

As part of a comprehensive shoulder examination, the compression-rotation test[7] may help in differentiating between subacromial impingement and chondral lesions. The patient actively internally and externally rotates the arm under axial compression of the glenohumeral joint. Pain in the mid ranges of motion has been shown to be both sensitive and specific for cartilage lesions in the shoulder.[7] Subacromial injection can make this test more specific by eliminating pain provocation from subacromial impingement. Crepitation or mechanical symptoms during active and passive range of motion may also arise from chondral lesions.

Diagnostic tools

Standard radiographs of the shoulder will easily detect large osteochondral defects of the humeral head or glenoid and show pathologic subluxation or locked dislocations. Specific views such as the Stryker notch or axillary view offer the best visualization of Hill-Sachs lesions. Computed tomography with 3-dimensional reconstruction will detect even subtle osteochondral defects. MRI remains the gold standard in diagnosing focal chondral lesions, although sensitivity of standard MRI is affected by the limited cartilage thickness of only 1.0 to 1.3 mm of the humeral head.[27] Magnetic resonance arthrography has been shown to be significantly more accurate, correctly identifying cartilage defects in over 70% of patients in 1 study with follow-up arthroscopy.[49] New high-field MRI at 3.0 T, capable of cartilage matrix assessment with quantitative delayed gadolinium-enhanced MRI of cartilage and T2 and T1 rho mapping techniques, may prove capable of detecting low-grade, partial-thickness lesions with unprecedented sensitivity.[50,51] We recommend MRI for preoperative planning, particularly in assessing the size and depth of focal lesions.

Classification of cartilage lesions

There are several classification systems for cartilage damage in current use, and most are based on arthroscopic findings. No recognized classification has been developed from standardized MRI alone, and radiologists typically use the Outerbridge classification.[52] This lacks precision in differentiating between early grade I and II changes, and measures of the depth of grade III lesions are not reliable.

The International Cartilage Repair Society (ICRS) expanded the classic Outerbridge grading to the ICRS Hyaline Cartilage Lesion Classification System published in 2003, which—though developed for the knee—is now considered the international standard.[53] The ICRS classification is based on the arthroscopic evaluation of the depth and expanse of the debrided defect (Table 3). A comprehensive arthroscopic assessment of chondral lesions requires debridement of unstable cartilage flaps and the frayed borders of lesions.

NONOPERATIVE TREATMENT

Treatment demands clear communication between surgeon and patient. A clear assessment of the patient's goals, expectations, and activity level should be complemented with an honest description of the strengths and weaknesses of all management options. Older patients, averse to surgery or at high operative risk because of concomitant diseases, may be better suited to nonoperative treatment that provides pain relief and maintains acceptable shoulder function. In such cases the surgeon should prescribe and monitor physical therapy to maintain or improve strength, motion, and function. The judicious use of intra-articular corticosteroid injections and topical or oral nonsteroidal anti-inflammatory drugs are mainstays of nonoperative treatment, although their true efficacy is unknown. Glenohumeral viscosupplementation remains off label for the shoulder but certainly is worth further study.[54,55]

OPERATIVE TREATMENT

Operative treatment has been classified as palliative, reparative, restorative, and reconstructive.[4] Many factors influence surgical decision making (Fig 2). The patient's age, activity level, and willingness to comply with postoperative rehabilitation should be considered. Other important factors include the size and depth of the lesion; unipolar versus bipolar, or "kissing," lesions; loss of structural integrity because of large bone defects; and concomitant injury patterns.

Focal full-thickness articular cartilage lesions

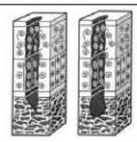
Focal full-thickness articular cartilage lesions of the shoulder are common and may progress to degenerative osteoarthritis. Especially in young patients, symptomatic, focal full-thickness cartilage lesions should be treated surgically. Treatment options are as follows:

- Debridement
- Microfracture
- Autologous osteochondral transfers (OATS [Osteochondral Autograft Transfer System]; Arthrex, Naples, FL)
- ACI/matrix-induced autologous chondrocyte implantation (MACI)

Debridement

For small lesions, simple debridement may suffice. Debridement acts to eliminate mechanical symptoms that are caused by edge instability and tends to stabilize the lesion, preventing delamination. For lesions that are at the periphery of the glenoid where loading may be critical or for small lesions, this may be adequate treatment. When a small GLAD lesion is encountered in the setting of a patient undergoing

TABLE 3. *ICRS and Outerbridge Cartilage Injury Classification*

Outerbridge	*ICRS Grade*	*ICRS Description*	Image
	0	Normal cartilage	
I	1 a	Cartilage softening, superficial fibrillation	
	1 b	Superficial lacerations and fissures	
II	2	Cartilage defect involves <50% of cartilage thickness	
III	3 a	Cartilage defect involves >50% of cartilage thickness	
	3 b	>50% of cartilage thickness with extend down to calcified cartilage layer	
	3 c	>50% of cartilage thickness with extend down to but not through the subchondral bone plate	
	3 d	>50% of cartilage thickness with blistering	
IV	4 a/ b	Cartilage defects which extend into the subchondral bone	 a b

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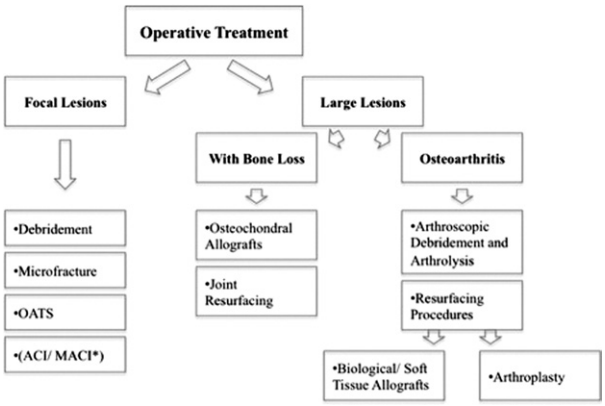

FIGURE 2. Flowchart of operative treatment for chondral lesions of humeral head. The asterisk indicates that more data are needed.

surgery for instability, 1 option is to simply debride the unstable articular flap and then advance the labrum into the defect when performing the labral repair (Fig 1). This effectively covers the defect and protects the underlying bone. When diffuse cartilage loss exists, such as in the setting of osteoarthritis, debridement may be undertaken to stabilize the remaining articular surface and to eliminate joint irritation and synovitis from cartilage detritus.

Microfracture

Steadman et al.[56] popularized the microfracture technique in the knee, and we have applied it effectively to treat chondral injuries in the shoulder.[57] The great advantage of microfracture in shoulder surgery is that it can be performed arthroscopically in 1 setting (Figs 1 and 3). Key technical points include adequate debridement of the calcified chondral layer (until punctate bleeding is observed) and proper placement of the awl holes perpendicular to the subchondral plate at 2- to 3-mm intervals. Protected loading conditions and motion are important for healing.[4] Cells proliferate and differentiate into a functional fibrocartilage repair tissue.[58,59]

In the series reported by Millett et al.[57] with 25 shoulders in 24 patients treated by arthroscopic microfracture, there was significant pain reduction and improved shoulder function. After a mean follow-up of 47 months, mean pain scores decreased from 3.8 to 1.6 postoperatively (0, no pain; 10, worst pain) and mean American Shoulder and Elbow Surgeons scores improved significantly from 60 (range, 22 to 80) preoperatively to 80 (range, 45 to 100) postoperatively ($P < .05$). We also found significant improvements postoperatively ($P < .05$) in patients' ability to work, activities of daily living, and sports activity. Overall, the greatest improvements were seen in patients who had microfracture of isolated lesions of the humerus.

In the knee microfracture yields comparable results to other techniques. A randomized trial comparing ACI with microfracture in the knee showed no differences at 5 years' follow-up,[60] whereas another study showed comparable clinical outcomes but histologically superior tissue regenerate in the ACI group.[61]

A prospective study in 5 patients evaluating the combination of microfracture and periosteal flap in focal chondral lesions in the glenohumeral joint showed a significant improvement in the Constant score from 43.4 preoperatively to 81.8 and significantly less pain at a mean follow-up of at least 24 months.[62]

OATS

The results of OATS for high-grade humeral head and glenoid lesions have been reported in a small series of patients.[63] The study showed a significant Constant score improvement from 73.9 to 88.7 after a mean of 32.6 months, but the development of osteoarthritis and the progression of pre-existing osteoarthritic changes were not altered. Although OATS and ACI have been studied extensively in the knee show-

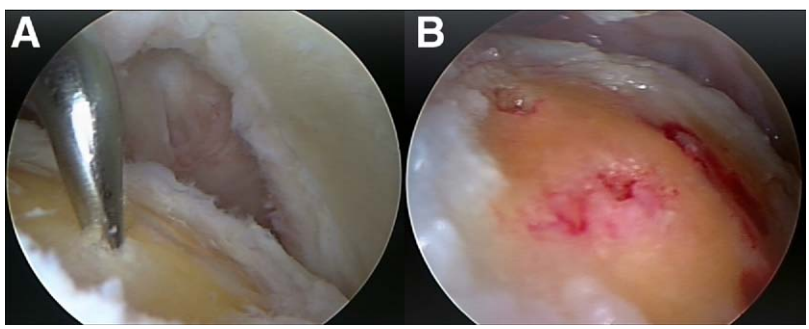

FIGURE 3. Arthroscopic microfracture humeral head. (A) Awl penetrating subchondral bone. (B) Punctual bleeding after microfracture.

ing their efficacy,[64,65] similar trials are needed to determine the relative success of these techniques in the shoulder.

ACI and MACI

ACI in the shoulder has only been reported in a single case study.[66] Outcome studies of full-thickness cartilage defects of the knee treated by ACI show good to excellent results, even at long-term follow-up.[67] Many surgeons continue to prefer alternate techniques that do not require the staged approach and additional expense of ACI.

MACI, a collagen bio-scaffold that delivers in vitro cultured autologous chondrocytes into the defect, is a more recent innovation.[68] Studies on MACI showed 75% hyaline-like cartilage regeneration after 6 months and good to excellent outcomes.[69] MACI might show promise for the glenohumeral joint, because it is a stable cell-based delivery system that enables the regeneration of hyaline-like cartilage in a high percentage.

Large full-thickness articular cartilage lesions

Lesions With Significant Bone Loss or Partial Humeral Head Necrosis

Large full-thickness articular cartilage lesions and lesions with significant bone loss and partial humeral head necrosis can be treated by osteochondral allografts or resurfacing procedures.

Osteochondral Allografts: Osteochondral allografts do not impair the overall results for the patient with donor-site morbidity, and thus there is no limitation to size. However, there are also potential disadvantages, such as limited chondrocyte viability, loss of matrix viability as shown by cationic staining, immunogenicity, and possible disease transmission.[70] Studies have shown better outcomes in traumatic osteochondral lesions and younger patients (<50 years) with success rates between 75% and 85% at a mean of 3.8 to 7.5 years' follow-up.[71,72] Rigid fixation of host bone to graft bone is important for long-term allograft survival.[70]

The use of osteochondral allografts in shoulder surgery is not common. Allografts have been used to fill engaging Hill-Sachs lesions,[73] as well as defects from proximal humeral bone tumor resections, with good results.[74] There is limited experience with the use of allografts for treatment of OCD of the glenohumeral joint.[36] Osteochondral allografts are a promising alternative to resurfacing procedures, particularly in young, athletic patients (Fig 4).

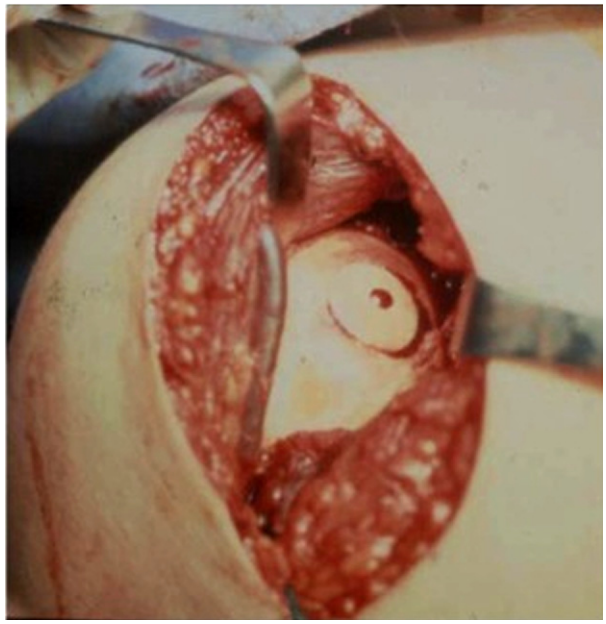

FIGURE 4. Allograft reconstruction of humeral head. (Courtesy of Jon J. P. Warner, M.D.)

Joint Resurfacing: Recently, new partial replacements and stem-less implants for shoulder arthroplasty have been developed that are particularly attractive for use in young patients. These implants preserve anatomy and leave open various options for subsequent revision surgery (Fig 5). In contrast to older prostheses, the new implants provide the ability to adjust them to the cortical rim of the resected humeral head (i.e., reconstruction of the humeral offset) and offer a variety of anatomic head sizes.[75] Unfortunately, outcomes analyses are still forthcoming.

Osteoarthritis

Palliative Procedures: Palliative procedures, such as arthroscopic debridement[31,76] and arthroscopic capsular release, can be performed in cases of osteoarthritis, especially in younger patients to delay more invasive surgery. The goals of the procedure are to diminish pain and increase motion, by reducing joint contact pressures and impingement from engaging osteophytes.[76,77] At our institution, 27 young patients, with a minimum 1-year follow-up, have been treated with a surgical procedure that we have called the CAM procedure—an acronym for "comprehensive arthroscopic management" of shoulder arthritis that includes an extensive debridement, capsular release, and humeral osteoplasty (Fig 6). At short-term follow-up, the mean patient satisfaction rate with the procedure was

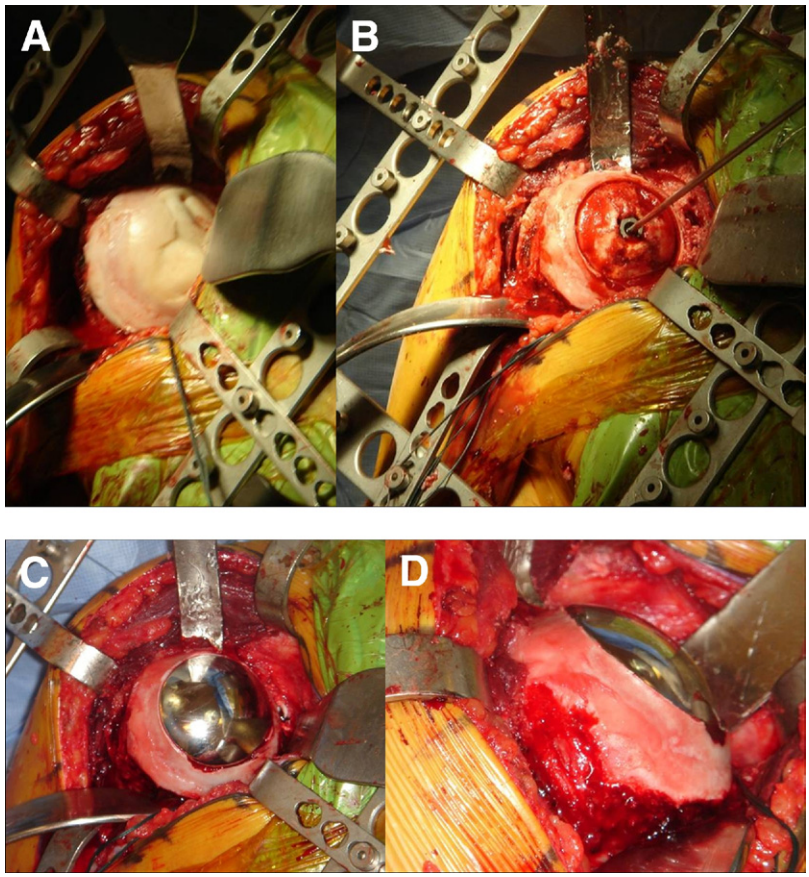

Figure 5. Partial prosthetic head replacement for focal osteoarticular defect of the humeral head from post-traumatic osteonecrosis in a young patient. (A) The left picture shows a large focal osteoarticular defect of the humeral head, (B) after preparation of the bone bed and insertion of the fixation screw; (C) status post implantation of the partial humeral head prosthesis; (D) on this coronal section notice how the radius of curvature has been restored. This is important to prevent erosion of the adjacent humeral articular cartilage and the articulating glenoid articular surface.

high and averaged 8.5 of 10 points, and only 1 patient has progressed to an arthroplasty (unpublished data, F.E., M.P.H., and P.J.M., October 2009).[78] The long-term durability is still unknown, but this approach does not preclude total shoulder arthroplasty.

Resurfacing Procedures: Diffuse, advanced osteoarthritic changes of the glenohumeral joint in younger, active patients are difficult to manage. Ideally, hemiarthroplasty or total shoulder replacement should be deferred until the patient is older and less active to achieve longer durability of the implant and minimize the necessity of future revision surgery. On the other hand, the results of total shoulder arthroplasty are substantially better with an intact rotator

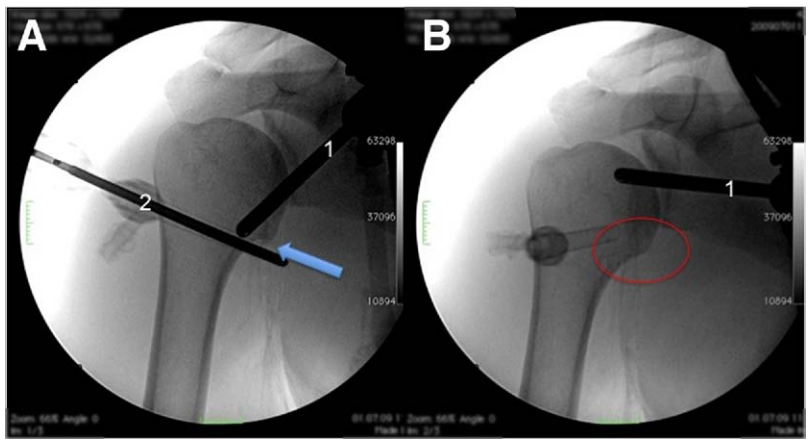

Figure 6. Comprehensive arthroscopic management (CAM) for glenohumeral osteoarthritis. (A) Resection of inferior spur of humeral head (arrow). (B) After resection (circle). (1, arthroscope; 2, shaver.)

cuff and good bone stock of the glenoid. New stemless implants for shoulder arthroplasty are promising options if palliative procedures fail. Results after conventional arthroplasty/hemiarthroplasty in younger patients performed for the treatment of osteoarthritis after failed surgical stabilization procedures are relatively poor.[3] Arthroplasty or hemiarthroplasty provided satisfactory pain relief and improved motion, but these procedures were associated with high rates of revision surgery because of component failure, recurrent instability, and pain.[3]

Biologic Glenoid Resurfacing/Interposition Arthroplasty: Alternative approaches to address unipolar glenoid arthritis have been proposed. Krishnan et al.[79] recently published a prospective study on biologic glenoid resurfacing using anterior capsule (7 shoulders), autogenous fascia lata (11 shoulders), and Achilles tendon allograft (18 shoulders). The early results were comparable to the results of total shoulder arthroplasty without the inherent risks of the nonbiological replacements. Achilles tendon allograft has evolved as a preferred resurfacing material for the glenoid.[79] Other authors, however, have not been able to reproduce these results.

The use of meniscal allografts to resurface the glenoid in conjunction with hemiarthroplasty has also been proposed. Nicholson et al.[80] published a series of 30 young patients (mean age, 42 years) using lateral meniscus allograft for glenoid resurfacing during hemiarthroplasty. Their overall results were good, documenting significant improvements in outcome scores, but they found a moderate complication rate of 17% requiring reoperation. Longer-term data, however, have shown deterioration of the procedure, and the initial enthusiasm for this technique seems to have faded somewhat.

Arthroplasty: For older patients with severe symptomatic osteoarthritis, total shoulder arthroplasty remains the gold standard. The number of shoulder replacement surgeries performed in the United States doubled from 10,000 to 20,000 per year over the last decade,[81] and it is now estimated to be closer to 50,000 per year. There is a clear correlation between the surgeon's volume and the outcome after surgery.[81] Mortality rates, rates of postoperative complications, and inpatient days have been shown to be significantly higher for operations that are performed by surgeons performing fewer than 5 procedures per year.[81] There is a role for shoulder replacement surgery in the treatment of young, active patients with diffuse cartilage loss, but given the high rates of revision surgery, non-arthroplasty options should be carefully considered and used if appropriate.

TABLE 4. *Key Points and Current Concepts in Treating Cartilage Lesions in Young, Active Patients*

At the center of the humeral head, the hyaline cartilage thickness is 1.2 to 1.3 mm, thinning at the periphery to 1.0 mm.
Chronic instability has been clearly linked to early glenohumeral osteoarthritis.
Active patients and athletes are at higher risk for traumatic cartilage lesions.
Given the diminished weight-bearing role of the shoulder, many cases of glenohumeral cartilage defects are not detected until advanced stages occur.
Intra-articular administration of local anesthetics (bupivacaine, lidocaine) through pain pumps should be avoided because of the risk of chondrolysis.
Early treatment of cartilage lesions is recommended in young, active patients to avoid the premature onset of osteoarthritis.
Innovative strategies for the treatment of cartilage lesions such as ACI/MACI, OATS, and microfracture show excellent promise, but they must still be established as effective techniques in the shoulder.
Microfracture has the best evidence for treating focal cartilage lesions in the glenohumeral joint to date. Furthermore, it is cost-effective and can be performed arthroscopically in 1 setting.

CONCLUSIONS

The key points of this article are summarized in Table 4. Evolving innovative strategies for the treatment of cartilage lesions in the knee joint have been followed by good short- to intermediate-term results but are yet to be established as effective techniques for similar lesions in the shoulder. Investigators must continue to evaluate the safety and efficacy of new cartilage repair and restorative strategies in the shoulder. There is a preponderance of evidence to support the early treatment of cartilage lesions of the glenohumeral joint in young, active patients to avoid the premature onset of osteoarthritis. Surgeons should maintain a heightened awareness of chondral injuries in the glenohumeral joint and should be more aggressive in early diagnostic and therapeutic interventions. There is growing evidence to support the claim that the progression of chondral injury may be slowed if the time between injury and surgery is minimized in patients with traumatic, recurrent shoulder instability. Although the next decade is certain to bring exciting new technologies to bear on the treatment of focal and diffuse cartilage injury, successful intervention will still depend on the sensitive diagnostic skills and sound, principled decision making of the shoulder surgeon.

REFERENCES

1. Gartsman GM, Taverna E. The incidence of glenohumeral joint abnormalities associated with full-thickness, reparable rotator cuff tears. *Arthroscopy* 1997;13:450-455.
2. Paley KJ, Jobe FW, Pink MM, Kvitne RS, ElAttrache NS. Arthroscopic findings in the overhand throwing athlete: Evidence for posterior internal impingement of the rotator cuff. *Arthroscopy* 2000;16:35-40.
3. Sperling JW, Antuna SA, Sanchez-Sotelo J, Schleck C, Cofield RH. Shoulder arthroplasty for arthritis after instability surgery. *J Bone Joint Surg Am* 2002;84:1775-1781.
4. Cole BJ, Yanke A, Provencher MT. Nonarthroplasty alternatives for the treatment of glenohumeral arthritis. *J Shoulder Elbow Surg* 2007;16:S231-S240.
5. Müller-Gerbl M, Putz R, Kenn R. Distribution pattern of subchondral mineralization in the glenoid cavity in normal subjects, athletes and patients. *Z Orthop Ihre Grenzgeb* 1993; 131:10-13 (in German).
6. Soslowsky LJ, Flatow EL, Bigliani LU, Mow VC. Articular geometry of the glenohumeral joint. *Clin Orthop Relat Res* 1992:181-190.
7. Ellman H, Harris E, Kay SP. Early degenerative joint disease simulating impingement syndrome: Arthroscopic findings. *Arthroscopy* 1992;8:482-487.
8. Guntern DV, Pfirrmann CW, Schmid MR, et al. Articular cartilage lesions of the glenohumeral joint: Diagnostic effectiveness of MR arthrography and prevalence in patients with subacromial impingement syndrome. *Radiology* 2003;226: 165-170.
9. Hermodsson I. Rontgenologische Studien Uber Die Traumatischen und Habituellen Schultergelenkverrenkungen Nach Vorn Und Nach Unten. *Acta Radiol* 1934;20:1-173.
10. Hill HA, Sachs MD. The grooved defect of the humeral head. A frequently unrecognized complication of dislocations of the shoulder joint. *Radiology* 1940;35:690-700.
11. Calandra JJ, Baker CL, Uribe J. The incidence of Hill-Sachs lesions in initial anterior shoulder dislocations. *Arthroscopy* 1989;5:254-257.
12. Taylor DC, Arciero RA. Pathologic changes associated with shoulder dislocations. Arthroscopic and physical examination findings in first-time, traumatic anterior dislocations. *Am J Sports Med* 1997;25:306-311.
13. Norlin R. Intraarticular pathology in acute, first-time anterior shoulder dislocation: An arthroscopic study. *Arthroscopy* 1993;9:546-549.
14. Amrami KK, Sperling JW, Bartholmai BJ, Sundaram M. Radiologic case study. Glenolabral articular disruption (GLAD) lesion. *Orthopedics* 2002;25:29, 95-96.
15. Neviaser TJ. The GLAD lesion: Another cause of anterior shoulder pain. *Arthroscopy* 1993;9:22-23.
16. Buscayret F, Edwards TB, Szabo I, Adeleine P, Coudane H, Walch G. Glenohumeral arthrosis in anterior instability before and after surgical treatment: Incidence and contributing factors. *Am J Sports Med* 2004;32:1165-1172.
17. McCarty LP III, Cole BJ. Nonarthroplasty treatment of glenohumeral cartilage lesions. *Arthroscopy* 2005;21:1131-1142.
18. Marx RG, McCarty EC, Montemurno TD, Altchek DW, Craig EV, Warren RF. Development of arthrosis following dislocation of the shoulder: A case-control study. *J Shoulder Elbow Surg* 2002;11:1-5.
19. Samilson RL, Prieto V. Dislocation arthropathy of the shoulder. *J Bone Joint Surg Am* 1983;65:456-460.
20. Cameron ML, Kocher MS, Briggs KK, Horan MP, Hawkins RJ. The prevalence of glenohumeral osteoarthrosis in unstable shoulders. *Am J Sports Med* 2003;31:53-55.
21. Brophy RH, Marx RG. Osteoarthritis following shoulder instability. *Clin Sports Med* 2005;24:47-56.
22. Hawkins RJ, Angelo RL. Glenohumeral osteoarthrosis. A late complication of the Putti-Platt repair. *J Bone Joint Surg Am* 1990;72:1193-1197.
23. O'Driscoll SW, Evans DC. Long-term results of staple capsulorrhaphy for anterior instability of the shoulder. *J Bone Joint Surg Am* 1993;75:249-258.
24. Singer GC, Kirkland PM, Emery RJ. Coracoid transposition for recurrent anterior instability of the shoulder. A 20-year follow-up study. *J Bone Joint Surg Br* 1995;77:73-76.
25. Borrelli J Jr, Silva MJ, Zaegel MA, Franz C, Sandell LJ. Single high-energy impact load causes posttraumatic OA in young rabbits via a decrease in cellular metabolism. *J Orthop Res* 2009;27:347-352.
26. Jeon IH, Wallace WA. Traumatic humeral articular cartilage shear (THACS) lesion in a professional rugby player: A case report. *Br J Sports Med* 2004;38:E12.
27. Carroll KW, Helms CA, Speer KP. Focal articular cartilage lesions of the superior humeral head: MR imaging findings in seven patients. *AJR Am J Roentgenol* 2001;176:393-397.
28. Ruckstuhl H, de Bruin ED, Stussi E, Vanwanseele B. Posttraumatic glenohumeral cartilage lesions: A systematic review. *BMC Musculoskelet Disord* 2008;9:107.
29. Cruess RL. Experience with steroid-induced avascular necrosis of the shoulder and etiologic considerations regarding osteonecrosis of the hip. *Clin Orthop Relat Res* 1978:86-93.
30. Mont MA, Maar DC, Urquhart MW, Lennox D, Hungerford DS. Avascular necrosis of the humeral head treated by core decompression. A retrospective review. *J Bone Joint Surg Br* 1993;75:785-788.
31. Weinstein DM, Bucchieri JS, Pollock RG, Flatow EL, Bigliani LU. Arthroscopic debridement of the shoulder for osteoarthritis. *Arthroscopy* 2000;16:471-476.
32. Hamada S, Hamada M, Nishiue S, Doi T. Osteochondritis dissecans of the humeral head. *Arthroscopy* 1992;8:132-137.
33. Anderson WJ, Guilford WB. Osteochondritis dissecans of the humeral head. An unusual cause of shoulder pain. *Clin Orthop Relat Res* 1983:166-168.
34. Ishikawa H, Ueba Y, Yonezawa T, Kurosaka M, Ohno O, Hirohata K. Osteochondritis dissecans of the shoulder in a tennis player. *Am J Sports Med* 1988;16:547-550.
35. Park TS, Kim TS, Cho JH. Arthroscopic osteochondral autograft transfer in the treatment of an osteochondral defect of the humeral head: Report of one case. *J Shoulder Elbow Surg* 2006;15:e31-e36.
36. Johnson DL, Warner JJ. Osteochondritis dissecans of the humeral head: Treatment with a matched osteochondral allograft. *J Shoulder Elbow Surg* 1997;6:160-163.
37. Neer CS II, Craig EV, Fukuda H. Cuff-tear arthropathy. *J Bone Joint Surg Am* 1983;65:1232-1244.
38. Peach CA, Zhang Y, Dunford JE, Brown MA, Carr AJ. Cuff tear arthropathy: Evidence of functional variation in pyrophosphate metabolism genes. *Clin Orthop Relat Res* 2007;462: 67-72.
39. Busfield BT, Romero DM. Pain pump use after shoulder arthroscopy as a cause of glenohumeral chondrolysis. *Arthroscopy* 2009;25:647-652.
40. Nakagawa Y, Ueo T, Miki T, Kotani H, Onishi E, Nakamura T. Glenohumeral osteoarthritis following a "color test" during rotator cuff repair. A case report and a review of the literature. *Bull Hosp Jt Dis* 1998;57:216-218.
41. Petty DH, Jazrawi LM, Estrada LS, Andrews JR. Glenohumeral chondrolysis after shoulder arthroscopy: Case reports and review of the literature. *Am J Sports Med* 2004;32:509-515.
42. Levine WN, Clark AM Jr, D'Alessandro DF, Yamaguchi K. Chondrolysis following arthroscopic thermal capsulorrhaphy to treat shoulder instability. A report of two cases. *J Bone Joint Surg Am* 2005;87:616-621.

43. Zoric B, Horn N, Braun S, Millett PJ. Factors influencing intra-articular fluid temperature profiles with radiofrequency ablation. *J Bone Joint Surg Am* 2009;91:2448-2454.
44. Karpie JC, Chu CR. Lidocaine exhibits dose- and time-dependent cytotoxic effects on bovine articular chondrocytes in vitro. *Am J Sports Med* 2007;35:1621-1627.
45. Chu CR, Izzo NJ, Coyle CH, Papas NE, Logar A. The in vitro effects of bupivacaine on articular chondrocytes. *J Bone Joint Surg Br* 2008;90:814-820.
46. Duncan SF, Sperling JW. Treatment of primary isolated shoulder sepsis in the adult patient. *Clin Orthop Relat Res* 2008; 466:1392-1396.
47. Wei AS, Callaci JJ, Juknelis D, et al. The effect of corticosteroid on collagen expression in injured rotator cuff tendon. *J Bone Joint Surg Am* 2006;88:1331-1338.
48. Millett PJ, Clavert P, Warner JJ. Open operative treatment for anterior shoulder instability: When and why? *J Bone Joint Surg Am* 2005;87:419-432.
49. Millett PJ, Neumann G, Yoshioka CS, Winalski CS, Carrino J, Lang P. Articular cartilage in the shoulder: Correlation of MR arthrography and surgical arthroscopy. Radiological Society of North America. Presented at the 90th Scientific Assembly and Annual Meeting, Chicago, Illinois, November 2004. Available at: http://rsna2004.rsna.org/rsna2004/V2004/conference/event_display.cfm?em_id=4409527. Accessed May 6, 2008.
50. Gold GE, Reeder SB, Beaulieu CF. Advanced MR imaging of the shoulder: Dedicated cartilage techniques. *Magn Reson Imaging Clin N Am* 2004;12:143-159, vii.
51. Link TM, Stahl R, Woertler K. Cartilage imaging: Motivation, techniques, current and future significance. *Eur Radiol* 2007; 17:1135-1146.
52. Outerbridge RE. The etiology of chondromalacia patellae. *J Bone Joint Surg Br* 1961;43:752-757.
53. Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am* 2003;85:58-69 (Suppl 2).
54. Blaine T, Moskowitz R, Udell J, et al. Treatment of persistent shoulder pain with sodium hyaluronate: A randomized, controlled trial. A multicenter study. *J Bone Joint Surg Am* 2008; 90:970-979.
55. Silverstein E, Leger R, Shea KP. The use of intra-articular hylan G-F 20 in the treatment of symptomatic osteoarthritis of the shoulder: A preliminary study. *Am J Sports Med* 2007;35: 979-985.
56. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: Surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res* 2001:S362-S369.
57. Millett PJ, Huffard BH, Horan MP, Hawkins RJ, Steadman JR. Outcomes of full-thickness articular cartilage injuries of the shoulder treated with microfracture. *Arthroscopy* 2009;25:856-863.
58. Shapiro F, Koide S, Glimcher MJ. Cell origin and differentiation in the repair of full-thickness defects of articular cartilage. *J Bone Joint Surg Am* 1993;75:532-553.
59. Alford JW, Cole BJ. Cartilage restoration, part 2: Techniques, outcomes, and future directions. *Am J Sports Med* 2005;33: 443-460.
60. Knutsen G, Drogset JO, Engebretsen L, et al. A randomized trial comparing autologous chondrocyte implantation with microfracture. Findings at five years. *J Bone Joint Surg Am* 2007;89:2105-2112.
61. Saris DB, Vanlauwe J, Victor J, et al. Characterized chondrocyte implantation results in better structural repair when treating symptomatic cartilage defects of the knee in a randomized controlled trial versus microfracture. *Am J Sports Med* 2008; 36:235-246.
62. Siebold R, Lichtenberg S, Habermeyer P. Combination of microfracture and periostal-flap for the treatment of focal full thickness articular cartilage lesions of the shoulder: A prospective study. *Knee Surg Sports Traumatol Arthrosc* 2003;11: 183-189.
63. Scheibel M, Bartl C, Magosch P, Lichtenberg S, Habermeyer P. Osteochondral autologous transplantation for the treatment of full-thickness articular cartilage defects of the shoulder. *J Bone Joint Surg Br* 2004;86:991-997.
64. Hangody L, Füles P. Autologous osteochondral mosaicplasty for the treatment of full-thickness defects of weight-bearing joints: Ten years of experimental and clinical experience. *J Bone Joint Surg Am* 2003;85:25-32 (Suppl 2).
65. Bentley G, Biant LC, Carrington RW, et al. A prospective, randomised comparison of autologous chondrocyte implantation versus mosaicplasty for osteochondral defects in the knee. *J Bone Joint Surg Br* 2003;85:223-230.
66. Romeo AA, Cole BJ, Mazzocca AD, Fox JA, Freeman KB, Joy E. Autologous chondrocyte repair of an articular defect in the humeral head. *Arthroscopy* 2002;18:925-929.
67. Peterson L, Brittberg M, Kiviranta I, Akerlund EL, Lindahl A. Autologous chondrocyte transplantation. Biomechanics and long-term durability. *Am J Sports Med* 2002;30:2-12.
68. Cherubino P, Grassi FA, Bulgheroni P, Ronga M. Autologous chondrocyte implantation using a bilayer collagen membrane: A preliminary report. *J Orthop Surg (Hong Kong)* 2003;11: 10-15.
69. Zheng MH, Willers C, Kirilak L, et al. Matrix-induced autologous chondrocyte implantation (MACI): Biological and histological assessment. *Tissue Eng* 2007;13:737-746.
70. Gross AE, Kim W, Las Heras F, Backstein D, Safir O, Pritzker KP. Fresh osteochondral allografts for posttraumatic knee defects: Long-term followup. *Clin Orthop Relat Res* 2008;466: 1863-1870.
71. McDermott AG, Langer F, Pritzker KP, Gross AE. Fresh small-fragment osteochondral allografts. Long-term follow-up study on first 100 cases. *Clin Orthop Relat Res* 1985:96-102.
72. Ghazavi MT, Pritzker KP, Davis AM, Gross AE. Fresh osteochondral allografts for post-traumatic osteochondral defects of the knee. *J Bone Joint Surg Br* 1997;79:1008-1013.
73. Chapovsky F, Kelly JD IV. Osteochondral allograft transplantation for treatment of glenohumeral instability. *Arthroscopy* 2005;21:1007.
74. DeGroot H, Donati D, Di Liddo M, Gozzi E, Mercuri M. The use of cement in osteoarticular allografts for proximal humeral bone tumors. *Clin Orthop Relat Res* 2004:190-197.
75. Jensen KL. Humeral resurfacing arthroplasty: Rationale, indications, technique, and results. *Am J Orthop* 2007;36:4-8.
76. Richards DP, Burkhart SS. Arthroscopic debridement and capsular release for glenohumeral osteoarthritis. *Arthroscopy* 2007;23:1019-1022.
77. Millett PJ, Gobezie R, Boykin RE. Shoulder osteoarthritis: Diagnosis and management. *Am Fam Physician* 2008;78:605-611.
78. Elser F, Horan MP, Millett PJ. Comprehensive arthroscopic management (CAM) of shoulder osteoarthritis in young active patients. Accepted for presentation by E-poster at the 2010 Arthroscopy Association of North America Annual Meeting, Hollywood, Florida, May 20-23, 2010.
79. Krishnan SG, Nowinski RJ, Harrison D, Burkhead WZ. Humeral hemiarthroplasty with biologic resurfacing of the glenoid for glenohumeral arthritis. Two to fifteen-year outcomes. *J Bone Joint Surg Am* 2007;89:727-734.
80. Nicholson GP, Goldstein JL, Romeo AA, et al. Lateral meniscus allograft biologic glenoid arthroplasty in total shoulder arthroplasty for young shoulders with degenerative joint disease. *J Shoulder Elbow Surg* 2007;16:S261-S266.
81. Jain N, Pietrobon R, Hocker S, Guller U, Shankar A, Higgins LD. The relationship between surgeon and hospital volume and outcomes for shoulder arthroplasty. *J Bone Joint Surg Am* 2004;86:496-505.