

The American Journal of Sports Medicine

<http://ajs.sagepub.com/>

Clinical Outcomes After Microfracture of the Glenohumeral Joint

Rachel M. Frank, Geoffrey S. Van Thiel, Mark A. Slabaugh, Anthony A. Romeo, Brian J. Cole and Nikhil N. Verma
Am J Sports Med 2010 38: 772 originally published online January 21, 2010
DOI: 10.1177/0363546509350304

The online version of this article can be found at:

<http://ajs.sagepub.com/content/38/4/772>

Published by:



<http://www.sagepublications.com>

On behalf of:



American Orthopaedic Society for Sports Medicine

Additional services and information for *The American Journal of Sports Medicine* can be found at:

Email Alerts: <http://ajs.sagepub.com/cgi/alerts>

Subscriptions: <http://ajs.sagepub.com/subscriptions>

Reprints: <http://www.sagepub.com/journalsReprints.nav>

Permissions: <http://www.sagepub.com/journalsPermissions.nav>

Clinical Outcomes After Microfracture of the Glenohumeral Joint

Rachel M. Frank, Geoffrey S. Van Thiel, MD, MBA, Mark A. Slabaugh, MD, Maj USAF, Anthony A. Romeo, MD, Brian J. Cole, MD, MBA, and Nikhil N. Verma,* MD

From the Division of Sports Medicine, Department of Orthopaedic Surgery, Rush University Medical Center, Chicago, Illinois

Background: Microfracture is an effective surgical treatment for isolated, full-thickness cartilage defects with current data focused on applications in the knee. No studies describing clinical outcomes of patients who have undergone microfracture in the shoulder joint have been reported.

Hypothesis: Treatment of glenohumeral joint articular defects using microfracture would demonstrate similar short-term clinical outcomes when compared with other joints.

Study Design: Case series; Level of evidence, 4.

Methods: From March 2001 to August 2007, 16 patients (17 shoulders) who underwent arthroscopic microfracture of the humeral head and/or glenoid surface were retrospectively reviewed. All patients were examined by an independent, blinded examiner and completed surveys containing the Simple Shoulder Test (SST), American Shoulder and Elbow Score (ASES), and visual analog scale (VAS).

Results: Two patients were lost to follow-up, for a follow-up rate of 88%. Three patients went on to subsequent shoulder surgery and were considered to have failed results. The mean age was 37.0 years (range, 18-55 years) with an average follow-up of 27.8 months (range, 12.1-89.2 months). The average size of humeral and glenoid defects was 5.07 cm² (range, 1.0-7.84 cm²) and 1.66 cm² (range, 0.4-3.75 cm²), respectively. There was a statistically significant decrease from 5.6 ± 1.7 to 1.9 ± 1.4 ($P < .01$) in VAS after surgery as well as statistically significant improvements ($P < .01$) in SST (5.7 ± 2.1 to 10.3 ± 1.3) and ASES (44.3 ± 15.3 to 86.3 ± 10.5). Twelve (92.3%) patients claimed they would have the procedure again.

Conclusion: Microfracture of the glenohumeral joint provides a significant improvement in pain relief and shoulder function in patients with isolated, full-thickness chondral injuries. Longer term studies are required to determine if similar results are maintained over time.

Keywords: glenohumeral joint; microfracture; focal chondral defect; clinical outcomes

Chondral lesions of the glenohumeral joint, although less common than chondral lesions in other joints such as the knee or ankle, can be a source of shoulder pain in an active population. While the incidence of such defects of the shoulder has been documented as 5% to 17%, these reports do not differentiate which of these defects are the primary symptom generators and meet the indications for

treatment.^{14,20,30} The treatment options for chondral defects of the shoulder remain poorly defined. Often, the diagnosis of a symptomatic shoulder chondral defect is difficult to make secondary to vague nonlocalizing complaints, with history and examination findings similar to other common shoulder conditions. Additionally, imaging studies are poor at detecting chondral injuries until late in the disease process because of the relatively thin cartilage in the shoulder.¹⁴ Often, diagnosis may be delayed until the time of shoulder arthroscopy. While the cause of most cartilage defects in the shoulder is unknown, there has been a reported association with recurrent instability, rotator cuff tears, iatrogenic injury, and capsulorrhaphy techniques.^{9,11,20,24,27}

Once a glenohumeral chondral defect is identified, no consensus exists among orthopaedic surgeons on the most appropriate treatment options. Nonsurgical treatments include physical therapy and steroid injections, while surgical options incorporate palliative, reparative, restorative, and reconstructive techniques such as arthroplasty.^{3,14} While total

*Address correspondence to Nikhil N. Verma, MD, Rush University Medical Center, 1725 West Harrison Street, Suite 1063, Chicago, IL 60612 (e-mail: nverma@rushortho.com).

One or more authors has declared a potential conflict of interest: Nikhil N. Verma: Smith & Nephew (consultant, research support), Arthrex (research support), and Donjoy (research support); Anthony A. Romeo: Arthrex (royalties); Brian J. Cole: Arthrex, Stryker (royalties), Genzyme, Zimmer, DuPuy, Arthrex, Carticept, Regentis (consulting), and Arthrex, Zimmer, DuPuy (research support).

shoulder arthroplasty remains an excellent treatment option for older patients with diffuse symptomatic cartilage disease, in younger patients, this option is less attractive because of functional limitations and relatively short implant survival time in active patients. Further, arthroplasty may not be the preferred option for focal defects. The clinical outcomes of debridement and reconstructive and restorative techniques have been discussed in the literature; however, there remains a paucity of information regarding reparative surgical treatment for glenohumeral chondral defects, specifically microfracture.^{8,10,12,21,23,26,41}

Microfracture has been established as an effective therapeutic solution for full-thickness cartilage defects of the knee because of its low surgical morbidity and technical feasibility as a first-line treatment with good clinical outcomes.³⁴ As there is minimal vascular supply to the articular cartilage, defects of any origin rarely heal spontaneously and often require surgical intervention secondary to a high prevalence of clinical symptoms and functional disability.^{7,13,22,31,34,35,38} In addition to the lack of blood supply, the limited healing capacity of articular cartilage is due to the virtual absence of an undifferentiated cell population that is able to respond to traumatic and/or degenerative injury. While marrow stimulation has been shown to be effective in other joints such as the knee and ankle, we are aware of only one study describing the clinical outcomes of patients who have undergone open microfracture in the shoulder joint and no study reporting the outcomes of arthroscopic microfracture available in the literature.³² The purpose of this study is to report the short-term clinical outcomes of microfracture for symptomatic articular defects of the glenohumeral joint. The hypothesis was that treatment of articular defects in the glenohumeral joint using microfracture would demonstrate similar short-term clinical outcomes when compared with other joints.

MATERIALS AND METHODS

The medical records of all patients who underwent arthroscopy of the shoulder including debridement of the glenohumeral joint performed by 1 of 4 senior shoulder surgeons at our institution between March 1, 2001 and August 31, 2007 were retrospectively reviewed. As there is no CPT code for microfracture in the glenohumeral joint, debridement codes (29882 and 29883) have been used in patients on whom microfracture was performed. Operative reports were reviewed, and patients who had undergone microfracture of the glenoid and/or humeral head were identified. Inclusion criterion for participation in the study was follow-up greater than 1 year, and exclusion criteria were concomitant labral or rotator cuff repair. However, patients were not excluded for having other additional procedures during the arthroscopy, including subacromial decompression, distal clavicle excision, and biceps tenodesis. Patients were included regardless of if microfracture was performed as the intended primary surgery or as a procedure indicated at the time of arthroscopy. After the inclusion and exclusion criteria were met, 16 patients (17 shoulders) were deemed eligible for inclusion into the study.

The study was approved by our institutional review board, and all eligible patients were invited via telephone to participate in the study including follow-up clinical evaluation. Once contacted, each patient provided informed consent to participate in the study. At the follow-up evaluation, each patient completed shoulder surveys containing the Simple Shoulder Test (SST), American Shoulder and Elbow Score (ASES), visual analog score (VAS), and University of California–Los Angeles (UCLA) Shoulder Scale. Each survey also contained the Short Form-12 (SF12) health status questionnaire. Additionally, patients were asked to rate whether they would undergo the same procedure again (yes/no). Of the eligible patients, 13 patients completed the written surveys; however, only 8 were available for clinical evaluation. Shoulder examination included active and passive range of motion and shoulder muscle strength testing with an Isobex (Cursor, Bern, Switzerland) device. The physical examination was performed by a single orthopaedic research fellow, independent of the operating surgeon. Each patient had completed the same shoulder questionnaires and surveys preoperatively, allowing for comparisons between the preoperative and postoperative scores. In addition, all patients received a preoperative bilateral shoulder examination, including range of motion testing, again allowing for comparisons between the preoperative and postoperative values.

There were 17 shoulders in 16 patients included in the study; 1 patient had microfracture performed on both the left and right shoulder. Despite multiple attempts, 2 patients were unable to be reached and were lost to follow-up. Fourteen patients (15 shoulders) were included in the study, for a total follow-up rate of 88%.

Of the 14 patients remaining (15 shoulders), 3 patients underwent subsequent shoulder surgery and were considered to have failed results. Two of these patients were not included in the final statistical analysis because they were considered failures within 3 months of microfracture and because they were revised to alternative procedures before this study was completed; however, they were included in the overall failure calculation rate for the procedure. Of the remaining 12 patients (13 shoulders) participating in follow-up, the mean age at the time of surgery was 37 years (range, 18–55 years). The average time to follow-up was 27.8 months (range, 12.1 months to 7.4 years; SD, 20.7 months). There were 7 male and 5 female (6 shoulders) patients, and both of the failed patients were female. All patients, including the failures and those lost to follow-up, had a variety of symptoms, typically including pain during activity and limited range of motion; 6 patients reported a history of injury, 5 patients had persistent pain following a previous shoulder surgery, 2 patients (3 shoulders) had avascular necrosis associated with steroid use, and 3 patients had long-standing pain of insidious onset.

Surgery was performed on the right shoulder in 11 cases, and the dominant arm was involved in 9 cases (52.9%). Several patients underwent additional procedures at the time of microfracture, including subacromial decompression and biceps tenodesis. The characteristics of the study group are described in Table 1.

TABLE 1
Patient Characteristics^a

Age, y	Sex	Dominant Arm; Operative Arm	Length of Follow-up, mo	Preoperative Diagnosis	Defect Location	Defect Size, mm	Concomitant Procedure(s)	Preoperative Subjective Scores	Postoperative Subjective Scores	Preoperative ROM and Constant (strength)	Postoperative ROM and Constant (strength)	Previous Surgery	Subsequent Surgery (age, y)
48	M	L; R	13.7	Injury (work)	Glenoid	10 × 15	Capsular release, SAD, BT	VAS -4 SST -8 ASES -60	VAS -0 SST -12 ASES -100 SANE -95 UCLA -33 SF-12 M -58.8 SF-12 P -57.2	FF -170 ABD -90 ER -50	FF -180 ABD -180 ER -90 Constant -81.8		No
55	F	R; L	22.9	Pain (insidious onset)	Glenoid	5 × 8	SAD	VAS -8 SST -5 ASES -25	VAS -1 SST -10 ASES -95 SANE -100 UCLA -33 SF-12 M -57.8 SF-12 P -55.5	FF -106 ABD -N/A ER -50	FF -155 ABD -150 ER -75 Constant -68.4		No
55	F	R; R	35.7	Pain (prior surgery)	Glenoid	15 × 25	SAD, DCR, BT	VAS -N/A SST -4 ASES -N/A	VAS -2 SST -6 ASES -80 SANE -70 UCLA -N/A SF-12 M -56.2 SF-12 P -46			SAD, DCR	No
36	M	R; L	30.0	Pain (insidious onset)	Glenoid	10 × 10		VAS -1 SST -11 ASES -85	VAS -1 SST -12 ASES -95 SANE -92 UCLA -N/A SF-12 M -55.9 SF-12 P -57.2				No
37	M	R; R	89.8	Pain (prior surgery)	Glenoid and humerus	10 × 10 and 20 × 20	SAD, BT	VAS -5 SST -10 ASES -N/A	VAS -1 SST -12 ASES -93.3 SANE -90 UCLA -33 SF-12 M -57.8 SF-12 P -55.5	FF -150 ABD -150 ER -50	FF -172 ABD -180 ER -65 Constant -78.3	Labrum repair, SAD, RCR	No
48	M	R; L	12.1	Pain (insidious onset)	Humerus	20 × 20	SAD	VAS -9 SST -1 ASES -8.3	VAS -0 SST -12 ASES - 100SANE -90 UCLA -35 SF-12 M -44.6 SF-12 P -61.2	FF -100 ABD -100 ER -80	FF -150 ABD -160 ER -75 Constant -71.3		No
24	F	R; R	16.3	AVN	Humerus	20 × 20	Loose body removal	VAS -N/A SST -N/A ASES -N/A	VAS -0 SST -12 ASES -100 SANE -100 UCLA -35 SF-12 M -46.7 SF-12 P -25	FF -180 ABD -165 ER -55	FF -180 ABD -156 ER -90 Constant -74.8		No
51	M	R; R	33.3	Injury (sports)	Humerus	25 × 35		VAS -6 SST -5 ASES -48.3	VAS -0 SST -11 ASES -98.3 SANE -100 UCLA -35 SF-12 M -57.8 SF-12 P -55.2	FF -N/A ABD -N/A ER -N/A	FF -180 ABD -160 ER -90 Constant -68.9		No
42	M	L; R	13.1	Injury (sports)	Humerus	25 × 25	SAD	VAS -3 SST -10 ASES -61.7	VAS -0 SST -12 ASES -100 SANE -85 UCLA -35 SF-12 M -57.8 SF-12 P -55.5	FF -180 ABD -175 ER -55	FF -180 ABD -180 ER -65 Constant -73.4		No
25	M	R; L	33.5	Pain (prior surgery)	Humerus	20 × 20	Capsular release	VAS -4 SST -5 ASES -41.7	VAS -5 SST -6 ASES -45 SANE -65 UCLA -20 SF-12 M -29.9 SF-12 P -36.1	FF -40 ABD -40 ER -40	FF -180 ABD -140 ER -45 Constant -72.9	Labrum repair, SAD	Debridement and capsular release ^b (28)

(continued)

TABLE 1 (continued)

Age, y	Sex	Dominant Arm; Operative Arm	Length of Follow-up, mo	Preoperative Diagnosis	Defect Location	Defect Size, mm	Concomitant Procedure(s)	Preoperative Subjective Scores	Postoperative Subjective Scores	Preoperative ROM and Constant (strength)	Postoperative ROM and Constant (strength)	Previous Surgery	Subsequent Surgery (age, y)
18	F	R; R	18.2	Pain (prior surgery)	Humerus	20 × 20	BT	VAS -6 SST -5 ASES -45	VAS -7 SST -10 ASES -61.7 SANE -N/A UCLA -N/A SF-12 M -53.2 SF-12 P -46			Capsulorrhaphy	No
18	F	R; R	20.7	AVN	Humerus	28 × 28		VAS -8 SST -2 ASES -33.3	VAS -3 SST -9 ASES -76.7 SANE -70 UCLA -N/A SF-12 M -60 SF-12 P -45.8				No
18	F	R; L	22.3	AVN	Humerus	10 × 10		VAS -8 SST -2 ASES -35	VAS -4 SST -10 ASES -76.7 SANE -70 UCLA -N/A SF-12 M -60 SF-12 P -45.8				No
35	F	R; L	N/A	Injury (work)	Glenoid	10 × 10	SAD						Biological resurfacing ^b (37)
21	F	R; R	N/A	Pain (prior surgery)	Humerus	Grade 4 focal defect (size unavailable)						Stabilization, debridement	Hemiarthroplasty ^b (21)
37	F	R; R	N/A	Injury (sports)	Glenoid	10 × 10	SAD						- ^c Lost to follow-up 3 mo postop
38	M	R; R	N/A	Injury (sports)	Humerus	Grade 4 focal defect (size unavailable)							- ^c Lost to follow-up 2 mo postop

^aR, right; L, left; AVN, avascular necrosis; VAS, visual analog scale; SST, Simple Shoulder Test; ASES, American Shoulder and Elbow Society; SANE, Single Assessment Numerical Evaluation; UCLA, University of California–Los Angeles; SF-12 M, P, Short Form Mental, Physical; BT, biceps tenodesis; DCR, distal clavicle resection; RCR, rotator cuff repair; SAD, subacromial decompression; FF, forward flexion; ABD, abduction; ER, external rotation; N/A, not applicable.

^bDenotes failure; patient was included in descriptive analysis but not in statistical analysis.

^cDenotes that patient was lost to follow-up; patient was not included in descriptive or statistical analysis.

Physical examination by a single orthopaedic research fellow was performed independent of the operating surgeon in 8 patients (8 shoulders). The examination consisted of active and passive range of motion measured with a goniometer, including active forward elevation in the scapular plane, external rotation at the side, and internal rotation behind the back. Comparison was made with the opposite shoulder. Strength testing was performed using an Isobex handheld dynamometer (Cursor) for both forward elevation and external rotation at the side. To perform the examination, the examiner resisted the patient's motion (forward flexion or external rotation) as the dynamometer measured the force of the movement. Forward elevation was measured with the arm abducted in the scapular plane to 90° of elevation, and external rotation was measured with the arm at the side and the elbow flexed 90° in neutral rotation. A total of 3 measurements were made, and the highest value was used in calculation

of the Constant score. Comparisons were made with the opposite shoulder.

All results were analyzed via statistical testing comparing preoperative measures with corresponding postoperative measures at their last follow-up. Statistical analysis was performed utilizing both parametric and nonparametric testing methods using SPSS software (SPSS, Chicago, Illinois). Both Wilcoxon matched-pair tests as well as paired *t* tests were performed for the entire patient cohort. Independent *t* tests were used for analysis of objective data, including range of motion. Results were considered statistically significant with *P* < .05. Independent *t* tests were used when necessary because some postoperative physical examination components, including range of motion and strength testing, could not be measured when patients were not available for clinical examination. Statistical analysis was also performed to compare the patients who received physical examination versus those who did not.



Figure 1. Arthroscopic image depicting an isolated cartilage lesion with vertical walls.

Surgical Technique and Rehabilitation

The technique used for microfracture in the glenohumeral joint in these patients was similar to the technique used in other joints such as the knee. Portal placement was extremely important before beginning the microfracture. The anterior portal was placed more laterally when microfracture was performed on the anterosuperior glenoid, while a lower portal just above the subscapularis was placed when the defect was located more inferiorly. Posterior glenoid lesions were treated via portal placement in the posterior 7-o'clock position (right shoulder), while most lesions on the humerus were able to be reached through the standard anterior portal facilitated by internal and external rotation of the arm. In each patient, once the chondral defect was located, all loose cartilage and cartilage flaps were debrided using an arthroscopic shaver, ring curette, or basket forceps. After confirming that the chondral lesion was contained, vertical walls were created around the defect using a curette or arthroscopic elevator (Figure 1). Once adequate vertical walls were ensured, the entire layer of calcified cartilage was debrided with a curette, without penetrating the subchondral bone.^{28,29} At this point, a clean area of subchondral bone surrounded by vertical walls was clearly visible. A microfracture awl (Linvatec, Largo, Florida) was used to penetrate the subchondral bone, with each hole created perpendicular to the surface of the bone (Figure 2).^{17-19,22,25,33,39} Each microfracture hole was spaced approximately 3 to 4 mm apart and penetrated to a depth of approximately 2 to 4 mm (approximately the depth of the awl tip) into the subchondral surface in order to expose the marrow elements. Once the chondral lesion was penetrated with microfracture holes, cutterage or shaving was used to remove any bony remains on the rims of the holes. At this point, the irrigation pump pressure was reduced to ensure marrow elements appeared from the microfracture holes (Figure 3).

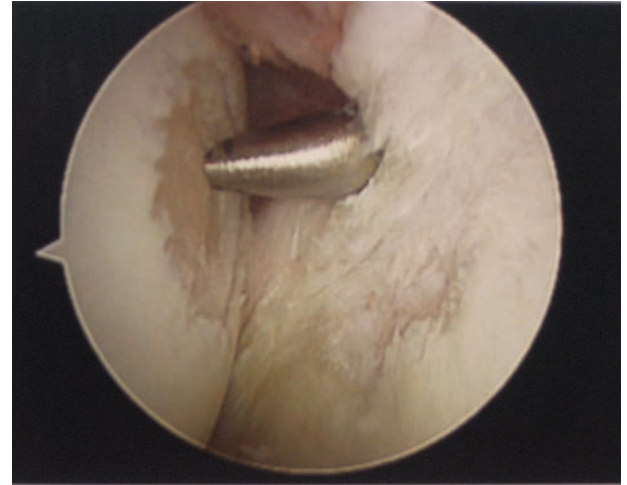


Figure 2. Arthroscopic image of microfracture awl penetrating subchondral bone.

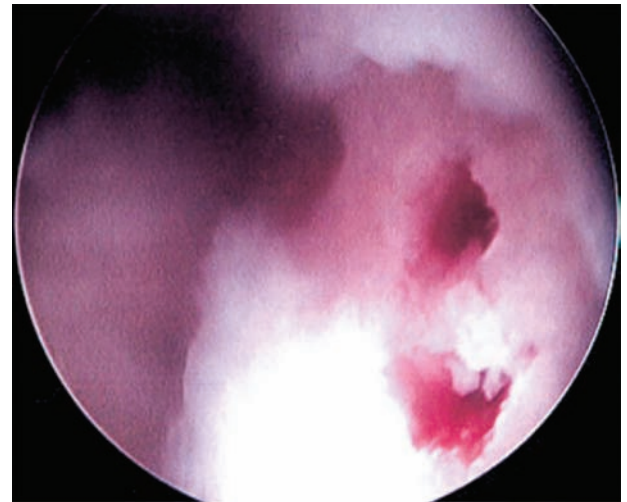


Figure 3. Arthroscopic image of marrow elements flowing through microfracture holes after tourniquet release.

Postoperatively, we did not use constant passive motion (CPM) to avoid stiffness, as is commonly utilized after microfracture in the knee.³⁶ Patients were provided sling immobilization for 2 to 4 weeks for comfort. Passive range of motion with progression to active assist and active range of motion was allowed and encouraged immediately after surgery. Standard protocol included a request for immediate pendulum exercises for at least 800 rotations per day. Light strengthening was initiated at 6 weeks if range of motion had been restored, with progression to unrestricted strengthening at 12 weeks postoperatively. All activities were allowed at 16 weeks, but overhead competitive athletics were restricted for 6 months.

TABLE 2
Outcomes of Microfracture^a

	Preoperative	Postoperative	<i>P</i> Value
VAS	5.6 ± 1.7	1.9 ± 1.4	<.001
ASES	44.3 ± 15.3	86.3 ± 10.5	<.001
SST	5.7 ± 2.1	10.3 ± 1.3	<.001

^aASES, American Shoulder and Elbow Surgeon; SST, Simple Shoulder Test; VAS, visual analog scale.

RESULTS

Of the 15 shoulders, microfracture was performed on the humeral head in 9 cases (60.0%), on the glenoid surface in 5 cases (33.3%), and on both surfaces in 1 case (6.7%). The average size of humeral defects was 5.07 cm² (range, 1.0-7.84 cm²), while that of glenoid defects was 1.66 cm² (range, 0.4-3.75 cm²). In addition to undergoing microfracture, 2 patients (13.3%) also underwent lysis of adhesions/capsular release, and 7 patients (46.7%) underwent subacromial decompression. Four patients (26.7%) had a biceps tenodesis at the time of surgery. Three patients (20.0%) underwent microfracture for treatment of avascular necrosis of the humeral head, while the other 12 patients received treatment for focal chondral defects.

Of the 13 shoulders for which all of the surveys and questionnaire-based follow-ups were completed, VAS, ASES, and SST scores improved significantly (*P* < .05) according to both the Student *t* test and Wilcoxon match-pair test analyses. The average preoperative VAS score was 5.6 ± 1.7 (range, 1-9), and at the time of follow-up, the average VAS score decreased significantly to 1.9 ± 1.4 (range, 0-7) (*P* < .01). The SST score also improved significantly from a mean of 5.7 ± 2.1 (range, 1-11) preoperatively to 10.3 ± 1.3 (range, 6-12) postoperatively (*P* < .01). Additionally, there was a statistically significant increase in ASES score from a mean of 44.3 ± 15.3 (range, 8.3-55) before surgery to 86.3 ± 10.5 (range, 45-100) at final follow-up (*P* < .01). These results are summarized in Table 2. After surgery, the Single Assessment Numeric Evaluation (SANE) score was 85.6 ± 13.3 (range, 65-100). Postoperatively, the University of California-Los Angeles (UCLA) Shoulder Score was 32.4 ± 5.1 (range, 27-35). Twelve of the 13 patients (92%) claimed that they would repeat the surgery. Finally, Short Form-12 Health Survey (SF-12) scores postoperatively were 49.3 ± 10.1 (range, 25-60.1) for the physical component (PCS) and 53.6 ± 8.6 (range, 29.9-60) for the mental component (MCS).

Physical examination was performed in 8 patients (8 shoulders). Postoperative objective measures including range of motion and strength were also compared with preoperative values. Preoperatively, the mean forward flexion of patients was 133.8° ± 27.8°, and this improved to 172.1° ± 10.4° (*P* = .08) postoperatively. The mean preoperative abduction value was 127.2° ± 37.2°, and this improved significantly to 163.3° ± 12.7° (*P* = .05) postoperatively. Finally, the mean external rotation value was 54.1° ± 8.7°, and this also improved significantly to 76°

TABLE 3
Comparison of Subjective Outcomes Among Patients Completing Questionnaires Versus Those Completing Both Questionnaires and Physical Examination^a

	Surveys Only	Surveys and Examination	<i>t</i> Test, <i>P</i> Value
<i>n</i>	5	8	
VAS	3.4 ± 2.3	0.9 ± 1.7	.045
ASES	78 ± 11.9	91.5 ± 18.9	>.05
SST	9.4 ± 2.2	10.9 ± 2.1	>.05
SANE	77.4 ± 10.4	90.6 ± 11.8	>.05
SF-12 Physical	48.2 ± 5.1	50.2 ± 12.6	>.05
SF-12 Mental	57.1 ± 2.9	51.4 ± 10.3	>.05

^aASES, American Shoulder and Elbow Surgeon; SST, Simple Shoulder Test; VAS, visual analog scale; SANE, Single Assessment Numerical Evaluation; SF-12, Short Form-12.

± 12.9° (*P* = .02) postoperatively. The mean postoperative Constant score, incorporating shoulder strength, was 73.2 ± 4.6 (range, 66.4-94.8).

Independent *t* tests were used to compare the postoperative subjective outcomes of patients who completed questionnaires only (*n* = 5) and those who completed both the questionnaires and the physical examination (*n* = 8). There was no statistical difference in SST, ASES, SANE, or SF-12 scores between the 2 groups. Interestingly, the patients who completed both the examination and surveys reported decreased pain levels compared with the patients who only completed the surveys, with a slightly lower mean postoperative VAS score (*P* = .045). These results are summarized in Table 3.

For the patients receiving microfracture only, and no concomitant procedures (*n* = 4), VAS improved from 5.75 ± 3.3 preoperatively to 2 ± 1.8 postoperatively. The SST scores also improved postoperatively in this subgroup (5.0 ± 4.2 to 10.5 ± 1.3), as did the ASES scores (50.4 ± 24.0 to 86.7 ± 11.6). In the subgroup of patients receiving a capsular release in addition to the microfracture (*n* = 2), postoperative improvements were also seen in VAS, SST, and ASES scores. Specifically, VAS improved from 4.0 ± 0 preoperatively to 2.5 ± 3.5 postoperatively; SST increased from 6.5 ± 2.1 to 9.0 ± 4.24; and ASES improved from 50.83 ± 12.96 to 72.5 ± 38.89. In these 2 patients, range of motion also substantially increased, with forward flexion improving from 105° ± 91.9° preoperatively to 180° ± 0° postoperatively, abduction improving from 65° ± 35.4° to 160° ± 28.3°, and external rotation improving from 45° ± 7.1° to 67.5° ± 31.8°. When compared with the patients who did not receive capsular release (but received other concomitant procedures), the postoperative flexion, abduction, and external rotation scores were 169.5° ± 13.6°, 164.3° ± 12.7°, and 78.9° ± 9.7°, respectively, values similar to those for the capsular release patients. Statistical analysis was not performed on these subgroups of patients because of the small number of patients within each group.

For the 2 patients requiring subsequent resurfacing or replacement shoulder surgery, the time between the

microfracture and the subsequent surgery was 16.4 months and 2.5 months, respectively. The age of the patients at the time of the subsequent surgery was 37 and 21 years old, respectively. The first patient was a workers' compensation patient, had no prior surgeries apart from the microfracture procedure, and at the time of microfracture, this patient also had concomitant subacromial decompression. Operative findings included a 10 × 10-mm grade 4 articular cartilage defect located on the anterior central glenoid with associated extensive synovitis. The patient had continued complaints of pain and poor function 3 months after surgery, and she had no response to subacromial or glenohumeral injection, although the articular lesions remained the only potential source of pain. This patient received an open osteochondral allograft to the glenoid 16.4 months after microfracture. The other failed patient had undergone 2 "stabilization" surgeries before microfracture procedure and underwent a subsequent hemiarthroplasty 2.5 months after the microfracture. Operative findings for this patient at the index procedure included a single focal defect of the humeral cartilage with circumferential fraying and thinning of the surrounding cartilage. At the time of the hemiarthroplasty, there was noted to be significant progression of the articular lesion with poor fibrocartilage fill. Both patients' subjective and objective outcomes after the microfracture procedure were not included in this study as these patients failed microfracture within 3 months of the procedure and went on to a revision procedure before this study was completed. The demographics of these patients are included in Table 1.

The patient who went on to arthroscopic debridement and capsular release 3 years after microfracture was also considered to have failed results. Two years before the microfracture, this patient had a previous arthroscopic shoulder stabilization that led to postoperative stiffness and pain. During the microfracture procedure, a 20 × 20-mm isolated lesion of the humeral head was found, and this patient also underwent capsular release. After surgery, the patient did well in terms of pain relief but had some residual stiffness and mechanical symptoms after 2 years. After failed conservative therapy including injections, the patient underwent arthroscopic debridement and capsular release 3 years after microfracture, at which time extensive degeneration of both the humeral and glenoid surfaces was found. At the time of follow-up for this study, 2.8 years after microfracture, this patient had an ASES of 45, a SANE of 65, SST of 6, VAS of 5, and forward flexion of 180°, abduction of 140°, and external rotation of 45°. Because of the low ASES and SANE scores, as well as his progression on to subsequent surgery, this patient was considered a failure.

Only 2 patients with work-related injuries were seen for workers' compensation evaluations. One of these patients, as described above, failed microfracture and went on to subsequent shoulder surgery, while the other patient improved both subjectively and objectively, with the exception of external rotation. Specifically, VAS, ASES, and SST scores improved in this patient from 9 to 0, 8.33 to 100, and 1 to 12, respectively, while range of motion improved from

100° to 150° (flexion) and 100° to 160° (abduction). External rotation actually decreased in this patient, from 80° preoperatively to 75° postoperatively; however, this change was considered negligible.

No additional complications or reoperations occurred in the entire patient cohort related to their surgical treatment.

DISCUSSION

The results from this study suggest that microfracture is an effective short-term treatment method for full-thickness chondral defects of the shoulder. The goal of microfracture is to encourage chondral resurfacing by gaining access to the underlying marrow and creating an environment poised for tissue regeneration through utilizing the body's natural vascular response to injury,^{4,31,37,39} and the basic science behind the microfracture technique has been thoroughly examined.^{22,33,39} Blood with marrow elements enters a prepared chondral lesion and organizes into a fibrous clot, which consists of mesenchymal stem cells, growth factors, fibrin, and platelets. Cells within the clot undergo metaplasia to initially form granulation tissue.^{37,40} Within the first postoperative week, the granulation tissue undergoes fibrosis and then over the course of 6 to 12 months hyalinization and chondrification to ultimately become fibrocartilage if proper rehabilitation and surgical technique are implemented. This resulting fibrocartilagenous tissue ultimately repairs what once was a full-thickness chondral defect. Because of the molecular and cellular features of articular cartilage, *de novo* healing of such defects is rare, and microfracture has proven to be a successful, minimally invasive surgical option to stimulate healing in the knee.^{23,34}

Recently, the arthroscopic microfracture technique has also been used as an attempt to correct chondral lesions in joints other than the knee that are arthroscopically accessible. In addition to the knee joint, microfracture has become common in joints that often experience articular injuries such as the hip, ankle, and elbow, and recently, articular injuries in the shoulder joint are receiving more clinical exposure.^{2,5,6,15,16} While the indications for microfracture as well as the surgical techniques have been clearly defined for the knee joint, there are no reviews in the literature that discuss the technique of microfracture specifically in the shoulder, nor are there any case reports. Because of the success of microfracture in the knee, we wondered if the results could be repeated in the shoulder. The purpose of this study was to report our initial results with the microfracture technique in the shoulder.

The treatment algorithm for articular cartilage defects in the shoulder is difficult because many of the cartilage defects encountered during shoulder arthroscopy may be incidental or secondary findings. Although chondral lesions in the shoulder are much less common than in other joints, they may be seen in association with trauma, instability, rotator cuff tears, or labral injury. Furthermore, it is impossible to determine the source of symptoms in these shoulders because alternate procedures are often planned and performed at the same time. For example,

in this study, several patients also received subacromial decompression in addition to their microfracture. As previously mentioned, the diagnosis of symptomatic chondral lesions is difficult and nonspecific. In addition, the symptoms are often overlapping with other potential diagnosis. Therefore, subacromial decompression was performed in an effort to address all potential sources of symptoms.

The reported outcomes from simple debridement in shoulders with chondral disease have been limited, and the majority of previous reports include patients with diffuse articular disease, not specific focal defects.^{10,41} A recent study by Kerr and McCarty²⁶ described the outcomes of patients who received arthroscopic debridement for either unipolar or bipolar cartilage defects of the shoulder joint and reported that patients with unipolar lesions did much better than those with bipolar lesions. However, while the majority of patients in this study (16/19) received additional procedures during the debridement, only 2 received microfracture.²⁶ Therefore, it is unknown if microfracture provides improved outcomes over debridement alone, and further study is warranted.

The indications for microfracture of articular cartilage defects in the shoulder remain poorly defined. In this series, our indication for treatment was younger, active patients with full-thickness defects of the glenoid and/or humeral surfaces. Additionally, we attempted to minimize confounding variables by choosing patients without significant coexisting pathological changes to explain their symptoms (rotator cuff tear or labral tear). The goal was to provide maximal potential benefit while maintaining minimal surgical morbidity. More invasive restorative options for the shoulder have been proposed, including allograft transplantation and autologous chondrocyte implantation.^{14,23} While each of these procedures are viable options, they all require an open procedure with significant increase in morbidity and risk for complications and thus are best suited for a second-line treatment option.

The biological implications for microfracture in the shoulder need further study. From a biological standpoint, there are significant differences between the shoulder joint and other joints, which may affect results. First, the articular cartilage thickness in the shoulder is significantly less than in the knee or ankle. Specifically, the articular depth of the glenoid fossa is 1.88 mm, and the humerus is 1.24 mm.⁴² Additionally, the shape of the glenohumeral joint may not be ideal for microfracture techniques. Specifically in the glenoid, the articular cartilage is thickest at the periphery and tapers toward the center with an area devoid of cartilage (the bare area) often present at the center of the glenoid surface. This would suggest that clinically relevant defects are often present at the periphery of the glenoid, making them more likely to be uncontained. On the humeral side, the joint surface is convex, which may make containment of the initial fibrin clot difficult and suboptimal. As discussed above, the initial formation and containment of the fibrin clot is the important initial step in fibrocartilage maturation. On the other hand, a potential advantage of the shoulder joint compared with other joints is that it is a nonweightbearing joint, which may decrease load across the repair tissue, resulting in improved results.

Similar to indications, contraindications for microfracture of articular lesions in the shoulder remain poorly defined. We would propose similar contraindications to those reported for the knee joint including diffuse degenerative disease, kissing lesions (focal defects with a bipolar reciprocal corresponding defect), and untreated instability. It should be noted that in this study, 1 patient received microfracture to both the glenoid surface and humeral head; however, we did not feel that this particular patient had a "kissing" lesion as the defects on both surfaces did not reciprocate each other in terms of location. Cartilage lesions due to tumors, infection, inflammatory arthritis, and/or systemic disease are not considered good candidates for microfracture.²⁸ The indications for microfracture in patients with avascular necrosis (AVN) are unclear, as the volume of patients presenting with this lesion and undergoing microfracture is limited, and thus, the success of microfracture is not yet known. Microfracture can certainly be considered in patients with AVN with articular cartilage involvement without collapse. At the current time, we do not have a size limit because of the paucity of literature on chondral defects in the shoulder. In this study, defect sizes ranged from 5 × 8 mm to 28 × 28 mm, and because of the variety of defect sizes among the relatively small study population, it was impossible to determine if defect size had an effect on outcomes. Relative contraindications include patients with concomitant injuries such as labral, biceps, or rotator cuff injury. Often intra-articular chondral defects are incidental findings in the setting of these more common or prevalent diagnoses and should be considered as such without primary treatment of the articular cartilage disease until further data are available.

The results from this study suggest that microfracture is an acceptable treatment method for isolated chondral defects of the glenohumeral joint, as the overall success rate was 80% (3 failures). The 4 patients who received only microfracture as well as those who also received concomitant procedures improved in terms of subjective and objective outcomes, and thus, it is difficult to evaluate whether the microfracture procedure itself was responsible for the improvement in symptoms. Because of the small number of patients receiving only microfracture ($n = 4$), these patients could not be compared statistically with the rest of the cohort. The 2 patients who received capsular release in addition to their microfracture also improved in subjective and objective outcomes, especially with regard to range of motion. Thus, while microfracture may have resulted in substantial pain relief for these patients, thereby allowing the patients to more comfortably move their shoulders, the capsular release component of the surgery may also have contributed to the improved range of motion.

The 3 patients who had further operations subsequent to the microfracture procedure were evaluated to determine if any specific factors contributed to the failed microfracture. In the patient who went on to osteochondral allograft transplantation, there were similar intraoperative findings compared with the rest of the patient cohort. This patient's initial cause of injury was work related. After microfracture, the patient experienced significant amounts of pain despite physical therapy and steroid

injections, and it remained unclear as to the origin of the pain, although the articular defect remained the focal source of injury. In the patient who required subsequent hemiarthroplasty, the intraoperative findings again were similar to the rest of the patient cohort, although some chondral degeneration was noted beyond the area of microfracture. The initial cause of pain was softball related, and the patient underwent 2 stabilization surgeries before microfracture. After microfracture, this patient continued to experience pain and elected to undergo hemiarthroplasty as a salvage procedure. There was clear progression of the disease at the time of hemiarthroplasty. Finally, in the patient who went on to arthroscopic debridement and capsular release, there were also similar intraoperative findings compared with the rest of the cohort. This patient initially had microfracture after previous surgery and continued to have mechanical symptoms several years after the microfracture, leading him to have further surgery.

We are aware of only 1 other study on microfracture in the shoulder. Siebold et al³² reported on a prospective cohort of patients in whom they treated symptomatic chondral lesions of the shoulder with open microfracture and a superimposed periosteal flap. There are several differences between their cohort of patients and ours. First, this study included only 5 patients the majority of whom had cartilage defects as a result of instability (80%). Three of the 5 had the instability treated previously, but 2 patients needed the instability treated at the time of microfracture. An additional 2 patients had chondral damage from prominent hardware, necessitating implant removal. The most obvious difference was that these patients were treated with an open procedure with the addition of a periosteal flap. Despite the differences in the study, they report similar results to ours with this technique, with a statistical improvement in the Constant and pain scores in a small patient cohort.³²

This study has several limitations, including the retrospective nature, lack of control group, and short time of clinical follow-up. While the average time for follow-up is over 27 months, some of the patients only had 1 year of follow-up. Further studies with a larger patient cohort and longer follow-up are needed to ensure that the results of microfracture in the shoulder will stand up over time. Another limitation of this study is the number of concomitant procedures performed in this patient population. A difficulty with treatment of shoulder chondral injuries is knowing which injuries are truly symptomatic and which are incidental. It is impossible to know if the articular defects were responsible for the preoperative symptoms in these patients and if the microfracture resulted in whole or in part for the clinical improvement. In the previously mentioned 2008 study by Kerr et al,²⁶ this same limitation was noted, as concomitant procedures, including biceps tenotomy and SLAP repairs, were performed in 16 of the 19 patients (20 shoulders) undergoing arthroscopic debridement for either unipolar or bipolar chondral defects of the glenohumeral joint. In the present study, we did attempt to minimize confounding variables in this regard by eliminating patients who underwent formal labral or rotator cuff repair.

Another limitation is that we did not perform a radiographic analysis of these patients postoperatively to determine if there had been any interval healing or progression of arthrosis. Additional studies on this patient population with magnetic resonance imaging and radiographs are needed to determine the degree of fibrocartilage fill and to note any progression of arthrosis. Further second-look arthroscopic studies with biopsy could be performed to determine the fill and nature of cartilage produced in a joint known to have less synovial fluid production and thinner cartilage.

These preliminary results suggest that isolated chondral defects of the shoulder treated with proper microfracture techniques can yield results similar to those reported for the knee.^{1,28} Patients can expect improvement in pain, function, and range of motion. Even those patients with relatively lower postoperative ASES and SANE scores still experienced improvement from their preoperative values. However, further study is needed to determine the indications for microfracture and further clarify factors predictive of outcome. Additionally, further long-term clinical studies with larger patient populations are needed to fully evaluate the effectiveness of microfracture in the shoulder joint.

REFERENCES

1. Asik M, Ciftci F, Sen C, Erdil M, Atalar A. The microfracture technique for the treatment of full-thickness articular cartilage lesions of the knee: midterm results. *Arthroscopy*. 2008;24(11):1214-1220.
2. Becher C, Driessen A, Thermann H. [Microfracture technique for the treatment of articular cartilage lesions of the talus]. *Orthopade*. 2008;37(3):196, 198-203.
3. Bishop JY, Flatow EL. Management of glenohumeral arthritis: a role for arthroscopy? *Orthop Clin North Am*. 2003;34(4):559-566.
4. Blevins FT, Steadman JR, Rodrigo JJ, Silliman J. Treatment of articular cartilage defects in athletes: an analysis of functional outcome and lesion appearance. *Orthopedics*. 1998;21(7):761-767, discussion 767-768.
5. Bojanic I, Ivkovic A, Boric I. Arthroscopy and microfracture technique in the treatment of osteochondritis dissecans of the humeral capitellum: report of three adolescent gymnasts. *Knee Surg Sports Traumatol Arthrosc*. 2006;14(5):491-496.
6. Bradley JP, Petrie RS. Osteochondritis dissecans of the humeral capitellum: diagnosis and treatment. *Clin Sports Med*. 2001;20(3):565-590.
7. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *N Engl J Med*. 1994;331(14):889-895.
8. Burkhead WZ Jr, Hutton KS. Biologic resurfacing of the glenoid with hemiarthroplasty of the shoulder. *J Shoulder Elbow Surg*. 1995;4(4):263-270.
9. 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(5):1165-1172.
10. Cameron BD, Galatz LM, Ramsey ML, Williams GR, Iannotti JP. Non-prosthetic management of grade IV osteochondral lesions of the glenohumeral joint. *J Shoulder Elbow Surg*. 2002;11(1):25-32.
11. Cameron ML, Kocher MS, Briggs KK, Horan MP, Hawkins RJ. The prevalence of glenohumeral osteoarthritis in unstable shoulders. *Am J Sports Med*. 2003;31(1):53-55.
12. Chapovsky F, Kelly JD 4th. Osteochondral allograft transplantation for treatment of glenohumeral instability. *Arthroscopy*. 2005;21(8):1007.

13. Cohen NP, Foster RJ, Mow VC. Composition and dynamics of articular cartilage: structure, function, and maintaining healthy state. *J Orthop Sports Phys Ther.* 1998;28(4):203-215.
14. Cole BJ, Yanke A, Provencher MT. Nonarthroplasty alternatives for the treatment of glenohumeral arthritis. *J Shoulder Elbow Surg.* 2007;16(5 Suppl):S231-240.
15. Crawford K, Philippon MJ, Sekiya JK, Rodkey WG, Steadman JR. Microfracture of the hip in athletes. *Clin Sports Med.* 2006;25(2):327-335, x.
16. Dodson CC, Nho SJ, Williams RJ 3rd, Altchek DW. Elbow arthroscopy. *J Am Acad Orthop Surg.* 2008;16(10):574-585.
17. Freedman KB, Cole BJ. Microfracture technique in the knee. In: Miller MD, Cole BJ, eds. *Textbook of Arthroscopy.* Philadelphia: Elsevier; 2004:575-579.
18. Frisbie DD, Morisset S, Ho CP, Rodkey WG, Steadman JR, McIlwraith CW. Effects of calcified cartilage on healing of chondral defects treated with microfracture in horses. *Am J Sports Med.* 2006;34(11):1824-1831.
19. Frisbie DD, Trotter GW, Powers BE, et al. Arthroscopic subchondral bone plate microfracture technique augments healing of large chondral defects in the radial carpal bone and medial femoral condyle of horses. *Vet Surg.* 1999;28(4):242-255.
20. Gartsman GM, Taverna E. The incidence of glenohumeral joint abnormalities associated with full-thickness, reparable rotator cuff tears. *Arthroscopy.* 1997;13(4):450-455.
21. Gerber C, Lambert SM. Allograft reconstruction of segmental defects of the humeral head for the treatment of chronic locked posterior dislocation of the shoulder. *J Bone Joint Surg Am.* 1996;78(3):376-382.
22. Gill TJ, Asnis PD, Berkson EM. The treatment of articular cartilage defects using the microfracture technique. *J Orthop Sports Phys Ther.* 2006;36(10):728-738.
23. Gomoll AH, McCulloch P, Kang RW, Cole BJ. Treatment of chondral defects in the shoulder. *Oper Tech Orthop.* 2006;16(4):232-243.
24. Hogan CJ, Diduch DR. Progressive articular cartilage loss following radiofrequency treatment of a partial-thickness lesion. *Arthroscopy.* 2001;17(6):E24.
25. Kelly BT, MacGillivray JD. Microfracture of articular cartilage. In: Simonian PT, Cole BJ, Bach BR Jr, eds. *Sports Injuries of the Knee: Surgical Approaches.* New York: Thieme Medical Publishers; 2006:42-46.
26. Kerr BJ, McCarty EC. Outcome of arthroscopic debridement is worse for patients with glenohumeral arthritis of both sides of the joint. *Clin Orthop Relat Res.* 2008;466(3):634-638.
27. McCarty LP 3rd, Cole BJ. Nonarthroplasty treatment of glenohumeral cartilage lesions. *Arthroscopy.* 2005;21(9):1131-1142.
28. Mithoefer K, Williams RJ 3rd, Warren RF, et al. Chondral resurfacing of articular cartilage defects in the knee with the microfracture technique: surgical technique. *J Bone Joint Surg Am.* 2006;88(Suppl 1 Pt 2):294-304.
29. Mithoefer K, Williams RJ 3rd, Warren RF, et al. The microfracture technique for the treatment of articular cartilage lesions in the knee: a prospective cohort study. *J Bone Joint Surg Am.* 2005;87(9):1911-1920.
30. 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(1):35-40.
31. Rodrigo JJ, Steadman JR, Silliman J. Improvement of full-thickness chondral defect healing in the human knee after debridement and microfracture using continuous passive motion. *Am J Knee Surg.* 1994;7:109-116.
32. Siebold R, Lichtenberg S, Habermeyer P. Combination of microfracture and periosteal-flap for the treatment of focal full thickness articular cartilage lesions of the shoulder: a prospective study. *Knee Surg Sports Traumatol Arthrosc.* 2003;11(3):183-189.
33. Sledge SL. Microfracture techniques in the treatment of osteochondral injuries. *Clin Sports Med.* 2001;20(2):365-377.
34. Steadman JR, Briggs KK, Rodrigo JJ, Kocher MS, Gill TJ, Rodkey WG. Outcomes of microfracture for traumatic chondral defects of the knee: average 11-year follow-up. *Arthroscopy.* 2003;19(5):477-484.
35. Steadman JR, Miller BS, Karas SG, Schlegel TF, Briggs KK, Hawkins RJ. The microfracture technique in the treatment of full-thickness chondral lesions of the knee in National Football League players. *J Knee Surg.* 2003;16(2):83-86.
36. Steadman JR, Rodkey WG, Briggs KK. Microfracture to treat full-thickness chondral defects: surgical technique, rehabilitation, and outcomes. *J Knee Surg.* 2002;15(3):170-176.
37. Steadman JR, Rodkey WG, Briggs KK, Rodrigo JJ. [The microfracture technic in the management of complete cartilage defects in the knee joint]. *Orthopade.* 1999;28(1):26-32.
38. Steadman JR, Rodkey WG, Rodrigo JJ. Microfracture: surgical technique and rehabilitation to treat chondral defects. *Clin Orthop Relat Res.* 2001;391(Suppl):S362-369.
39. Steadman JR, Rodkey WG, Singleton SB, Briggs KK. Microfracture technique for full thickness chondral defects: technique and clinical results. *Oper Tech Orthop.* 1997;7:300-304.
40. Uchio Y, Ochi M. [Biology of articular cartilage repair: present status and prospects]. *Clin Calcium.* 2004;14(7):22-27.
41. Weinstein DM, Bucchieri JS, Pollock RG, Flatow EL, Bigliani LU. Arthroscopic debridement of the shoulder for osteoarthritis. *Arthroscopy.* 2000;16(5):471-476.
42. Yeh LR, Kwak S, Kim YS, et al. Evaluation of articular cartilage thickness of the humeral head and the glenoid fossa by MR arthrography: anatomic correlation in cadavers. *Skeletal Radiol.* 1998;27(9):500-504.