Arthroscopic Cartilage Lesion Preparation in the Human Cadaveric Knee Using a Curette Technique Demonstrates Clinically Relevant Histologic Variation

Boguslaw Sadlik, M.D., Ph.D., Adrian Matlak, M.D., Adrian Blasiak, M.D., Wojciech Klon, M.D., Mariusz Puszkarz, M.D., and Graeme P. Whyte, M.D., M.Sc., F.R.C.S.C.

Purpose: To examine the quality of arthroscopic cartilage debridement using a curette technique by comparing regional and morphologic variations within cartilage lesions prepared in human cadaveric knee specimens for the purpose of cartilage repair procedures. A secondary aim was to compare the histologic properties of cartilage lesions prepared by surgeons of varying experience. **Methods:** Standardized cartilage lesions (8 mm × 15 mm), located to the medial/lateral condyle and medial/lateral trochlea were created within 12 human cadaver knees by 40 orthopaedic surgeons. Participants were instructed to create full-thickness cartilage defects within the marked area, shouldered by uninjured vertical walls of cartilage, and to remove the calcified cartilage layer, without violating the subchondral plate. Histologic specimens were prepared to examine the verticality of surrounding cartilage walls at the front and rear aspects of the lesions, and to characterize the properties of the surrounding cartilage, the cartilage wall profile, the debrided lesion depth, bone sinusoid access, and the bone surface profile. Comparative analysis of cartilage wall verticality measured as deviation from perpendicular was performed, and Spearman's rank correlation analysis was used to examine associations between debrided wall verticality and surgeon experience. **Results:** Mean cartilage wall verticality relative to the base of the lesion was superior at the rear aspect of the lesion compared to the front aspect (12.9° vs 29.2°, $P < .001$). Variability was identified in the morphology of the surrounding cartilage ($P < .001$), cartilage wall profile ($P = .016$), debrided lesion depth ($P = .028$), bone surface profile ($P = .040$), and bone sinusoid access ($P = .009$), with sinusoid access identified in 42% of cases. There was no significant association of cartilage lesion wall verticality and surgeon years in practice ($r_s = 0.161$, $P = .065$) or arthroscopic caseload ($r_s = -0.071$, $P = .419$). **Conclusions:** Arthroscopic cartilage lesion preparation using standard curette technique in a human cadaveric knee model results in inferior perpendicularity of the surrounding cartilage walls at the front aspect of the defect, compared to the rear aspect. This technique has shown significant variability in the depth of debridement, with debridement depths identified as either too superficial or too deep to the calcified cartilage layer in more than 60% of cases in this study. Surgeon experience does not appear to impact the morphologic properties of cartilage lesions prepared arthroscopically using ring curettes. **Clinical Relevance:** To optimize restoration of hyaline-like cartilage tissue, careful attention to prepared cartilage lesion morphology is advised when arthroscopically performing cartilage repair, given the tendency for standard curette technique to create inferior verticality of cartilage walls at the front of the lesion, and the variable depth of debridement achieved.

From the Biological Joint Reconstruction Department, St. Luke's Hospital (B.S., A.M., A.B., W.K., M.P.), Bielsko-Biala, Poland; Weill Medical College, Cornell University; New York Presbyterian Hospital/Queens (G.P.W.), New York, NY, U.S.A.; and Orthopaedic Arthroscopic Surgery International (OASI) Bioresearch Foundation (G.P.W.), Milan, Italy.

The authors report that they have no conflicts of interest in the authorship and publication of this article. Full ICMJE author disclosure forms are available for this article online, as supplementary material.

Received June 6, 2017; accepted January 31, 2018.

Address correspondence to Graeme P. Whyte, M.D., M.Sc., F.R.C.S.C., Weill Medical College, Cornell University; New York Presbyterian Hospital/Queens, 56-45 Main Street, New York, NY 11355, U.S.A. E-mail: gphwhyte@gmail.com

0749-8063/17719/$36.00

https://doi.org/10.1016/j.arthro.2018.01.049

As cartilage repair techniques and technologies progress, greater attention is being paid to the functional biology of the osteochondral unit, and the need to create an optimal environment to restore hyaline-like cartilage.[1-3] Regeneration of articular cartilage typically occurs by a process of resorption and reformation, and appositional outgrowth from the underlying subchondral bone likely plays an important role in the repair process.[4-6]

When undertaking a cartilage repair procedure, the method of cartilage lesion preparation should be highly scrutinized, to provide the optimal environment for articular cartilage restoration to proceed. Two important considerations in cartilage lesion preparation are the

depth of debridement and the structure of the cartilage walls that contain the lesion peripherally. The tidemark and the layer of calcified cartilage are metabolically active regions that act as an interface between the articular cartilage and the underlying subchondral bone.[7,8] Removal of the calcified cartilage layer within chondral defects, while maintaining integrity of the subchondral end-plate, has been shown to result in cartilage repair tissue of superior quality in an animal model,[9] although there continues to be debate regarding the optimal depth of cartilage lesion preparation. Furthermore, using animal models, Drobnič et al.[10] reported that violation of the subchondral end-plate was a frequent occurrence during cartilage lesion debridement, whereas Mika et al.[11] reported that this occurred rarely.

Achieving perpendicularity of the surrounding cartilage walls when preparing a cartilage defect is of great clinical significance. In cases of cell-based cartilage repair techniques in particular, ensuring vertical walls about the periphery will help minimize any residual partial-thickness cartilage tissue at the defect base, and will also assist with containment of the cell-based graft and security of fixation within the defect.[12-16] The retention of partial-thickness cartilage tissue at the base of the lesion should be avoided, as this residual tissue may impair the process of appositional cartilage restoration that is thought to originate from the articular surface.[5]

Arthroscopic methods of cell-based cartilage repair such as autologous chondrocyte implantation and stem cell-embedded scaffolds are becoming more frequently used, and are an attractive treatment option, given the positive clinical outcomes that may be achieved in a minimally invasive setting.[17-21] There is currently a lack of detailed analysis regarding the capability of standard arthroscopic methods to achieve optimal cartilage lesion preparation before undertaking cartilage repair procedures in humans. The purpose of this study was to examine the quality of arthroscopic cartilage debridement using standard ring curette technique by comparing regional and morphologic variations within cartilage lesions prepared in human cadaveric knee specimens for the purpose of cartilage repair procedures. A secondary aim was to compare the histologic properties of cartilage lesions prepared by surgeons of varying experience. We hypothesized that the arthroscopically prepared cartilage lesions would show region-dependent variation in morphology and that lesions prepared by more experienced surgeons would be associated with superior cartilage wall verticality about the prepared defects.

Methods

Specimens and Arthroscopy Setup

Twelve fresh frozen human cadaver knee specimens were thawed and secured to an apparatus in a manner consistent with positioning for standard knee arthroscopy. All specimens were skeletally mature, free of significant chondral injury or degenerative change, and were obtained from the United Tissue Network. The arthroscopic setup consisted of the Synergy HD3 Imaging Platform and the Continuous Wave III Arthroscopy Pump (Arthrex, Naples, FL). Standardized ovoid areas of cartilage (8 mm × 15 mm) were arthroscopically demarcated superficially within the medial femoral condyle, lateral femoral condyle, medial trochlea, and lateral trochlea of fresh human cadaver knee specimens using arthroscopic cautery by a supervising orthopaedic surgeon experienced in cartilage lesion debridement and repair.

Arthroscopic Procedure

Forty orthopaedic surgeons experienced in knee arthroscopy participated in the study. A survey was completed by each participant before the trial procedure to assess years of experience and arthroscopic caseload. Three or four surgeons were randomly assigned to each specimen, and they completed the surgical task in pairs, alternating the role of operator and assistant. Each participant prepared a single cartilage lesion. Participants were instructed to independently create full-thickness cartilage defects within the marked area, shouldered by uninjured vertical walls of cartilage, and to remove the calcified cartilage layer, without violating the subchondral plate. Twelve minutes were given for each cartilage lesion preparation.

Surgeons used standard anterolateral and anteromedial portals for visualization and instrumentation. Surgeons were allowed to alternate portal use and to create additional working portals for instrumentation as they preferred to optimize cartilage lesion preparation as per the stated goals. Within the apparatus, the secured knee joint specimen was free to flex and extend as desired by the operator. The role of the assistant was to retrieve surgical instruments under the direction of the operator, and to assist with positioning of the knee specimen through the range of flexion and extension, as directed by the operator. To complete the surgical task, each surgeon had available a cup curette (Arthrex), a 5.4-mm ring curette one side cutting (Arthrex), and a 5.4-mm ring curette both sides cutting (Arthrex). It was expected that ring curettes would be preferentially used. After lesion preparation, the front aspect of the defect was marked with electrocautery 10 mm beyond the lesion margin by the surgeon. After harvesting the osteochondral block, the rear aspect of the lesion was marked with a chisel at the edge of the block.

Histologic Evaluation

Individual osteochondral blocks encompassing each lesion with a 1-cm margin surrounding this area were

Table 1. Histological Criteria for the Evaluation of Cartilage Lesion Debridement

Criteria	Description	Measurement Categories
Lesion angle	The analysis/angle function was applied to: (a) Outer angles—defined between the surrounding cartilage surface and the lesion wall on both sides of the samples (b) Inner angles—defined between the lesion wall and its base	Degrees
Surrounding cartilage	Up to 2 mm of the intact cartilage on each side of the rim of the debrided lesion was analyzed using a semiquantitative scale	Intact Shallow fissure(s) (<20% depth) Partial-thickness injury (20% to 80% depth) Full-thickness injury (>80%)
Cartilage wall profile	The cartilage wall on both sides of the lesion was analyzed for any present disturbances using a semiquantitative scale	Straight, flat Superficial disturbances <1 mm Concave Convex
Debrided lesion depth	Each histological slide was analyzed by a stereological cycloid grid incorporated into a microscope lens at 100× magnification. The depth level at each intersection with the gridline was determined and the corresponding percentages were calculated	Deep zone Calcified zone Subchondral end-plate Deep bone (sinusoids)
Bone sinusoids access	The openings to the bone sinusoids were counted	Number of openings.
Bone surface profile	Bone surface profile	Straight, flat Superficial disturbances <1 mm Crater (central part deeper) Bump (peripheral part deeper)

Adapted from Drobnič et al.[10]

harvested using an oscillating saw and chisel. Each specimen was fixed in 10% buffered formalin for 72 hours, and subsequently decalcified for a 4-week period. Samples were embedded in paraffin and then cut longitudinally through the debrided area in the anterior to posterior direction. Two to six serial parallel tissue sections for each sample were prepared to identify the best quality histologic section for each sample. Serial sections were stained with hematoxylin and eosin, scanned with a high-resolution scanner (Epson Perfection V37), and examined using computer-aided design and drafting computer software (AutoCAD LT, 2016). Each section showed the longitudinal plane of the lesion, consisting of the subchondral bone at the base, and the cartilage walls located at the front and rear aspects of each prepared lesion. A histological analysis was performed to evaluate the morphology of the simulated cartilage defects using criteria adapted from Drobnič et al.[10] (Table 1). All samples were evaluated by a PhD candidate and reviewed by a senior histopathologist. The verticality of the cartilage walls about the periphery of each lesion, measured as the angle deviation from perpendicular, was measured at the front and rear aspects of the lesion, depending on the arthroscopic approach to the lesion (Fig 1). The front aspect of the lesion corresponded to the anterior aspect of condylar lesions and the inferior/distal aspect of trochlear lesions, whereas the rear aspect corresponded to the posterior aspect of condylar lesions and the superior/proximal aspect of trochlear lesions. Perpendicularity of the cartilage wall to the chondral surface and to the subchondral surface were both examined, given that these surfaces may not necessarily be perfectly parallel. Front-inner (FI) and rear-inner (RI) measurements were made and corresponded to the angle between the cartilage wall and the lesion base. Front-outer (FO) and Rear-outer (RO) measurements were used to examine the angle between the cartilage wall and the surrounding cartilage. Additional morphologic parameters of prepared defects that were examined consisted of the properties of the surrounding cartilage, the cartilage wall profile, the debrided lesion depth, bone sinusoid access within the subchondral bone, and the bone surface profile (Fig 2).

Statistical Analysis

Data analysis was performed by a statistician experienced in clinical data science, using SPSS software (version 20.0; IBM Corp., Armonk, NY). Normality of continuous variables was examined using the Shapiro-Wilks test and quantile-quantile plots. Continuous variables were expressed as mean ± standard deviation. Spearman's rank correlation coefficient was calculated to examine the monotonic relation between surgeon experience and cartilage wall verticality after curette lesion preparation. The absolute values of angle measurements were used for comparative analysis of cartilage wall verticality. Analysis of variance was used to compare mean angle measurements, with post hoc Tukey correction performed for multiple comparisons where appropriate. One-sample χ-square testing was used to examine frequency distribution within categories of histologic morphology of cartilage lesions after curette preparation. A post hoc pairwise analysis of histologic morphology was performed, and the Benjamini and Hochberg procedure was applied to control

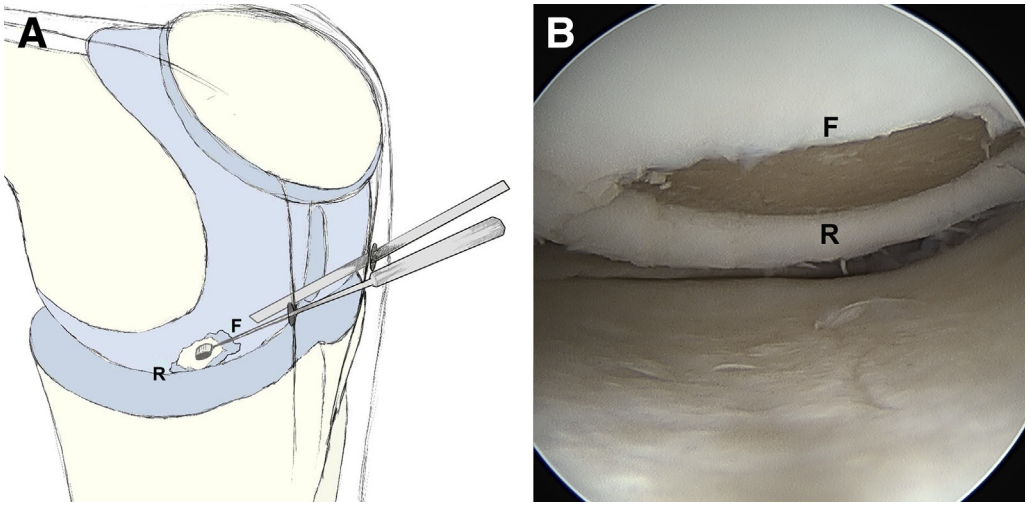

Fig 1. (A) Diagrammatic representation of arthroscopic cartilage lesion preparation on the medial femoral condyle and (B) an arthroscopic image of lesion preparation on a medial femoral condyle. Front (F) and rear (R) aspects of the cartilage lesion depicted.

the false discovery rate for multiple comparisons.[22] A priori power analysis was performed to determine the sample sizes required for comparison of mean angle differences and for correlation analysis of surgeon experience and prepared lesion angles, using an α value of 0.05 and β of 0.2. To identify a 10° difference in mean angles, with an expected standard deviation of 10°, 17 measurements were required for each grouping of mean angle measurements. To examine correlation of surgeon experience and angle measurements using Spearman's rho, a total of 82 measurements were necessary, using a medium effect size ($\rho = 0.3$). All analysis was 2-tailed.

Results

There were 40 surgeons who performed arthroscopic cartilage lesion preparation, and 40 osteochondral blocks were subsequently harvested for histologic analysis. Because of specimen damage during harvesting, or inadequacy of the prepared histologic sample, 7 specimens were excluded, resulting in 33 specimens available for analysis. The mean duration of surgeon time in active practice was 10.7 ± 6.5 years. The mean number of surgeon arthroscopies performed per month was 14.6 ± 7.8. Rank correlation analysis did not show any significant association of cartilage lesion wall verticality and surgeon years in practice ($r_s = 0.161$, $P = .065$) or arthroscopic caseload ($r_s = -0.071$, $P = .419$). The relation between surgeon experience and cartilage lesion wall verticality is depicted in scatterplots (Fig 3).

The mean absolute values of angle measurements of cartilage wall verticality for the RO, RI, FO, and FI locations were 12.9° ± 12.1°, 12.9° ± 12.4°, 25.6° ± 17.5°, and 29.2° ± 20.5°, respectively (Table 2). There were no differences in cartilage wall verticality between the RO and RI locations ($P \geq .999$), or between the FO and FI locations ($P = .793$). Mean cartilage wall verticality, as measured perpendicular to the lesion base, was superior at the RI location compared with the FI ($P < .001$) location. Mean cartilage wall verticality, as measured between the vertical wall and surrounding cartilage, was superior at the RO location compared with the FO location ($P = .009$).

Histologic analysis of lesion morphology showed that in the majority (61%) of prepared cartilage lesions, the surrounding cartilage was characterized as intact, with only 3% of specimens having full-thickness lesions identified. With respect to the cartilage wall profile, the most commonly identified morphology was that of superficial disturbances. Thirty-six percent of lesions were debrided to a depth consistent with the calcified zone, with 27% of lesions debrided to the subchondral

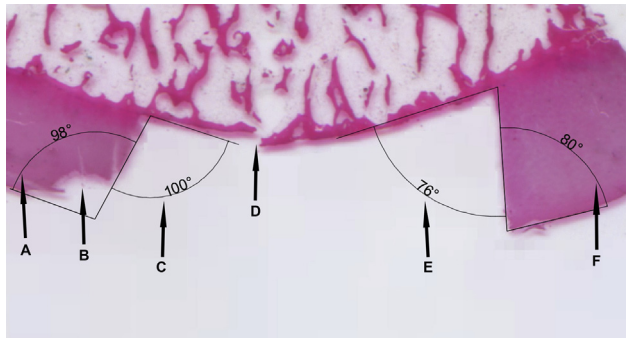

Fig 2. Histologic section from a cartilage lesion prepared arthroscopically. (A) Rear-outer angle measurement between the cartilage wall and the chondral surface. (B) Partial-thickness lesion of the surrounding cartilage. (C) Rear-inner angle measurement between the cartilage wall and the subchondral surface. (D) Bone sinusoids access. (E) Front-inner angle measurement between the cartilage wall and the subchondral surface. (F) Front-outer angle measurement between the cartilage wall and the chondral surface. Lines of best fit used for angle measurements, as depicted in (A), (C), (E), and (F).

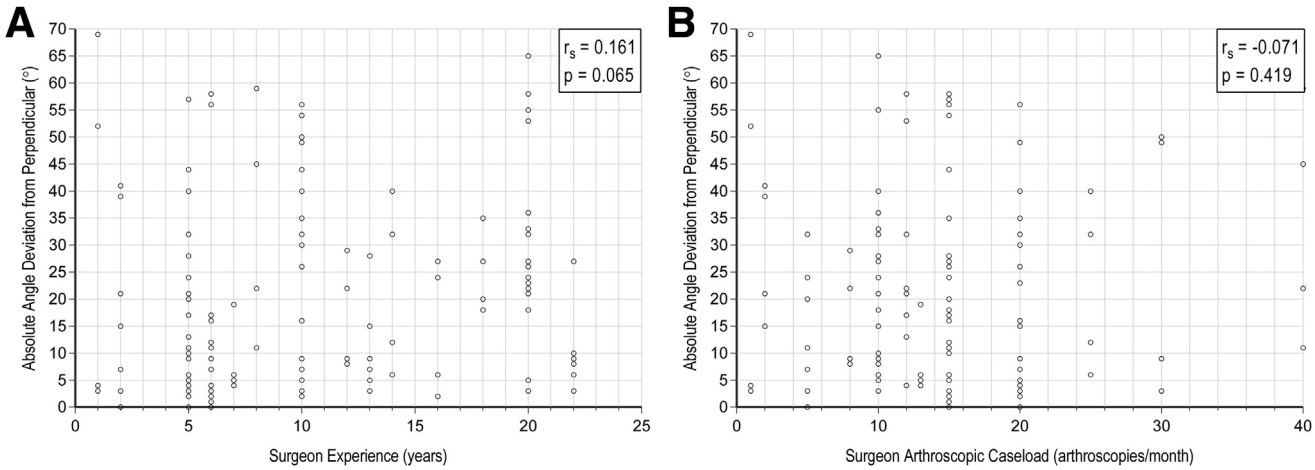

Fig 3. Scatterplots depicting (A) the relation of surgeon years in practice and verticality of the cartilage walls about the periphery of prepared cartilage lesions and (B) the relation of surgeon arthroscopic caseload and verticality of the cartilage walls about the periphery of prepared cartilage lesions. Spearman's rho and associated *P* values are presented to describe the monotonic relation between variables.

end-plate, and 33% debrided to the level of deep bone. The majority of lesion debridements (58%) did not result in any bone sinusoid access. At the base of the prepared lesion, the bone surface profile was characterized as flat in 42%, with 30% of lesions described as having a crater within the bone surface profile. The histologic morphologies of prepared cartilage lesions are detailed in Table 3.

Standard deviations of angle measurements were greater than the expected value used for a priori power analysis. Post hoc power analysis using the calculated mean and standard deviations of RI (12.9° ± 12.4°) and FI (29.2° ± 20.5°) wall angles, using an α value of 0.05, with a sample of 33 measurements within each grouping, determined the power to be 0.97.

Discussion

Using standard arthroscopic ring curette technique, the cartilage walls located to the FI and FO areas of the lesions were significantly less vertical compared to areas of the wall located to the RI and RO regions. In addition, there was no association of surgeon experience and the ability to prepare a cartilage lesion with vertical walls about the periphery.

The greater difficulty achieving near-perpendicularity of cartilage walls at the front aspect of the defects compared with the rear aspect that was shown in this study is an important consideration, given that this finding would be less likely in cases of open debridement, where the front and rear aspects of the lesion are easily visible. This is of particular importance where

Table 2. Comparison of Cartilage Wall Perpendicularity About the Periphery of Debrided Cartilage Lesions

Location of Curette Debridement About Lesion Periphery	Mean of Cartilage Wall Absolute Angle Deviation from Perpendicular (90°) ± SD	*P* Value[†]
Rear outer	12.9° ± 12.1°	<.001*
Rear inner	12.9° ± 12.4°	
Front outer	25.6° ± 17.5°	
Front inner	29.2° ± 20.5°	
Multiple Comparisons of Mean Angles		***P* Value[‡]**
Rear outer vs Rear inner		>.999
Rear outer vs Front outer		.009*
Rear outer vs Front inner		<.001*
Rear inner vs Front outer		.008*
Rear inner vs Front inner		<.001*
Front outer vs Front inner		.793

ANOVA, analysis of variance; SD, standard deviation.
*Statistically significant.
[†]One-way ANOVA group analysis to examine for statistical significance between locations of debridement about the lesions.
[‡]Post hoc Tukey analysis for multiple pairwise comparisons between each location of debridement.

Table 3. Histologic Assessment of Cartilage Lesion Morphologic Characteristics After Arthroscopic Ring Curette Preparation

Surrounding Cartilage	Percentage of Cartilage Lesions	*P* Value[†]	Paired Comparison	*P* Value[‡]
Intact (I)	61	<.001*	I vs Sh	.006*
Shallow fissure(s) (Sh)	12		I vs P	.072
Partial-thickness lesion (P)	24		I vs F	.001*
Full-thickness lesion (F)	3		Sh vs P	.388
			Sh vs F	.450
			P vs F	.058
Cartilage wall profile				
Straight, flat (Sf)	12	.016*	Sf vs Sd	.114
Superficial disturbances (Sd)	46		Sf vs Cv	>.999
Concave (Cv)	12		Sf vs Cx	.540
Convex (Cx)	30		Sd vs Cv	.114
			Sd vs Cx	.848
			Cv vs Cx	.540
Debrided lesion depth				
Deep zone (Dz)	3	.028*	Dz vs Cz	.018*
Calcified zone (Cz)	36		Dz vs Se	.042*
Subchondral end-plate (Se)	27		Dz vs Db	.018*
Deep bone (Db)	33		Cz vs Se	.996
			Cz vs Db	>.999
			Se vs Db	.989
Bone sinusoids access				
None (N)	58	.009*	N vs M	.131
Minor (M)	27		N vs S	.021*
Severe (S)	15		M vs S	.424
Bone surface profile				
Straight, flat (Sf)	42	.040*	Sf vs Sd	.230
Superficial disturbances (Sd)	18		Sf vs C	.541
Crater (C)	30		Sf vs B	.078
Bump (B)	9		Sd vs C	.684
			Sd vs B	.610
			C vs B	.276

*Statistically significant.
[†]One-sample χ-square testing to examine significance of frequency distribution.
[‡]Post hoc pairwise analysis, *P* values adjusted to account for multiple comparisons using the Benjamini and Hochberg procedure.

arthroscopic visibility tends to be more problematic, such as in the case of large lesions, or lesions positioned more posteriorly on the femoral condyles. This highlights the importance for the treating surgeon to ensure maximum visibility at the front of the lesion, to minimize areas of suboptimal cartilage wall preparation about the lesion periphery. Cutting away from the lesion periphery toward the center of the defect arthroscopically is problematic at the front aspect of cartilage lesions, given the design limitations of standard ring curettes. Arthroscopic instruments that incorporate a front-cutting design may enable the surgeon to shape cartilage walls at the front of defects that more consistently achieve perpendicularity to the subchondral plate.

With respect to cartilage lesion preparation in anticipation of cartilage repair procedures, including marrow stimulation or cell-based procedures such as autologous chondrocyte implantation, the cartilage about the periphery of the lesion is ideally uninjured circumferentially. In vivo animal studies have shown that partial-thickness defects or beveling at the margins of the defects are associated with deficiencies of cartilage reformation,[23-25] which has the potential to impact all manner of repair procedures that rely on restoration of durable cartilage tissue. In addition, the structure of the surrounding wall is important to contain marrow elements in cases of marrow stimulation procedures,[26] and to optimize stability of grafts used in cell-based repair. The majority of the prepared lesions in this study showed completely intact surrounding cartilage, with less than 30% of lesions having either partial- or full-thickness injury peripherally. Importantly, however, the profile of the surrounding cartilage wall was less ideal, with almost 50% having superficial disturbances, and 30% containing the least ideal type of profile, described as convex on histologic examination. This could potentially affect uniform integration of cartilage repair tissue about the periphery of lesions treated with cartilage restoration procedures.

The subchondral bone is an integral part of the osteochondral unit that is needed to sustain overlying articular cartilage.[6] Pathologic processes of articular cartilage are intimately associated with changes to the subchondral bone.[27,28] Subchondral end-plate growth, intra-lesional osteophyte development, and other alterations in subchondral bone anatomy may impact the quality of cartilage repair tissue and limit clinical outcome success in cases of cartilage restoration procedures.[29-32] These factors highlight the importance of maintaining integrity of subchondral bone when preparing cartilage lesions as a component of cell-based cartilage repair procedures. The calcified cartilage layer and tidemark play an important role in solute transport from underlying subchondral bone to overlying articular cartilage, and are metabolically active layers with important function within the osteochondral unit that change with age and degenerative joint injury.[28,33-40] The calcified cartilage layer also acts as an interface between the articular cartilage and subchondral bone, providing a stabilizing function.[8,41] Longevity of repaired cartilage tissue depends not only on restoration of uncalcified articular cartilage, but

also on maintenance of a healthy osteochondral unit that includes the calcified cartilage and tidemark layers. Regarding the depth of lesion debridement, there is evidence that improved cartilage restoration may be achieved when the depth of debridement at the lesion base is performed past the level of calcified cartilage, while leaving the subchondral end-plate intact.[9] This may lead to qualitative improvements in restoration of the calcified cartilage layer and overlying tidemark, which are important functional structures necessary for longevity of cartilage repair tissue. Both the calcified cartilage and tidemark layers are dynamic structures that are metabolically active, with the tidemark layer being particularly important for preventing vascular encroachment on the articular cartilage, thereby preventing calcification.[42] In the current study, 36% of lesions were debrided to the calcified cartilage layer, but not beyond. The level of debridement was to the subchondral end-plate in 27% of lesions, and to the deep bone in 33%. Inconsistency of achieving the target depth of debridement has potential clinical implications related to the uniform regeneration of articular cartilage, given that appositional growth from the articular surface is likely a crucial component of this repair process.[5] Although debriding to the depth of deep bone occurred in a third of cases, histologic examination of the subchondral bone surface showed that the majority of lesion preparations did not result in any bone sinusoid access. With regard to the bone surface profile, the morphology was categorized as the most ideal "straight, flat" in 42% of lesions, although 30% of lesions were described as containing a "crater" within the bone surface profile. Histologic assessment of the cartilage lesions prepared arthroscopically in this study using a standardized ring curette technique showed significant variability with respect to depth of debridement, and improvement in both technique and instrumentation may provide more consistency in this regard. If arthroscopic cartilage lesion preparation is to be undertaken by the treating surgeon when performing cartilage repair procedures, complete visualization of the entirety of the defect base should be ensured, and instruments should be capable of adequately approaching all of the treated surface area in a trajectory that enables uniform depth of debridement.

By examining various techniques of cartilage preparation, Drobnič et al.[10] showed that mechanical curettage was superior to debridement using an arthroscopic shaver or electrocautery, according to the histologic examination of prepared lesion morphology using the criteria presented in Table 1. This experimental study examined cartilage lesion preparation in both human and equine specimens. It is of note that the equine specimens were deemed to lack skeletal maturity, and there was no tidemark or calcified cartilage layer identified, making the use of these specimens of questionable validity, given the high frequency of subchondral end-plate violation. Furthermore, the ability to achieve verticality of cartilage walls at the front and rear aspects of prepared lesions was not examined in this work. We believe this to be an important limitation with respect to investigation of arthroscopic lesion preparation, as our findings have shown significantly superior perpendicularity of peripheral cartilage walls at the rear aspect of the lesions, compared with the front. The decreased arthroscopic visibility in this region of the defect and the limitations of the curette instrumentation are likely contributors to this finding.

Mika et al.[11] investigated the histologic properties of cartilage lesions prepared in both human and sheep models. Using the standard ring curette technique of lesion preparation, there were no cases of subchondral end-plate violation, unless a brute force was intentionally applied. Importantly, however, the described technique in this study prepared defects with a force sufficient to remove uncalcified cartilage, leaving the underlying tidemark and calcified cartilage layer intact. This is in contrast to the current study, where the technique of lesion preparation was intended to remove the tidemark and calcified cartilage layer, as this method has been shown to provide a more optimal environment for cartilage restoration.[9] It is notable that the methodology of the current study relied entirely on human cadaver specimens, which is a significant advantage compared with other recent studies such as the works by Mika et al.[11] and Drobnič et al.[10] that have used various animal models. The suitability of animal specimens in the field of cartilage restoration can pose difficulties, given morphologic and physiologic differences from human articular cartilage and subchondral bone.[10,11,43-49]

Overall, with respect to the described histological parameters used in this study to assess the quality of arthroscopic cartilage lesion preparation using standard curette technique, there appears to be significant variability with respect to the debridement depth, as well as suboptimal verticality of the cartilage wall at the front aspect of the prepared lesions. It is not clear if these findings are primarily operator dependent, or if improvement to the standard curette instrumentation and technique would result in more ideal qualities of prepared lesions. Given that surgeon experience was not significantly associated with differences in the morphology of prepared lesions, it is likely that the type of instruments used for the task of arthroscopic lesion debridement more greatly contributed to the variability shown in the morphology of defects prepared in this study. Further examination of cartilage lesion preparation using instruments specifically designed to optimize accuracy and uniformity of arthroscopic debridement will better clarify the shortcomings of the standard curette technique.

Limitations

There are a number of limitations to this study. Cadaver specimens may not provide identical tissue properties to that of an in vivo model. All histologic sections available in this study were not analyzed, as there were several samples rejected because of damage during the harvesting procedure or histologic preparation. With respect to the orientation of prepared histologic samples, it was not technically feasible to obtain full length transverse sections of the cartilage defects after sectioning in the longitudinal plane. As the authors believe that preparation of the front and rear defect walls pose a greater technical challenge, the sectioning orientation was chosen to be in the longitudinal plane, as opposed to the transverse plane. With regard to the histologic samples used for analysis, a single section was used for final analysis, based on the determination of the most complete specimen according to the examiner. Although examining multiple sections for each defect may be possible, it was believed that the variable histologic quality of sections obtained for each sample may have introduced greater bias. Although deviation of surrounding cartilage walls from perpendicular alignment was an important focus of our analysis, it is not clear the degree to which this deviation impacts clinical outcomes in the setting of cartilage repair procedures. Moreover, although histologic evaluation did not identify concern of cartilage injury related to the superficial markings at the chondral surfaces using electrocautery, it is possible that this method of templating affected the tissue properties. Additionally, measurements from each location of debridement within the knee were pooled for analysis, and there is location-dependent variation in the radius of curvature of the articular surfaces within the knee that may have impacted analysis. With regard to surgeon years of experience and arthroscopic caseload, data were compiled and analyzed using self-reported values, potentially introducing recall bias. Although the value of mean standard deviation of angle measurements used for a priori power analysis was greater than expected, post hoc analysis determined the power to be adequate at 0.97.

Conclusions

Arthroscopic cartilage lesion preparation using standard ring curette technique in a human cadaveric knee model results in inferior perpendicularity of the surrounding cartilage walls at the front aspect of the defect, compared to the rear aspect. This technique has shown significant variability in the depth of debridement, with debridement depths identified as either too superficial or too deep to the calcified cartilage layer in over 60% of cases in this study. Surgeon experience does not appear to impact the morphologic properties of cartilage lesions prepared arthroscopically using ring curettes.

References

1. McCormick F, Harris JD, Abrams GD, et al. Trends in the surgical treatment of articular cartilage lesions in the United States: An analysis of a large private-payer database over a period of 8 years. *Arthroscopy* 2014;30:222-226.
2. Goldring SR, Goldring MB. Changes in the osteochondral unit during osteoarthritis: Structure, function and cartilage-bone crosstalk. *Nat Rev Rheumatol* 2016;12:632-644.
3. Madry H. The subchondral bone: A new frontier in articular cartilage repair. *Knee Surg Sports Traumatol Arthrosc* 2010;18:417-418.
4. Hunziker EB, Kapfinger E, Geiss J. The structural architecture of adult mammalian articular cartilage evolves by a synchronized process of tissue resorption and neoformation during postnatal development. *Osteoarthr Cartil* 2007;15:403-413.
5. Hayes AJ, MacPherson S, Morrison H, Dowthwaite G, Archer CW. The development of articular cartilage: Evidence for an appositional growth mechanism. *Anat Embryol (Berl)* 2001;203:469-479.
6. Pan J, Zhou X, Li W, Novotny JE, Doty SB, Wang L. In situ measurement of transport between subchondral bone and articular cartilage. *J Orthop Res* 2009;27:1347-1352.
7. Jiang J, Leong NL, Mung JC, Hidaka C, Lu HH. Interaction between zonal populations of articular chondrocytes suppresses chondrocyte mineralization and this process is mediated by PTHrP. *Osteoarthr Cartil* 2008;16:70-82.
8. Oegema TR, Carpenter RJ, Hofmeister F, Thompson RC. The interaction of the zone of calcified cartilage and subchondral bone in osteoarthritis. *Microsc Res Tech* 1997;37:324-332.
9. 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;34:1824-1831.
10. Drobnič M, Radosavljevic D, Cör A, Brittberg M, Strazar K. Debridement of cartilage lesions before autologous chondrocyte implantation by open or transarthroscopic techniques: A comparative study using post-mortem materials. *J Bone Joint Surg Br* 2010;92:602-608.
11. Mika J, Clanton TO, Pretzel D, Schneider G, Ambrose CG, Kinne RW. Surgical preparation for articular cartilage regeneration without penetration of the subchondral bone plate: In vitro and in vivo studies in humans and sheep. *Am J Sports Med* 2011;39:624-631.
12. Marlovits S, Striessnig G, Kutscha-Lissberg F, et al. Early postoperative adherence of matrix-induced autologous chondrocyte implantation for the treatment of full-thickness cartilage defects of the femoral condyle. *Knee Surg Sports Traumatol Arthrosc* 2005;13:451-457.
13. Nehrer S, Spector M, Minas T. Histologic analysis of tissue after failed cartilage repair procedures. *Clin Orthop Relat Res* 1999;(365):149-162.
14. Whyte GP, McGee A, Jazrawi L, Meislin R. Comparison of collagen graft fixation methods in the porcine knee: Implications for matrix-assisted chondrocyte implantation and second-generation autologous chondrocyte implantation. *Arthroscopy* 2016;32:820-827.

15. Gobbi A, Whyte GP. One-stage cartilage repair using a hyaluronic acid-based scaffold with activated bone marrow-derived mesenchymal stem cells compared with microfracture: Five-year follow-up. *Am J Sports Med* 2016;44:2846-2854.
16. Puszkarz M, Sadlik B, Solecki A. Chondrectomy—review of surgical instrumentation and its effectiveness. *Ortop Traumatol Rehabil* 2015;17:333-342.
17. Petersen W, Zelle S, Zantop T. Arthroscopic implantation of a three dimensional scaffold for autologous chondrocyte transplantation. *Arch Orthop Trauma Surg* 2008;128:505-508.
18. Whyte GP, Gobbi A, Sadlik B. Dry arthroscopic single-stage cartilage repair of the knee using a hyaluronic acid-based scaffold with activated bone marrow-derived mesenchymal stem cells. *Arthrosc Tech* 2016;5:e913-e918.
19. Ibarra C, Izaguirre A, Villalobos E, et al. Follow-up of a new arthroscopic technique for implantation of matrix-encapsulated autologous chondrocytes in the knee. *Arthroscopy* 2014;30:715-723.
20. Sadlik B, Gobbi A, Puszkarz M, Klon W, Whyte GP. Biologic inlay osteochondral reconstruction: Arthroscopic one-step osteochondral lesion repair in the knee using morselized bone grafting and hyaluronic acid-based scaffold embedded with bone marrow aspirate concentrate. *Arthrosc Tech* 2017;6:e383-e389.
21. Sadlik B, Jaroslawski G, Puszkarz M, et al. Cartilage repair in the knee using umbilical cord Wharton's jelly-derived mesenchymal stem cells embedded onto collagen scaffolding and implanted under dry arthroscopy. *Arthrosc Tech* 2018;7:e57-e63.
22. Benjamini Y, Yosef H. Controlling the false discovery rate: A practical and powerful approach to multiple testing. *J Roy Stat Soc Ser B Stat Methodol* 1995;57:289-300.
23. Hunziker EB, Quinn TM. Surgical removal of articular cartilage leads to loss of chondrocytes from cartilage bordering the wound edge. *J Bone Joint Surg Am* 2003;85:85-92 (suppl).
24. Rudd RG, Visco DM, Kincaid SA, Cantwell HD. The effects of beveling the margins of articular cartilage defects in immature dogs. *Vet Surg* 1987;16:378-383.
25. DePalma AF, McKeever CD, Subin DK. Process of repair of articular cartilage demonstrated by histology and autoradiography with tritiated thymidine. *Clin Orthop Relat Res* 1966;48:229-242.
26. Steadman JR, Rodkey WG, Briggs KK. Microfracture technique for full-thickness chondral defects: Technique and clinical results. *Op Tech Orthop* 1997;7:300-304.
27. Pape D, Filardo G, Kon E, van Dijk CN, Madry H. Disease-specific clinical problems associated with the subchondral bone. *Knee Surg Sports Traumatol Arthrosc* 2010;18:448-462.
28. Burr DB, Schaffler MB. The involvement of subchondral mineralized tissues in osteoarthrosis: Quantitative microscopic evidence. *Microsc Res Tech* 1997;37:343-357.
29. Orth P, Cucchiarini M, Kohn D, Madry H. Alterations of the subchondral bone in osteochondral repair—translational data and clinical evidence. *Eur Cell Mater* 2013;25:299-316.
30. Henderson IJP, La Valette DP. Subchondral bone overgrowth in the presence of full-thickness cartilage defects in the knee. *Knee* 2005;12:435-440.
31. Qui YS, Shahgaldi BF, Revell WJ, Heatley FW. Observations of subchondral plate advancement during osteochondral repair: A histomorphometric and mechanical study in the rabbit femoral condyle. *Osteoarthr Cartil* 2003;11:810-820.
32. Gomoll AH, Madry H, Knutsen G, et al. The subchondral bone in articular cartilage repair: Current problems in the surgical management. *Knee Surg Sports Traumatol Arthrosc* 2010;18:434-447.
33. Lane LB, Bullough PG. Age-related changes in the thickness of the calcified zone and the number of tidemarks in adult human articular cartilage. *J Bone Joint Surg Br* 1980;62:372-375.
34. Arkill KP, Winlove CP. Solute transport in the deep and calcified zones of articular cartilage. *Osteoarthr Cartil* 2008;16:708-714.
35. Lyons TJ, Stoddart RW, McClure SF, McClure J. The tidemark of the chondro-osseous junction of the normal human knee joint. *J Mol Histol* 2005;36:207-215.
36. Müller-Gerbl M, Schulte E, Putz R. The thickness of the calcified layer of articular cartilage: A function of the load supported? *J Anat* 1987;154:103-111.
37. Koszyca B, Fazzalari NL, Vernon-Roberts B. Calcified cartilage, subchondral and cancellous bone morphometry within the knee of normal subjects. *Knee* 1996;3:15-22.
38. Burr DB. Anatomy and physiology of the mineralized tissues: Role in the pathogenesis of osteoarthrosis. *Osteoarthritis Cartilage* 2004;12:S20-S30 (suppl A).
39. Duer MJ, Friščić T, Murray RC, Reid DG, Wise ER. The mineral phase of calcified cartilage: Its molecular structure and interface with the organic matrix. *Biophys J* 2009;96:3372-3378.
40. Bonde HV, Talman MLM, Kofoed H. The area of the tidemark in osteoarthritis—A three-dimensional stereological study in 21 patients. *APMIS* 2005;113:349-352.
41. Madry H, van Dijk CN, Mueller-Gerbl M. The basic science of the subchondral bone. *Knee Surg Sports Traumatol Arthrosc* 2010;18:419-433.
42. Hoemann C, Lafantaisie-Favreau C-H, Lascau-Coman V, Chen G, Guzmán-Morales J. The cartilage-bone interface. *J Knee Surg* 2012;25:85-97.
43. Chevrier A, Kouao ASM, Picard G, Hurtig MB, Buschmann MD. Interspecies comparison of subchondral bone properties important for cartilage repair. *J Orthop Res* 2015;33:63-70.
44. Hunziker EB, Quinn TM, Häuselmann HJ. Quantitative structural organization of normal adult human articular cartilage. *Osteoarthr Cartil* 2002;10:564-572.
45. Hunziker EB. Biologic repair of articular cartilage. Defect models in experimental animals and matrix requirements. *Clin Orthop Relat Res* 1999;:S135-S146 (suppl).
46. Orth P, Meyer HL, Goebel L, et al. Improved repair of chondral and osteochondral defects in the ovine trochlea

compared with the medial condyle. *J Orthop Res* 2013;31:1772-1779.
47. Namba RS, Meuli M, Sullivan KM, Le AX, Adzick NS. Spontaneous repair of superficial defects in articular cartilage in a fetal lamb model. *J Bone Joint Surg Am* 1998;80:4-10.
48. Munirah S, Samsudin OC, Chen HC, Salmah SHS, Aminuddin BS, Ruszymah BHI. Articular cartilage restoration in load-bearing osteochondral defects by implantation of autologous chondrocyte-fibrin constructs: An experimental study in sheep. *J Bone Joint Surg Br* 2007;89:1099-1109.
49. Terukina M, Fujioka H, Yoshiya S, et al. Analysis of the thickness and curvature of articular cartilage of the femoral condyle. *Arthroscopy* 2003;19:969-973.