Letters to the Editor

Lamina-Specific Double-Row Fixation of Rotator Cuff Tears

To the Editor:

We read with great interest the recent article by Mori et al.\(^1\) published in the December 2014 issue of *Arthroscopy Techniques* (available at arthroscopytechniques.org). We congratulate the authors for their work but would like to elucidate some aspects of lamina-specific rotator cuff repairs.

Mori et al.\(^1\) have published a well-illustrated description of a technique to repair delaminated rotator cuff tears entitled “Arthroscopic lamina-specific double-row fixation for large delaminated rotator cuff tears.” The technique is characterized by passing lamina-specific lateral-row simple sutures through the articular-sided layer and lateral-row simple sutures through the bursa-sided layer—in addition to medial-row mattress sutures through both layers.

By addressing both layers, the authors expect their technique to create a larger contact area between the inferior layer and the tendon’s footprint and to “form an adaptation between the superficial and inferior layers.”

By using full-thickness mattress sutures for the medial row in addition to the lamina-specific single sutures, they aim for higher initial fixation strength. At least in part, this technique has been described previously by Hepp et al.\(^2\) in their article “Knotless anatomic double-layer double-row rotator cuff repair: A novel technique re-establishing footprint and shape of full-thickness tears.”

Mori et al.\(^1\) correctly reference Sugaya et al.\(^3\), who were the first authors to describe layer-specific rotator cuff repairs. In his publication, Spencer\(^4\) was the first investigator to also apply the concept of layer-specific repair to tears that were not clearly delaminated. Both articles described techniques that used single stitches, which may result in reduced biomechanical stability. For both methods and similar to the technique described by Mori et al., the knots for the articular-sided lamina repair had to be buried within the tendon, which may lead to an inflammatory foreign-body response and affect tendon healing.\(^1,3,4\)

To address these problems, Hepp et al.\(^2,5\) described a knotless double-layer double-row technique with lamina-specific mattress sutures. The use of a knotless technique avoids intratendinous knots and results in a low profile of the repair, preventing it from potential catching on the acromion—coracoacromial ligament arch.\(^2\) When compared with a single-suture lamina-specific configuration in a biomechanical setup, the lamina-specific mattress sutures proved to provide better initial fixation strength and to be as strong as a standard full-thickness double-row repair.\(^5\)

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References


Authors’ Reply

We highly appreciate the comments of Drs. Osterhoff and Hepp on our article “Arthroscopic lamina-specific double-row fixation for large delaminated rotator cuff tears” (available at arthroscopytechniques.org). Actually, comparing techniques between lamina-specific double-row (LSDR) and knotless anatomical double-layer double-row (DLDR) rotator cuff repair, the
DLDR repair is superior to LSDR repair in terms of avoiding suture knots buried within the repaired tendons.

The purpose of our article was to present the operative technique and indications for DLDR repair. As described in the article, we indicate DLDR repair for delaminated rotator cuff tears whose inferior layer is retracted medial to the glenoid (large-sized rotator cuff tears). Moreover, DLDR repair is limited to cases in which the inferior layer can be pulled laterally to the footprint with a tendon grasper, avoiding DLDR repair for shoulders that have friable inferior and superior layers. This is why we believe more suture knots and their proper placement is more beneficial in tendon healing for repairing larger rotator cuff tears.¹

We would expect DLDR repair to achieve better tendon healing in terms of the following aspects: The technique (1) creates a larger area of contact between the inferior layer and the footprint, (2) leads to higher initial fixation strength of the articular-side components of the repaired rotator cuff tendon, and (3) forms an adaptation between the superficial and inferior layers. However, we have not yet presented the clinical and structural results or complications, such as an inflammatory foreign-body response. Therefore clinical and structural studies are required to confirm our expectation.

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Reference

The New Microfracture: All Things Considered

To the Editor:

Microfracture, as introduced by Drs. Steadman and Rodkey, contributed a great deal in the pursuit of cartilage repair. They carefully described their patient and pathologic selection, surgical technique, and reported the long-term outcomes.¹ They initially introduced their procedure for partial-thickness traumatic cartilage lesions that did not penetrate the calcified layer. The purpose was to access the subchondral bone vascularity that presumably carried pluripotential cells. Subsequently, they expanded their method to include Grade IV, full-thickness, end-stage lesions.

Jack Bert’s expert opinion (Level V evidence) and related Editorial Commentary in the March issue of Arthroscopy, has properly summarized a growing consensus on the status of microfracture, its limitations, and potential complications.²,³ I am a long-time close personal friend of Dr. Bert and I know that no one ever leaves a conversation with him wondering about his opinion. This is exemplified in his article, and I quote; “it is my strong opinion that there is no indication to perform MF or abrasion arthroplasty for isolated articular cartilage lesions.” Just in case you missed this thrust of his argument, he concludes with “There is simply no justification in the literature to support the use of marrow-stimulation procedures, especially MF, at this time.” He provided supportive evidence from the literature.

There are many factors that contribute to the success or failure of microfracture and or arthroscopic abrasion arthroplasty. Let me count the ways...

Microfracture

I have never performed a microfracture because I never saw a lesion requiring it.

The Nature of the Lesion. The traumatic lesion is usually 2 cm or less in diameter with strong normal cartilage shoulders that protect a fibrin clot in the lesion. The depth of the traumatic lesion may be only partial thickness to the calcified cartilage layer or even to subchondral bone. Under tourniquet control or with intra-articular hydrostatic pressure, there appears to be no bleeding at the site, even from bone.

Availability of Fibrin Clot Matrix. However, there is an abundant source of blood in the joint for either type of lesion. The synovium bleeds and provides a blood clot for each and every pathologic site or ones that have been prepared surgically.¹ There will be a fibrin clot naturally and spontaneously filling the defect. The issue becomes whether the geometry of the lesion will house and hold a blood clot. The 3-dimensional geometry of the traumatic lesion with normal cartilage shoulders houses and holds a fibrin blood clot, thereby providing an autogenous matrix (Fig 1).

No Lack of Available Progenitor Cells. In addition, there is an abundance of progenitor cells available to the lesion. The traumatic lesion that penetrates to exposed bone, bleeds and brings in progenitor cells from the bone. The traumatic lesion only to the calcified layer has an abundant source of progenitor cells from the surgical portal lacerations of the synovium. In addition, the passage of instrumentation back and forth may course through the infrapatellar fat pad. The arthroscopic surgery may free synovial cells following a synovectomy,