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Author Reply to "Regarding 'Repair Augmentation of Unstable, Complete Vertical Meniscal Tears With Bone Marrow Venting Procedure: A Prospective, Randomized, Double-Blind, Parallel-Group, Placebo-Controlled Study'"

We gratefully thank the Editor for providing us the opportunity to respond to the letter by Wei et al. entitled "Regarding 'Repair Augmentation of Unstable, Complete Vertical Meniscal Tears With Bone Marrow Venting Procedure: A Prospective, Randomized, Double-Blind, Parallel-Group, Placebo-Controlled Study'".

The authors have explained their concerns about the bone marrow venting procedure in the state of meniscus healing. We appreciated their feedback on our study and the opportunity to discuss meniscus healing in reference to anterior cruciate ligament reconstruction and bone marrow element stimulation. Similar observations to ours were made in an independent study by Dean et al.[1] Additionally, at least two randomized clinical trials investigating the influence on meniscal healing of bone marrow venting procedures are ongoing (NCT05053646,NCT04775004).

As the authors said, studies have demonstrated correlation with better meniscus healing after ACL reconstruction compared with ACL-deficient knee.[2,3] We agree with the authors that reestablishment of proper knee kinematics after ACL reconstruction is correlated with better meniscus healing and lower retear rate. Some studies showed significant differences in anterior shift and external rotation during anterior tibial translation in ACL-deficient knee.[4,5] We are confident that changes of knee kinematics cause secondary microinjuries to knee structures (i.e., meniscus)[6,7] and thus, in our opinion, interrupt healing of the repaired meniscus.

We also agree with the authors that postoperative rehabilitation is an important factor influencing meniscal and cartilage healing. Many experimental studies provided evidence that changes in hydrostatic pressure load increases the production of proteoglycans and glycosaminoglycan[8] (in articular cartilage), as well as boosts the replication rate in chondrocytes.[9] On the contrary, variable rehabilitation protocols were studied (no weight bearing and restricted motion vs. full weight bearing and full range of motion), and no significant difference on the rate of successful meniscal healing was noted.[10,11] Indeed, rehabilitation principles have taken steps forward, but the ideal conditions allowing for the successful healing of a repaired meniscus remains to be fully elucidated.

We suggest that even a short release of growth factors (bone tunnel drilling, bone marrow venting procedures, or a growth factor injections) could provide increased healing potential. In animal models, Koch et al. showed macroscopical and histological enhanced regeneration of teared meniscus by single bone marrow aspirate concentrate injection.[12] Also, Abdel-Hamid et al. made a similar observation.[13] If a single injection of bone marrow aspirate significantly increases the potential of meniscal healing in animal models, the same mechanism could play a role in human meniscus healing. Girolamo et al. presented increased PDGF concentration after ACL reconstruction in joint fluid 30 min after the end of the surgical procedure.[14] Galliera et al. measured VEGF and VEGFR2 levels after 30 min of arthroscopy in knee joint fluid. They showed significantly higher levels of those factors in the ACL reconstruction group compared to the control group.[15] However, both studies quoted by the authors, have some weak points. The first is joint

drainage—allowing for "cytokine mixture" escape form the joint. The second is the collection of joint fluid sample at a single time point (30 minutes after the end of surgical procedure). Such results do not allow us to draw any conclusion on the process of growth factor concentration kinetics. On the contrary, the study by Beckmann et al., which evaluated abrasion arthroplasty provide additional data.[16] They studied mesenchymal stem cell content and growth factor concentration in postoperative joint effusions (samples were collected after 21 h ± 2.5 h postoperation). They noticed a significantly increased concentration of TGF-β, IGF-1, mononuclear cells, and abrasion arthroplasty released cells with phenotypes typical to mesenchymal stem cells. Interestingly, those cells, expanded in high-density cultures, showed positive staining for markers indicating the chondrogenic differentiation. Similarly, techniques allowing for recruitment of synovial mesenchymal cells is a synovial abrasion. One cannot expect that this technique will recruit a massive number of mesenchymal stem cells or cause a massive release of growth factors into the joint. Despite this, it is successfully applied into the clinical setting, allowing for meniscal healing even with no suture repair in selected patients.[17,18]

In our procedure described in the article,[19] we perform bone marrow venting as a last step during arthroscopy, with no drainage to the joint. We believe, this simple measure may prevent rinsing of the growth factors and cells released.

In conclusion, according to multiple studies, we believe that meniscal healing is a multifactorial process, equally requiring biologic and kinematic factors. Restoring proper knee biomechanics plays a pivotal role. Nowadays, there is contradictory data on the optimal rehabilitation protocol. We are convinced the best possible protocol should be guided by biomechanics, but more studies are necessary to resolve this issue. We also believe that proper surgical technique and appliance of additional techniques that may lead to significantly higher meniscus healing rate (such as bone marrow venting, recruitment of mesenchymal stem cells) is beneficial to the patient. Despite the progress in orthopedics and basic sciences, a lot of questions remain unanswered. Does every patient with a meniscal tear have disturbed knee kinematics? Do we require more sensitive diagnostic tools? What is the optimal timeframe for growth factors and mesenchymal stem cell release? Is a high concentration of these factors in joint fluid necessary? Or perhaps, the process of meniscal healing is more similar to wound healing or morphogenesis, with local gradient of growth factors that is interpreted by target cells, according to preset concentration thresholds? Further studies are required to enable us to understand the process of meniscal healing. Hopefully, new answers will benefit our patients achieving 100% meniscal healing rate with 0% of retears in the future.

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Regarding "Subacromial Decompression in Patients With Shoulder Impingement With an Intact Rotator Cuff: An Expert Consensus Statement Using the Modified Delphi Technique Comparing North American to European Shoulder Surgeons"

We read with great interest the study by Hohmann et al.,[1] "Subacromial Decompression in Patients With Shoulder Impingement With an Intact Rotator Cuff: An Expert Consensus Statement Using the Modified Delphi Technique Comparing North American to European Shoulder Surgeons." Undoubtedly, the management of patients with subacromial impingement (SI) is still controversial, expressed in this study by the fact that consensus could only be reached for 22% of the 71 Likert style items included.

We wish to congratulate the authors for the enormous effort involved in carrying out this consensus, and we would like to raise a few observations about the interpretation of the study results and the role of subacromial decompression (SAD) in patients with SI.

In 2009, Ketola et al.[2] performed a randomized controlled trial (RCT) including 134 patients in which they compared a supervised exercise program (n = 66) with arthroscopic acromioplasty followed by a supervised exercise program (n = 68). No statistically significant difference was found neither in relation to the visual analog scale score nor in the secondary outcomes considered, which were pain at night, disability, shoulder disability questionnaire score, number of days experiencing pain, and number of patients without pain. In a similar study, Farfaras et al.[3] randomized 55 patients with SI into open acromioplasty (n = 15), arthroscopic acromioplasty (n = 19), or physiotherapy (n = 21) treatment, and the authors also found no significant differences between the 3 groups in a period up to 3 years after the intervention.[3] Some recent RCTs were performed, including a placebo surgery control group.[4,5]

In 2018, Beard et al.[4] performed a multicenter, randomized, placebo-controlled, 3-group trial in 32 British hospitals. They included 313 patients who were randomly assigned to 1 of 3 treatment groups: (1) decompression surgery (n = 106), (2) diagnostic arthroscopy only (n = 103), and (3) no treatment (n = 104). No clinically significant difference was found by the authors in terms of pain or functional scores when comparing the surgical groups with the no-treatment group. Moreover, surgical decompression did not result in any additional positive effect when compared with arthroscopy only.

In 2018, Paavola et al.[5] performed another multicenter, 3-group, randomized, double-blind, sham-controlled trial in 3 Finnish public hospitals. They included 210 patients suffering from SI who were randomly classified into 1 of the following 3 treatment groups: (1) decompression surgery (n = 59), (2) diagnostic arthroscopy only (n = 63), and (3) exercise therapy (n = 71). They found that SAD was not any better than diagnostic arthroscopy at 24 months. Likewise, there appear to be no long-term benefits associated with SAD in patients with SI. In 2017, Ketola et al.[6] published the long-term outcomes from their previously published RCT in 2009.[2] From the initial 134