Stabilizing the posteriorly Unstable shoulder: Identifying engagement patterns and thresholds of bone grafting in bipolar lesions

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INTRODUCTION: Posterior shoulder instability is an uncommon but challenging condition, frequently associated with bipolar bone loss involving the posterior glenoid and reverse Hill-Sachs lesions (RHSLs). Clinical outcomes after isolated soft tissue repair are poor when significant bone loss is present, and failure to recognize critical thresholds for bone grafting may result in recurrent instability. While anterior instability has well-established concepts of engagement and "on-track/off-track" mechanics, the biomechanical consequences of posterior bipolar lesions remain poorly defined. In particular, the relationship between lesion size, engagement patterns, and the ability of bone grafting to restore stability is not fully understood. The purpose of this study was to systematically characterize posterior engagement across progressive states of glenoid bone loss and RHSLs, and to determine thresholds at which distal tibia and talus allograft reconstructions restore stability. We hypothesized that posterior dislocation resistance would diminish in proportion to the extent of bone loss and lesion engagement, and that bone grafting procedures would restore stability toward native levels.

METHODS: Eight fresh-frozen cadaveric shoulders were dissected free of all soft tissue (mean age: 62.3 range:56-65, 5 females, 3 males); seven have been tested at the time of submission. Each specimen was mounted to a 6-degree-of-freedom robotic arm and sequentially tested across 11 conditions: 1) Native, 2) Posterior labrum removed, 3) Small Posterior Glenoid Bone Loss (GBL, 12.5%) 4) Large GBL (25%), 5) GBL reconstructed with Distal Tibia Allograft (DTA) 6) GBL + small reverse Hill-Sachs lesion (RHSL) 7) Large GBL + small RHSL, 8) Large GBL + large RHSL 9) Large GBL + large RHSL 10) DTA + large RHSL 11) DTA + RHSL reconstructed with Talus Allograft (TA). Posterior dislocation testing was performed at neutral, 45°, and 90° of shoulder flexion with the arm internally rotated. The humeral head was translated postero-inferiorly along the 7 o'clock axis of the glenoid clockface by one humeral head radius, while maintaining an 80 N compressive load, over 15 seconds. Outcomes were: 1) maximum postero-inferior force (N), and 2) maximum lateral translation (mm) as the humeral head passed over the glenoid rim. Dynamic flexion testing involved robotic motion from 0°–90° of flexion over 60 seconds, with 50 N of compressive load and no applied sagittal plane force. This allowed the joint to follow its natural path. The outcome was posterior humeral head translation, indicating whether lesions engaged and caused the joint to go "off-track.". To match the repeated measures study design, a two-factor linear mixed effects model will be established for the dislocation tests with using glenoid bone state and humeral bone state as categorical factors with 4 levels (native, small bone loss, large bone loss, reconstruction). The dynamic flexion test will be analyzed using statistical parametric mapping. ANOVA analysis will determine the significance of each factor, and post-hoc pairwise comparisons will be used to establish significant differences. Currently, the data is presented as means and standard deviations.

RESULTS: The results for the 90° dislocation tests are shown on figure 1, and the analysis for the dynamic flexion test is underway. The results are presented in the natural order for data interpretation, which differed slightly from the order of testing. Glenoid bone loss caused progressive loss of posterior shoulder stability as demonstrated by decreased dislocation force and lateral humeral head translation, and glenoid reconstruction with DTA restored shoulder stability to near-native levels. RHSLs caused progressive loss of posterior shoulder stability, which was partially restored by Talar Allograft reconstruction.

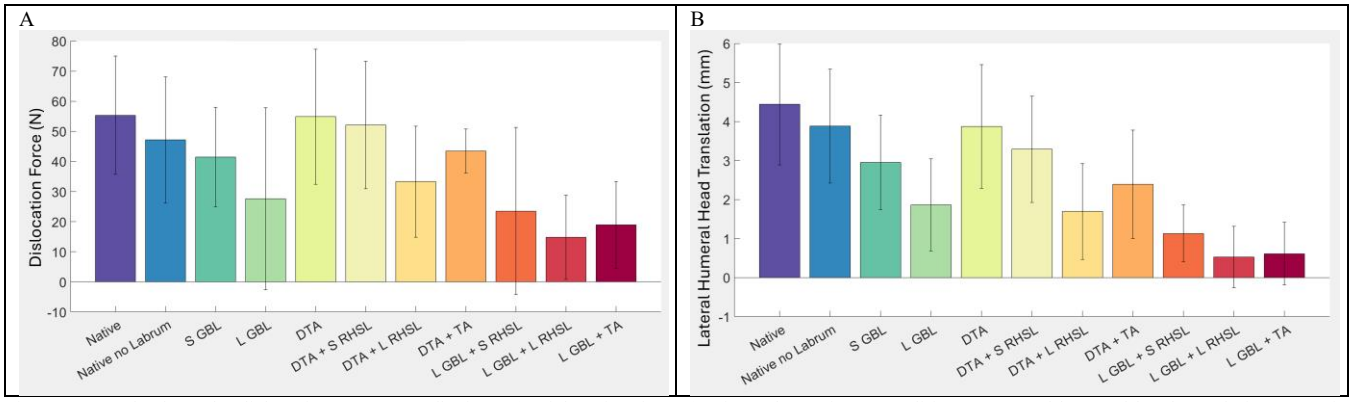

Figure 1: Results from the dislocation test at 90° flexion. A) Dislocation force (N) in each specimen condition B) Lateral humeral head translation (mm) in each specimen condition. Error bars represent 1 standard deviation.

DISCUSSION: This study demonstrates that posterior shoulder stability is progressively compromised by increasing magnitudes of glenoid bone loss and RHSL size. Both dislocation resistance and humeral head translation were adversely affected in bone loss states, confirming that bipolar lesions act synergistically to destabilize the joint. Reconstruction of the glenoid with distal tibia allograft effectively restored stability to near-native levels, while talus allograft for RHSLs provided partial restoration. These findings mirror established principles of anterior instability, suggesting that posterior bipolar lesions also follow predictable engagement patterns that can be quantitatively characterized. Importantly, this investigation provides the first robotic analysis of posterior engagement thresholds across a range of clinically relevant lesion sizes and reconstructions. Although ongoing dynamic flexion analyses will further define the engagement envelope, the current data suggest that bone grafting procedures are biomechanically justified when glenoid bone loss exceeds 12.5% or when combined bipolar lesions cause the shoulder to go "off-track". Limitations include cadaveric variability and the absence of dynamic stabilizers such as the capsule and rotator cuff, but the robotic testing model isolates the contribution of osseous anatomy to stability.

CLINICAL RELEVANCE: This study provides quantitative values to characterize loss of stability based on the severity of bony defects and demonstrates that glenoid and humeral reconstructions can restore stability in shoulders with significant bone loss. These findings support the use of structural allografts in the management of posterior bipolar lesions and provide a biomechanical foundation for guiding surgical treatment algorithms.