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SYMPOSIUM: COMPLEX ISSUES IN GLENOHUMERAL INSTABILITY

A Large Humeral Avulsion of the Glenohumeral Ligaments Decreases Stability That Can Be Restored With Repair

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Abstract

Background Humeral avulsion of the glenohumeral ligaments (HAGL) has become a recognized cause of recurrent shoulder instability; however, it is unknown whether small and large HAGL lesions have similarly destabilizing effects and if large lesion repair results in restoration of stability.

Questions/purposes In a cadaver model, we evaluated the effect of small and large HAGL lesions and large HAGL lesion repair on glenohumeral ROM, translation, and kinematics.

Methods We measured rotational ROM, humeral head translation under load, and humeral head apex position in eight cadaveric shoulders. Each specimen was tested in 60° glenohumeral abduction in the scapular and coronal planes under four conditions: intact, small HAGL lesion (mean ± SD length, 18 ± 1.8 mm), large HAGL lesion (36.8 ± 3.6 mm), and after large HAGL lesion repair. For each condition, we measured maximum internal and external rotation with 1.5 Nm of torque; glenohumeral translation in 90° external rotation with 15- and 20-N force applied in the anterior, posterior, superior, and inferior directions; and humeral head apex position throughout ROM. Repeated-measures ANOVA was used for statistical analysis.

Results Small HAGL lesions did not change ROM, translation, or kinematics from the normal shoulder; however, these parameters changed with large HAGL lesions. Maximum external rotation and total ROM increased in the scapular (13.8° ± 9.4°, $p < 0.001$; 19.0° ± 16.5°, $p < 0.001$) and coronal (21.4° ± 10.6°, $p < 0.001$; 29.1° ± 22.1°, $p < 0.001$) planes. With anterior force, anterior-inferior translation increased in both planes (mean increase for both loads and planes: anterior: 9.1 ± 9.5 mm, $p < 0.01$; inferior, 5.7 ± 6.6 mm, $p < 0.03$). In the coronal plane, posterior and inferior translation also increased (4.9 ± 5.4 mm, $p < 0.01$; 7.1 ± 9.9 mm, $p < 0.03$; averaged for both loads). The humeral head apex shifted 3.7 ± 4.9 mm anterior ($p = 0.04$) and 2.8 ± 2.6 mm lateral ($p = 0.004$) in the scapular plane and 3.7 ± 3.4 mm superior ($p = 0.006$) and 4.1 ± 2.6 mm lateral ($p < 0.001$) in the coronal plane. HAGL lesion repair decreased ROM and translation in both planes and restored humeral head position in maximum external rotation.

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Conclusions Anterior large HAGL lesions increase ROM and glenohumeral translation. After large HAGL lesion repair, stability of the shoulder can be restored.
Clinical Relevance Surgeons should be aware of the possibility of HAGL lesions in patients with shoulder instability, and if large HAGL lesions are diagnosed, surgeons should consider repairing the lesions.

Introduction

The inferior glenohumeral ligament is an important static stabilizer of anterior translation and external rotation in the abduction position [14, 21, 24]. Failure of the inferior glenohumeral ligament can occur with anterior shoulder dislocation, most commonly with avulsion of the glenoid insertion of the inferior glenohumeral ligament, or Bankart lesion; however, while more rare, humeral avulsion of the glenohumeral ligaments (HAGL) can also occur. Regarding the mechanism of injury that leads to a HAGL lesion, Nicola [13] found, in a cadaveric study, that a HAGL lesion resulted with the arm in 105° of hyperabduction and external rotation whereas a Bankart lesion occurred when the arm was hyperabducted and compressed. McMahon et al. [11], reporting on failure modes of the inferior glenohumeral ligament with tensile testing of 12 specimens, found that eight failed at the site of the glenoid insertion, representing a Bankart lesion, two at the humeral insertion, representing a HAGL lesion, and two at the midsubstance. Any disruption of the inferior glenohumeral ligament can potentially lead to increased laxity and propensity for dislocation.

The HAGL lesion may occur in isolation or, more commonly, may be associated with other abnormalities, such as a rotator cuff tear, Bankart lesion, Hill-Sachs lesion, or labral tear [6]. In 1942, Nicola [13] first described the avulsion of the capsule from the neck of the humerus in four of five acute dislocations that were surgically explored. In 1988, Bach et al. [2] reported two cases of the HAGL lesion and produced restorative results by repairing the lateral capsule. HAGL lesions have become a recognized cause of recurrent shoulder instability, reported in 2% to 9% of patients with shoulder instability [5, 17, 23, 25]. Recognizing HAGL lesions during shoulder arthroscopy can by difficult [17], which may contribute to the variability in its reported incidence. Two previous biomechanical studies have been performed evaluating glenohumeral stability with HAGL lesions. Pouilart and Gagey [15] reported that a large HAGL lesion covering three of four zones of the capsule must be present for glenohumeral dislocation to occur. More recently, Southgate et al. [19], in a cadaveric study, reported increased anterior translation with a large anterior HAGL lesion that was restored with repair. Their study was limited to anterior translation results only and did not measure translation in multiple directions, ROM, or kinematics. Despite the increasing awareness of the HAGL lesion, it is unknown whether small and large HAGL lesions have similarly destabilizing effects and whether repair of large lesions will result in restoration of stability.

We therefore biomechanically evaluated the effects of small HAGL lesions, large HAGL lesions, and repair of large HAGL lesions on glenohumeral ROM, translation, and kinematics in a cadaveric model.

Materials and Methods

Specimen Preparation

We used eight fresh-frozen human cadaveric shoulders, five female and three male, with a mean age of 68 years (range, 44–86 years). Specimens found on dissection to have preexisting pathology, such as limited ROM, osteoarthritis, or a rotator cuff tear, were excluded from the study. The specimens were stored at −20° C until the day before testing and thawed overnight at room temperature in preparation for dissection and testing. The specimens were kept moist with physiologic saline solution to prevent dehydration. All soft tissues were removed except the glenohumeral joint capsule, the coracoacromial ligament, the coracohumeral ligament, and the biceps tendon. The glenohumeral joint was vented by a small incision through the rotator interval to isolate the influence of intraarticular pressure.

The scapula was potted in a rectangular aluminum potting box using plaster of paris, aligning the glenoid surface parallel to the long and short axes of the box. The humerus was potted in an 18-cm-long, 38-mm-diameter polyvinylchloride pipe, aligning the humeral intramedullary axis with the long axis of the pipe. After the plaster set, two screws were drilled through the pipe and the humerus to ensure rigidity. To measure glenohumeral translation and kinematics, six screws were used as markers for digitizing points to define local coordinate systems on the humerus and glenoid. Specifically, the screws were placed on the coracoid, the anterior acromion, the posterior acromion, the proximal biceps groove, the distal biceps groove, and the mid biceps (posterior and in between the proximal and distal biceps groove).

Shoulder Testing System

The shoulder was mounted on a custom testing system (Fig. 1). The scapular box was mounted onto a bearing and lever arm system that was attached to the top of two bidirectional translation plates. The humeral polyvinylchloride pipe was inserted into a holder attached to the arc

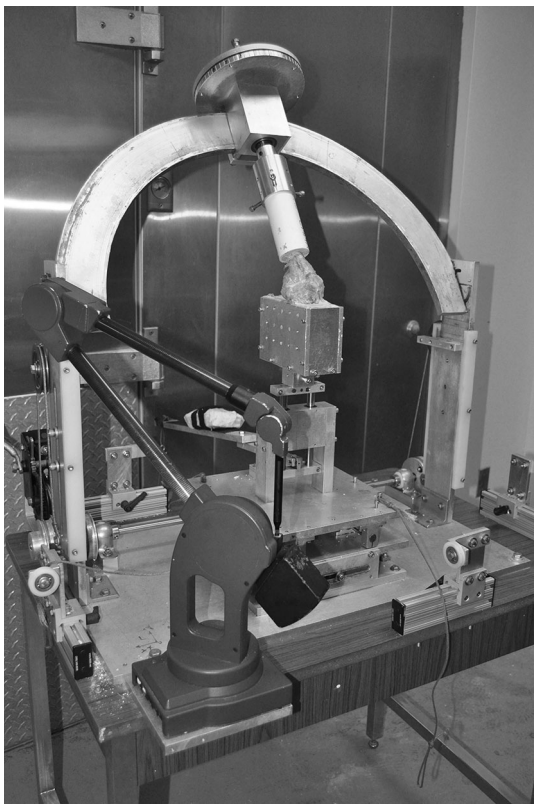

Fig. 1 A photograph shows the custom testing system with MicroScribe®.

of the testing system. A 22-N compressive force was applied across the glenohumeral joint. This amount of compressive force is adequate to keep the glenohumeral joint located and provide a consistent centering point of the humerus on the glenoid [16, 20, 22]. The glenohumeral joint was positioned at 60° of glenohumeral abduction to simulate 90° of shoulder abduction, accounting for a 2:1 ratio of glenohumeral to scapulothoracic abduction. This position was maintained through the entirety of testing. The humerus was placed in two positions for testing: in the scapular plane and in the coronal plane, which was set as 20° of horizontal abduction from the scapular plane, simulating the position of apprehension. We defined 90° of external rotation by aligning the anterior edge of the bicipital groove with the anterior edge of the acromion.

Testing Conditions

Each specimen was tested in four conditions: intact, small HAGL lesion, large HAGL lesion, and after repair of the large HAGL lesion. After testing the intact condition, the small HAGL lesion was created. An anterior HAGL was tested in this study since it is the most common type of HAGL lesion [5]. The diameter of the humeral head was measured using a digital caliper placed extraarticularly at the articular margin. The circumference was calculated, and the humeral head was then divided into 12 equal sections, simulating a clock face. The 6:00 position of the clock face was defined as the most inferior point on the humeral head. To verify this 6:00 position, the joint was distracted and the 6:00 position of the glenoid was identified. The humerus was then rotated until the capsular fibers were in line from the glenoid to the humeral head. The capsular fibers were then traced from the 6:00 position on the glenoid to the humeral head to define the 6:00 position of the humeral head.

Since HAGL lesions are most commonly seen in the anterior-inferior capsular region [1, 5], where the anterior band of the inferior glenohumeral ligament attaches, we chose to create anterior small and large HAGL lesions. The small HAGL lesion was made by cutting the insertion of the capsule from 6 to 7:30 o'clock on the right humeral head clock face and the large HAGL lesion was made by cutting an additional section anterior from the 7:30 position to the 9 o'clock position. Therefore, the total large HAGL lesion ranged from 6 to 9 o'clock on a right humeral head clock face. The length of each lesion was measured using a caliper. The mean ± SD sizes of the small and large HAGL lesions were 18.4 ± 1.8 mm and 36.8 ± 3.6 mm, respectively.

The large HAGL lesion was repaired using three single-loaded anchors (Arthrex, Inc, Naples, FL, USA) placed equidistantly on the capsule insertion site. All devices have been FDA approved for use as described. The length of the large HAGL lesion was divided by four to determine how far apart the anchors should be placed. A 5-mm bite size from the margin of the capsule was taken. Simple suture technique and SMC knot tying were used to repair the lesion.

Measured Parameters

Before taking measurements in each condition, the specimens were preconditioned in internal and external rotation 10 times using a torque wrench to a torque of 1.5 Nm. Translation was also preconditioned with a force of 15 N, 10 times in the anterior-posterior direction and 10 times in the superior-inferior direction. Maximum internal and external rotation was measured with a torque of 1.5 Nm. Glenohumeral kinematics throughout the rotational ROM were then measured by recording the position of the humerus and scapula by digitizing the six bony markers with a MicroScribe® 3DLX digitizer (Revware, Raleigh, NC, USA) at maximum internal and external rotation and at 0°, 30°, 60°, and 90° of external rotation in the scapular and coronal planes. Glenohumeral translations were

measured with the humerus locked in 90° of external rotation by applying 15 N and 20 N in the anterior, posterior, superior, and inferior directions in both the scapular and coronal planes. The positions of the scapula and humerus at the end point of translation were recorded with the MicroScribe® 3DLX digitizer.

After all testing procedures, we carefully disarticulated the specimens and digitized the humeral head and glenoid geometry relative to the local coordinate systems of each bone using the MicroScribe® 3DLX digitizer to calculate the position of the humeral head apex with respect to the geometric center of the glenoid [10]. The humeral head apex was defined as the farthest point on the articular surface of the humeral head from a plane defined by the articular margin. The geometric center of the glenoid was defined as the center of the superior-inferior and anterior-posterior glenoid axes at the level of the articular surface. During testing, all measurements were performed twice to ensure reproducibility. If the values were not repeatable to within 3° or 1 mm, the measurements were repeated. The mean values of the two most repeatable measurements were then used for comparison. We used a repeated-measures ANOVA with a Tukey post hoc test (Statistica®; StatSoft, Tulsa, OK, USA) to determine significant differences. The level of significance was set at p values of less than 0.05.

Results

The small HAGL lesion did not change glenohumeral ROM, translation, or kinematics compared to normal.

Maximum external rotation and total rotational ROM increased with the large HAGL lesion in both the scapular plane (13.8° ± 9.4°, $p < 0.001$; 19.0° ± 16.5°, $p < 0.001$) (Fig. 2) and the coronal plane (21.4° ± 10.6°, $p < 0.001$; 29.1° ± 22.1°, $p < 0.001$) (Fig. 3). Repairing the HAGL lesion decreased maximum external rotation and total ROM in both the scapular plane ($p < 0.001$ and $p < 0.001$) and the coronal plane ($p < 0.001$ and $p = 0.003$).

When an anterior force was applied, anterior-inferior translation increased with the large HAGL lesion in the scapular (Fig. 4) and coronal (Fig. 5) planes ($p < 0.05$). After repairing the large HAGL lesion, anterior and inferior translation decreased to levels of the intact shoulder in both the scapular and coronal planes. Posterior and inferior translation in the coronal plane also increased with a large HAGL lesion (4.9 ± 5.4 mm, $p < 0.01$; 7.1 ± 9.9 mm, $p < 0.03$; averaged for 15- and 20-N translational loads), which was restored to the translation of the intact shoulder when repaired (Fig. 6).

Abnormal shift in the humeral head apex position was seen with the large HAGL lesion only in maximum external rotation. This shift was 3.7 ± 4.9 mm anteriorly ($p = 0.04$) and 2.8 ± 2.6 mm laterally ($p = 0.004$) in the scapular plane (Fig. 7) and 3.7 ± 3.4 mm superiorly ($p = 0.006$) and 4.1 ± 2.6 mm laterally ($p < 0.001$) in the coronal plane (Fig. 8) at maximum external rotation compared to intact. These abnormal shifts in the humeral head apex position at maximum external rotation were restored after repair.

Discussion

As first described by Nicola [13], the HAGL lesion is an avulsion of the capsule from the neck of the humerus. HAGL lesions have become a recognized cause of recurrent shoulder instability, reported in 2% to 9% of patients with shoulder instability [5, 17, 23, 25]. Despite the increasing awareness of the HAGL lesion, it is unknown whether small and large HAGL lesions have similarly destabilizing effects and whether repair of large lesions will result in restoration of stability. In this study, glenohumeral ROM, translation, and kinematics did not change with a small HAGL lesion; however, a large HAGL lesion increased both ROM and glenohumeral translation in the anterior-inferior, posterior, and inferior directions and caused an abnormal shift of the humeral head in maximum external rotation. The abnormal biomechanics seen with a large HAGL lesion were reversed when the HAGL lesion was repaired.

One limitation of this study is that it was performed on normal cadaveric specimens, which do not have the redundant capsular tissue that patients with clinical instability have. Although cadaveric biomechanical studies have been an effective method to study simulated injuries, these models and testing systems cannot recreate the multiple factors that act in synergy in vivo to provide glenohumeral stability. The use of a cadaveric model has multiple limitations, including tissue stretching and the absence of dynamic muscle forces, joint proprioception, and coordination. The advanced ages of our cadaveric specimens may be a confounding variable in correlating our findings to the shoulder of a younger overhead athlete. However, previous studies in our laboratory have demonstrated that the biomechanical properties of the soft tissues in the glenohumeral joint may be similar between young and old cadaver specimens within the functional ROM [9]. We only evaluated anterior HAGL lesions in this study, since HAGL lesions have been reported to be more commonly located in the anterior-inferior aspect of the glenohumeral joint [1, 5]. Further studies should be performed to evaluate different HAGL lesion locations. No differences were found after creation of a small HAGL lesion compared to intact. These findings may have been a result of low power to detect small

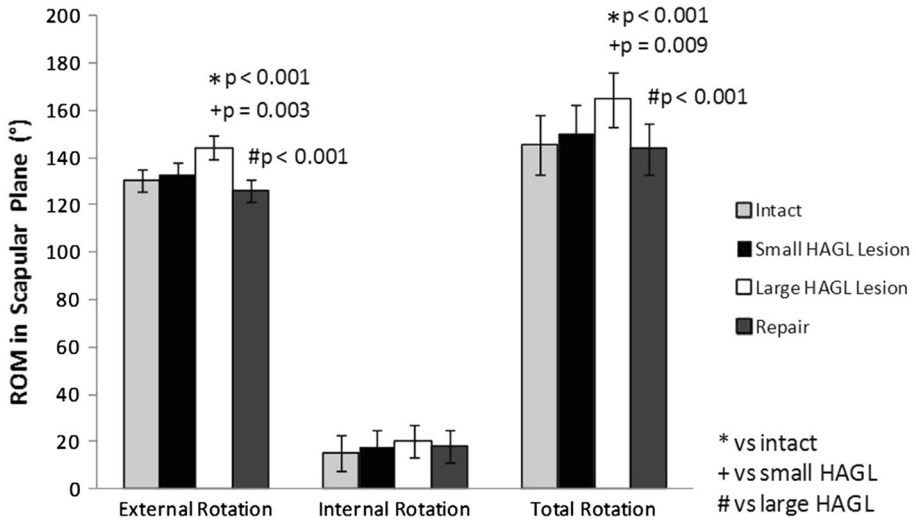

Fig. 2 A graph shows rotational ROM in the scapular plane. Maximum external rotation and total rotational ROM increased with the large HAGL lesion in the scapular plane.

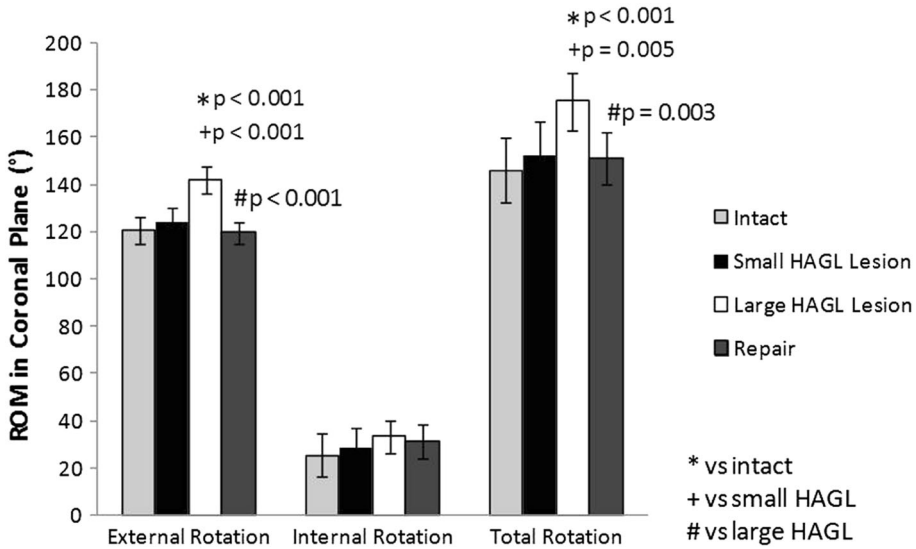

Fig. 3 A graph shows rotational ROM in the coronal plane. Maximum external rotation and total rotational ROM increased with the large HAGL lesion in the coronal plane.

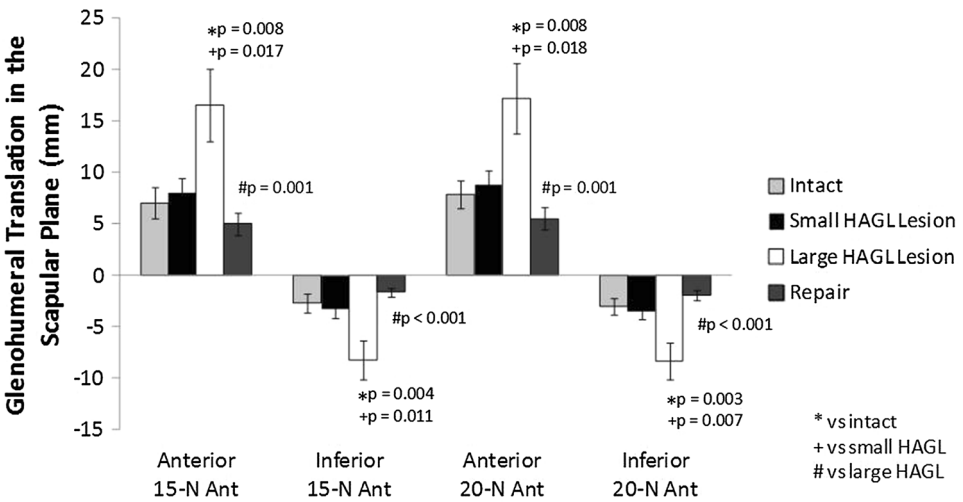

Fig. 4 A graph shows anterior-inferior glenohumeral translation in the scapular plane with a 15-N and 20-N anterior force applied. Anterior-inferior translation increased with the large HAGL lesion in the scapular plane. Ant = anterior.

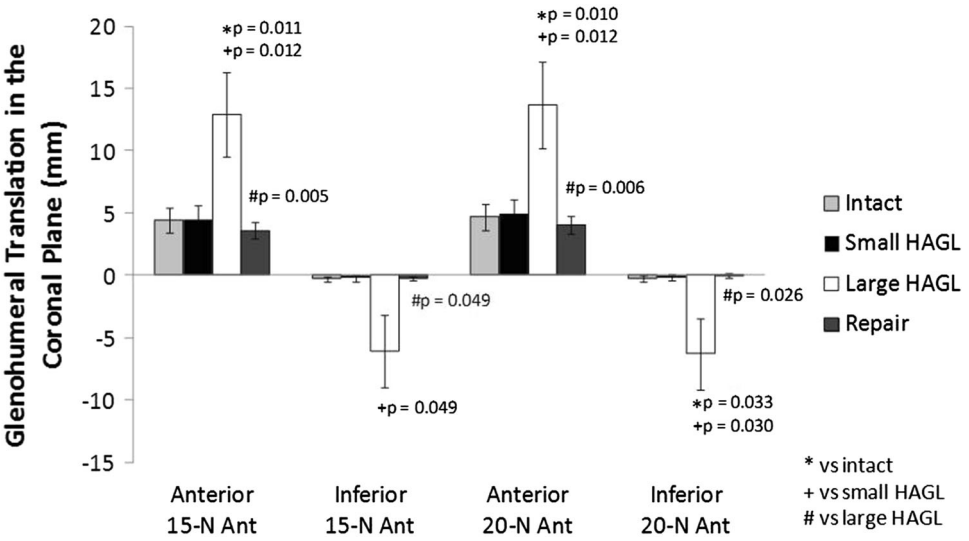

Fig. 5 A graph shows anterior-inferior glenohumeral translation in the coronal plane with a 15-N and 20-N anterior force applied. Anterior-inferior translation increased with the large HAGL lesion in the coronal plane. Ant = anterior.

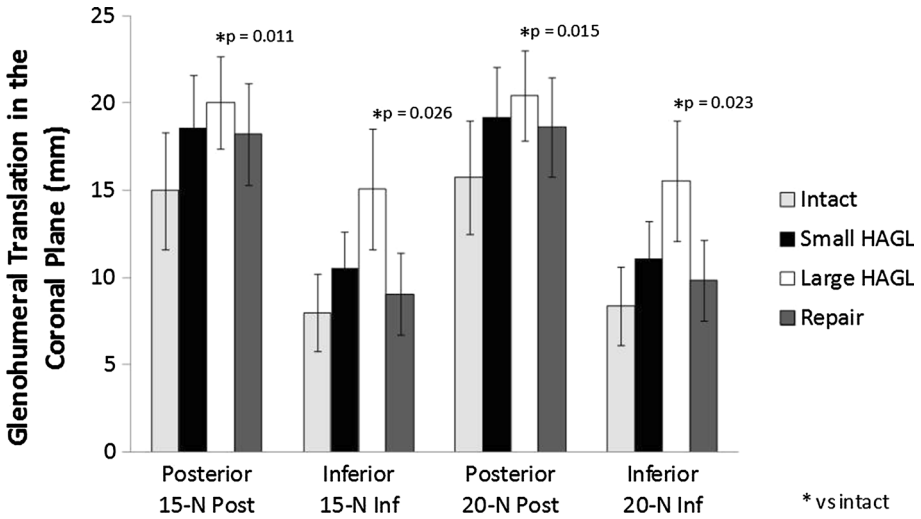

Fig. 6 A graph shows posterior and inferior glenohumeral translation in the coronal plane with a 15-N and 20-N posterior or inferior force applied. Posterior and inferior translation increased with the large HAGL lesion in the coronal plane. Post = posterior; Inf = inferior.

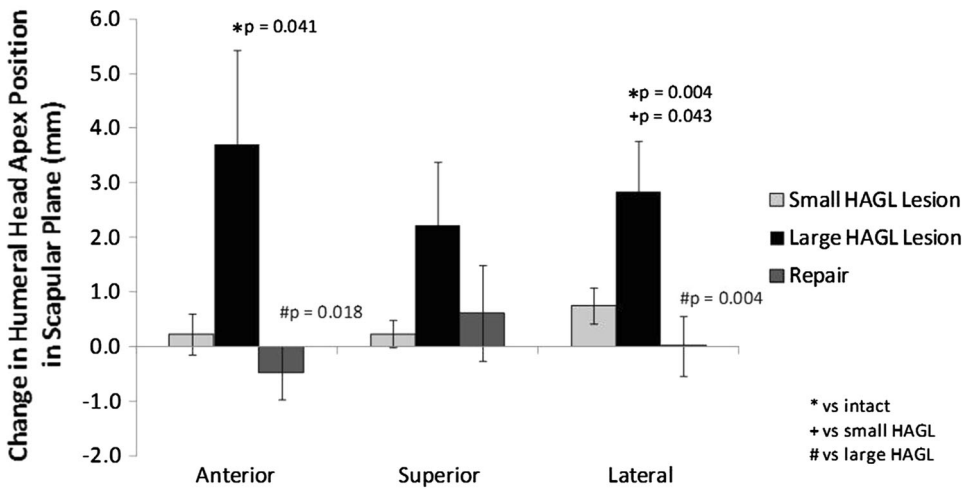

Fig. 7 A graph shows the change in humeral head apex position from intact at maximum external rotation in the scapular plane. The humeral head apex with the large HAGL lesion shifted anteriorly and laterally in the scapular plane at maximum external rotation.

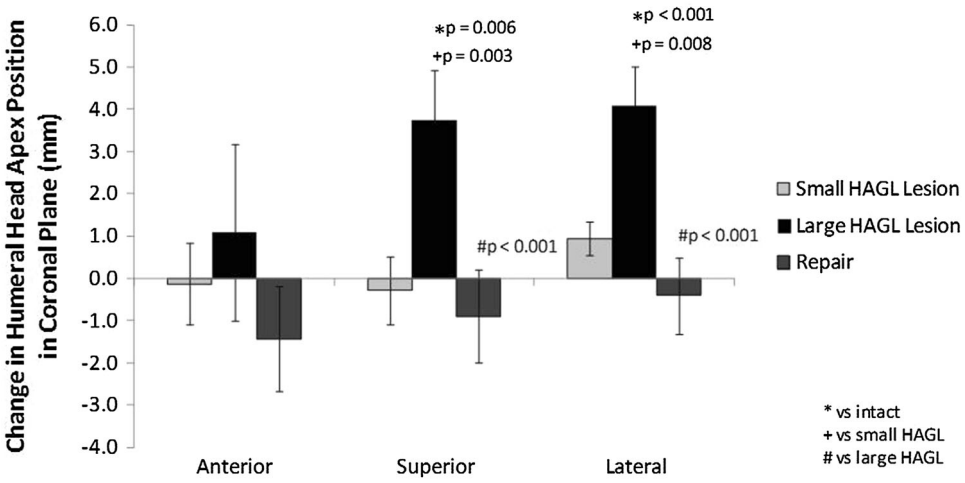

Fig. 8 A graph shows the change in humeral head apex position from intact at maximum external rotation in the coronal plane. The humeral head apex with the large HAGL lesion shifted superiorly and laterally in the coronal plane at maximum external rotation.

differences; however, there were no comparisons that were approaching statistical significance. The small number of specimens tested also did not allow for comparisons based on sex. Even though female shoulders are generally more lax than male shoulders clinically, we do not believe sex would play a role in changes in laxity after a capsular lesion in this model isolated to bony and soft tissue restraints.

In this study, no changes in ROM or translation were observed with the small HAGL lesion. This finding is similar to previous biomechanical studies on HAGL lesions that show dislocation only occurring when three capsular zones are violated [15]. With a small anterior HAGL lesion, only a portion of the inferior glenohumeral ligament is released; therefore, the superior and posterior portions of the inferior glenohumeral ligament are still able to provide stability. A cadaveric study by Southgate et al. [19] recently evaluated medium and large HAGL lesions. They reported increased anterior translation with a large HAGL lesion and no change with a medium HAGL lesion, similar to our findings. The location of the HAGL lesions in their study was slightly different from that in our study, with their large HAGL lesion extending slightly more posterior on the clock face from 3:30 to 6:30 compared to 3 to 6 o'clock. The HAGL lesion extending more posteriorly may have influenced inferior and posterior translation to a greater degree than in this study. However, they did not report changes in glenohumeral translation in directions other than anteriorly or changes in ROM.

The increases in glenohumeral translation and ROM seen with a large HAGL lesion observed in this study were greater than those found in previous biomechanical studies of Bankart lesions corresponding to similar clock face positions [3, 7, 20]. By removing the capsule from its attachment on the humerus relative to the clock face position, a much larger lesion is created as compared to the glenoid. In our study, the mean length of the large HAGL lesion was approximately 37 mm, which is about the mean superior-inferior dimension of the glenoid [8, 18].

The glenohumeral kinematics are represented by tracking the humeral head apex relative to the geometric center of the glenoid. We chose to track this point on the humeral head as it is most closely located to the glenoid and simulates the path of articulation of the humeral head with the glenoid. Since this point is not located within the center of axial rotation of the humerus, its location can be affected by changes in rotation position. The humeral head apex position was altered with a large HAGL lesion in positions of external rotation, anterior-lateral in the scapular plane and superior-lateral in the coronal plane, possibly due to increased external rotation for this condition. These data may also suggest that without the intact capsule anteriorly the humeral head shifts from its normal position in maximum external rotation and is subluxing on the anterior rim of the glenoid. The kinematics at other rotation positions were not affected, most likely due to the influence of the bony geometry dictating the position of the humeral head when not in extreme ROMs. After repairing the HAGL lesion, the capsule repair was able to keep the humeral head in its normal position in maximum external rotation.

Studies disagree over the likelihood of a HAGL lesion in a patient with shoulder instability. In 1995, Wolf et al. [25] reported the occurrence of a HAGL lesion in six of 64 shoulders (9% of patients) undergoing arthroscopy and treatment of anterior shoulder instability. They insisted that, in patients with documented anterior shoulder instability without a demonstrable primary Bankart lesion, a HAGL lesion should be ruled out. In 1999, Bokor et al. [4] reported a HAGL lesion in 7.5% of their patients with anterior shoulder instability and in 26.9% of patients with anterior instability without a Bankart lesion and stressed that a traumatic anterior dislocation that did not have a Bankart lesion must be assumed to have a HAGL lesion. In 2007, Rhee and Cho [17] insisted that, during an operation to treat anterior shoulder instability, a thorough examination for not only a Bankart lesion but also other associated

lesions including a HAGL lesion is considered necessary to lower the risk of redislocation and that a HAGL lesion can be easily overlooked.

The difficulty of diagnosis may be one of the reasons why a low rate of HAGL lesions is reported. The arthroscopic finding of an exposed subscapularis muscle should alert the surgeon to the possibility that a HAGL lesion exists and can potentially cause shoulder instability. Another possible reason for a low reported rate of HAGL lesions may be the possibility of their healing. Murphy et al. [12] reported three cases of HAGL lesions seen initially on MRI and subsequently resolving on followup imaging. Yiannakopoulos et al. [26] reported that all HAGL lesions were found in acute cases but not in chronic cases, which may be due to the healing of HAGL lesions in chronic cases.

Despite its rarity in clinical diagnoses, a large HAGL lesion in this cadaveric model does have important effects on glenohumeral stability, specifically rotational ROM, translation, and humeral head apex position, and repair of the lesion can return these values to those of the intact joint. Surgeons should be aware of the possibility of a HAGL lesion in patients with shoulder instability and if diagnosed should consider repairing the lesion. Future studies evaluating effects of HAGL lesions in different arm positions or different sizes and locations of HAGL lesions are needed to increase our understanding of these lesions.

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