INTRODUCTION

The anteroinferior glenohumeral capsule (anteroinferior band of the inferior glenohumeral ligament (AB-IGHL), axillary pouch) limits anterior translation, particularly in positions of external rotation, and as a result is frequently injured during anterior dislocation. [1,2] A common capsular injury is permanent tissue deformation, however, the extent and effects of this injury are difficult to evaluate as the deformation cannot be seen using diagnostic imaging. In addition, clinical exams to diagnose this injury are not reliable [3] and poor patient outcome still exists following repair procedures. [4] Previous experimental models have observed increased joint mobility following permanent tissue deformation. [5] While other models have quantified the permanent deformation using nonrecoverable strain [6], no model has correlated the amount of tissue damage to altered capsule function. Understanding the relationship between the extent of tissue damage and changes in capsule function following anterior dislocation could aid surgeons in diagnosing and treating anterior instability. Therefore, the objectives of this work were to 1) quantify the nonrecoverable strain in the anteroinferior capsule resulting from an anterior dislocation and 2) evaluate capsule function (strain distribution in anteroinferior capsule, anterior translation) during a simulated clinical exam at three joint positions, in the intact and injured joint.

METHODS

Six cadaveric shoulders (71±8 yrs.) were dissected down to the glenohumeral capsule and the maximum anterior-posterior (AP) glenoid width was measured using digital calipers. A 7x11 grid of strain markers was then fixed to the anteroinferior capsule dividing it into 60 elements. The humerus and scapula were fixed in epoxy putty and each joint was mounted to a robotic/universal force-moment sensor testing system that was used to apply external loads and torques to the humerus. [7] The 3D positions of the strain markers were recorded in the reference position, which was determined via inflation, using a three-camera motion tracking system (Spicatek, accuracy: 0.05mm). [8] Simulated clinical exams were then performed on each intact shoulder at 60° abduction (Ab), and 0°, 30° and 60° of external rotation (ER) by applying a 25N AP load to the humerus while maintaining a 22N compressive load to center the humeral head on the glenoid. The resulting kinematics of the intact joint were recorded. The 3D positions of the strain markers were recorded along the paths of motion where the 25N anterior load was applied.

Each joint was dislocated at 60° Ab and 60° ER by applying a maximum of 300N anterior load to the humerus, while maintaining the 22N compressive load. The humeral head was allowed to move until the anterior translation reached one half the maximum AP width of the glenoid plus three millimeters. Following dislocation, the joint was returned to 60° Ab, 0° ER and allowed to recover for 30 minutes. [9] The capsule was then re-inflated in the reference position and the 3D positions of the strain markers were recorded to serve as the nonrecoverable strain state. Finally, the simulated clinical exams were repeated on the injured joint at 60° Ab and 0°, 30° and 60° of ER and the resulting kinematics and strain marker positions were recorded.

Maximum principle strain was computed by comparing the 3D marker positions in the intact and injured joint using the finite element solver ABAQUS. The strain distributions between the intact and injured states at 0°, 30° and 60° ER were compared for each shoulder using projection plots. [10] For each distribution, the strain values were grouped into 100 quantiles and projection plots were created by plotting quantile difference values (injured - intact) vs. average quantile values (paired intact and injured). The range of the...
projection plot was defined as the difference between the maximum and minimum quantile difference and quantified differences between the intact and injured strain distributions at 0°, 30° and 60° ER. Therefore, higher range values are indicative of greater differences between two strain distributions. Spearman rank correlation coefficients were computed between the range of the projection plots at 0°, 30° and 60° ER and the peak nonrecoverable strain in the anteroinferior capsule. Anterior translations were compared between the intact and injured joint at 0°, 30° and 60° of ER using a paired t-test. Significance was set at α = 0.05 for all analyses.

RESULTS
The amount of nonrecoverable strain in the anteroinferior capsule resulting from anterior dislocation varied greatly between shoulders with peak values of 46.5%, 11.8%, 7.5%, 9.0%, 7.3%, and 5.5% in each shoulder demonstrating localized areas of increased tissue damage. Further, the peak nonrecoverable strain in the axillary pouch (12.8 ± 15.7%) was greater than the AB-IGHL (6.1 ± 3.6%).

The projection plots demonstrated differences in the strain distributions, with the 25N anterior load applied, between the intact (Figure 1A) and injured (Figure 1B) states at all three joint positions. Range values increased from 0° (9.6 ± 9.2%) to 30° (10.8 ± 7.2%) to 60° (12.1 ± 10.7%) ER representing increasing differences between the strain distributions with ER. Further, elements containing the higher strain (higher quantile average) also exhibited greater changes in strain (quantile difference) between the intact and injured states. (Figure 2) Significant correlations were found between the peak nonrecoverable strain in each shoulder and the range values at 30° (r = 0.83, p = 0.04) and 60° (r = 0.89, p = 0.02) ER but not at 0° ER (r = 0.26, p = 0.62).

Significant differences were found in anterior translation, with the 25N anterior load applied, between the intact and injured states at 0° (p = 0.03), 30° (p = 0.03), and 60° (p < 0.01) ER. Increases in anterior translation were 1.5 ± 1.3 mm, 2.1 ± 1.7 mm, and 2.0 ± 1.1 mm at 0°, 30°, and 60° ER, respectively. The percent increase in anterior translation increased from 17.7 ± 29.2% at 0° ER to 25.5 ± 39.5% at 30° ER and again to 48.4 ± 26.9% at 60° ER.

DISCUSSION
This study simulated injury to the glenohumeral capsule resulting from glenohumeral dislocation due to an excessive force in the anterior direction. Permanent tissue deformation was quantified as nonrecoverable strain in the capsule and was correlated to changes in capsule function following dislocation. Strain distributions in the anteroinferior capsule under a simulated clinical exam were altered following dislocation with areas of greatest strain experiencing the most change following injury at all three joint positions. These changes were found to be significantly correlated to the amount of peak nonrecoverable strain in positions of external rotation. This indicates that the axillary pouch, which experiences areas of greater localized damage from dislocation compared to the AB-IGHL, exhibits a loss in stabilizing function particularly in positions of external rotation. Therefore, surgeons may need to focus more on the axillary pouch during repair procedures following anterior dislocation.

Significant increases in anterior translation during simulated clinical exams were also found following dislocation. The percent increase in anterior translation almost tripled from 0° to 60° ER further indicating compromised capsule function. The changes in anterior translation, although similar to those reported for other experimental models creating permanent capsular deformation [11], are on the order of a few millimeters. While larger dislocations may result in greater changes in anterior translation, surgeons may not be capable of accurately detecting the small changes in anterior translation between normal and injured shoulders found here. This may contribute to the poor reliability of current clinical examinations. [3] As larger changes in joint kinematics occur at joint positions where the damaged region of the capsule is most important for maintaining joint stability, developing standardized clinical exams to diagnose damage in specific capsule regions may improve patient outcome due to misdiagnosis. This work investigated one mechanism of glenohumeral dislocation and other mechanisms may result in more tissue damage. Further, only the strain distributions in the anteroinferior capsule midsubstance were examined. Due to the continuous nature of the capsule, damage may have occurred in other regions or at the insertion sites. However, insight into the relationship between permanent tissue deformation and capsule function following anterior dislocation was achieved. In the future, this experimental injury model can be used to examine the effect of dislocation on the material properties and collagen fiber alignment of the capsule to aid in the development of finite element models of the injured glenohumeral joint.

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REFERENCES