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EFFECTS OF SIMULATED INJURY ON THE ANTEROINFERIOR GLENOHUMERAL CAPSULE

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Abstract

Glenohumeral dislocation results in permanent deformation (nonrecoverable strain) of the glenohumeral capsule which leads to increased range of motion and recurrent instability. Minimal research has examined the effects of injury on the biomechanical properties of the capsule which may contribute to poor patient outcome following repair procedures. The objective of this study was to determine the effect of simulated injury on the stiffness and material properties of the AB-IGHL during tensile deformation. Using a combined experimental and computational methodology, the stiffness and material properties of six AB-IGHL samples during tensile elongation were determined before and after simulated injury. The AB-IGHL was subjected to $12.7 \pm 3.2\%$ maximum principal strain which resulted in $2.5 \pm 0.9\%$ nonrecoverable strain. The linear region stiffness and modulus of stress-stretch curves between the normal (52.4 ± 30.0 N/mm, 39.1 ± 26.6 MPa) and injured (64.7 ± 21.3 N/mm, 73.5 ± 53.8 MPa) AB-IGHL increased significantly ($p=0.03$, $p=0.04$). These increases suggest that changes in the tissue microstructure exist following simulated injury. The injured tissue could contain more aligned collagen fibers and may not be able to support a normal range of joint motion. Collagen fiber kinematics during simulated injury will be examined in the future.

Keywords

Shoulder; Glenohumeral Joint; Finite Element Analysis; Mechanical Properties; Soft Tissue Injury

INTRODUCTION

The glenohumeral joint suffers more dislocations than any other major diarthrodial joint, most of which occur in the anterior direction.^[4,15] The glenohumeral capsule, a complex sheet of ligamentous tissue, functions to stabilize the glenohumeral joint in end ranges of motion.^[8,24] Following dislocation, a variety of capsular lesions are known to occur.^[1,2] However, in cadaver models, lesions alone were not capable of increasing joint translations enough to result in glenohumeral dislocation^[30,35] suggesting that permanent tissue deformation plays a substantial role in the development of instability following dislocation. Permanent tissue deformation has been investigated and modeled in ligamentous tissue such as the cervical ligaments during whiplash^[32,33,36,37,45], following repetitive sub-failure loading of the glenohumeral capsule^[28] and following a single sub-failure stretch of the rabbit anterior cruciate ligament.^[27] This deformation has previously been quantified by measuring the resulting amount of nonrecoverable strain.^[17,21,31] Despite the number of studies examining permanent deformation of the capsule, research on the properties of the

injured glenohumeral capsule is not nearly as prevalent. It is likely that this lack of knowledge regarding injury to the capsule during dislocation contributes to recurrent dislocations in up to 18% of patients following surgical repair for traumatic anterior instability.^[5,26,44]

Of all the capsule regions, the anteroinferior capsule (anterior band of the inferior glenohumeral ligament (AB-IGHL) and axillary pouch) is the primary restraint to anterior dislocation and experiences the highest strains during these events.^[3,7,19,39] As a result, this region is commonly injured during joint dislocation. The collagen fibers in the anteroinferior capsule have been shown to have regions of alignment.^[14,25,29] However, other work has demonstrated that the fibers do not exhibit a global preferred orientation despite localized areas of alignment.^[6] Therefore, an isotropic hyperelastic constitutive model has been used to characterize the material properties of the normal glenohumeral capsule.^[34] As the collagen fibers of other biologic soft tissues have been shown to become more aligned under loading ^[13,16,32] an isotropic constitutive model may not appropriately describe the potentially anisotropic behavior of the injured glenohumeral capsule.

The previously used isotropic hyperelastic constitutive model utilizes a phenomenological strain energy function which does not distinguish between matrix and collagen fiber responses, thus the material coefficients of the model do not have direct physical meaning. Therefore, changes in the material properties of the glenohumeral capsule following injury may not correspond to detectable differences in the material coefficients of this phenomenological isotropic constitutive model. A method for simulating injury (creating nonrecoverable strain) in the glenohumeral capsule should be developed and used to determine if the previously utilized isotropic constitutive model is capable of detecting changes in the material behavior of the capsule via changes in the material parameters following simulated injury. Therefore, the objective of this study was to determine the effect of the simulated injury on the stiffness and material properties of the AB-IGHL during tensile deformation. A combined experimental-computational protocol has been developed and used to characterize the material properties of the glenohumeral capsule.^[34,41]

METHODS

Approval for this study was obtained from the University of Pittsburgh – Committee for Oversight of Research Involving the Dead (CORID No. 131) for use of cadaveric shoulders. Six cadaveric shoulders (57 ± 8 yrs., 4 males, 2 females) were stored at -20°C and thawed for 24 hours at room temperature prior to testing. Based on the mechanical properties of other regions of the normal glenohumeral capsule, a variation of 40–86% was expected, and an effect size of 1.3 was computed. With significance set at $\alpha = 0.05$, a total of six specimens would be required to detect differences in the stress-stretch curves of the normal and injured AB-IGHL with 80% power. The shoulders were dissected free of all skin and musculature leaving only the scapula, humerus and glenohumeral capsule. Each joint was examined using radiographs and dissection, and determined to be free of pathology, osteoarthritis and any visible signs of injury. The AB-IGHL was identified by applying distraction and external rotation to the joint as the band is most visible in this position.^[24]

Tissue samples (20×15 mm) were then excised from the AB-IGHL. (Figure 1) This aspect ratio was chosen to capture the mechanical response of the glenohumeral capsule in a similar loading environment as it would experience *in vivo* since it functions as a continuous sheet rather than a discrete ligament. However, due to the small aspect ratio uniaxial extension was not achieved but rather a combination of tension and shear. Each sample was mounted in custom soft tissue clamps and the superior margin of the AB-IGHL **was visually approximated** and used as an anatomic reference to apply a tensile deformation in the

direction parallel to the longitudinal axis of the AB-IGHL. A 3×3 grid of black delrin markers (diameter: 1.6 mm) was attached to each tissue sample covering approximately a 10×10 mm area of the tissue midsubstance for strain tracking using a small amount of cyanoacrylate. Strain tracking was performed optically with a custom designed motion analysis system (Spicetek, Inc., Accuracy: 0.01mm).^[10,11,22,23] Tissue samples were hydrated using physiologic saline solution throughout the entire testing protocol.

Each AB-IGHL was then elongated in the direction parallel to the longitudinal axis of the AB-IGHL using a materials testing machine (Thumler, Model #TH2730) with a load cell (Interface, Scottsdale, AZ, Model #SM-1000N, range: 0 – 1000 N, resolution: 0.015 N) to determine the stiffness and material properties of the normal tissue sample. (Figure 2) This non-destructive loading protocol consisted of applying a 0.5 N pre-load and measuring tissue sample geometry using a ruler (width and clamp-to-clamp distance, accuracy: 0.5 mm) and digital calipers (thickness, accuracy: 0.03mm). Width and thickness measurements were made three times along the length of the tissue and averaged together. Each tissue sample then underwent preconditioning consisting of ten cycles of elongation to 1.5 mm at a rate of 10 mm/min. Immediately following preconditioning the tissue sample was elongated to 2.75 mm again at a rate of 10 mm/min. This elongation was chosen to elongate the tissue sample into the linear region of the load-elongation curve. Previously published literature on the mechanical properties of other regions of the glenohumeral capsule (axillary pouch and posterior capsule) using the same methodology as the current work provided an initial estimate for the non-destructive elongation of 2.25 mm.^[34,41] However, preliminary experiments performed on the AB-IGHL showed that 2.25 mm was not enough to consistently elongate the AB-IGHL into the linear region therefore this elongation was increased to 2.75 mm. The tissue was then allowed to recover for 30 minutes at an unstrained state. This was the shortest recovery time between two applications of the same loading condition required to produce repeatable load-elongation curves.^[34] This non-destructive tensile deformation was used to determine the stiffness and material properties of the normal AB-IGHL.

Following the 30 minute recovery period, injury was simulated in the tissue samples in order to create nonrecoverable strain. To achieve this, the same preconditioning and pre-load protocol was applied to each tissue sample extracted from the AB-IGHL. The positions of the strain markers were captured in the pre-loaded state and served as the *reference state* for the strain calculations. The amount of elongation needed to injure each tissue sample was determined quantitatively from its non-destructive load-elongation curve. The percent elongation at which the R^2 value of the linear region deviated from 0.999 was defined as the end of the toe region. The tissue sample was then injured by elongating it to twice the percent elongation at the end of the toe region of the non-destructive load-elongation curve. Preliminary experiments determined that this elongation was consistently enough to elongate the tissue into the plastic region of the load-elongation curve in order to produce nonrecoverable strain. The positions of the strain markers were also captured at the maximum elongation for each tissue sample and served as the *injured strain state* for the strain calculations. Following simulated injury the tissue sample was returned to an unstrained state and allowed to recover for 30 minutes.

Next, the tissue sample was pre-loaded again to 0.5 N. The positions of the strain markers were captured at this pre-load and served as the *nonrecoverable strain state*. The experimental repeatability of the strain calculations was determined to be 0.25%, therefore, it was verified that the nonrecoverable strain in the tissue midsubstance was at least an order of magnitude greater before proceeding. Finally, the same pre-load, tissue sample geometry measurements, preconditioning, and non-destructive elongation protocol previously performed on the normal tissue was repeated on the injured AB-IGHL.

The material coefficients of the normal and injured AB-IGHL were determined computationally utilizing a previously developed methodology.^[34,41] Since the experimental tests produced inhomogeneous deformations, specimen-specific finite element models were used to predict the response of the tissue. The tissue sample geometry, applied elongation and clamp reaction forces from the nondestructive tests were used as boundary conditions to create finite element models of each tissue sample (normal and injured). A hyperelastic isotropic strain energy function was used to describe the response of the tissue to the applied elongation and the material coefficients were determined using an inverse finite element optimization technique.^[42] The strain energy was based on the form originally used by Veronda and Westmann^[40], but with an uncoupled dilatational and deviatoric response.^[34,41,43] C_1 and C_2 are the material coefficients that were determined using the inverse finite element optimization routine, where C_1 scales the magnitude of the stress-stretch curve and C_2 governs the magnitude and nonlinearity of the stress-stretch curve. Both material coefficients were constrained to be greater than 0 to ensure physically reasonable behavior and strong ellipticity but less than 5 (C_1) or 50 (C_2).^[20,34,41]

The optimized coefficients for the normal and injured AB-IGHL were then used to generate stress-stretch curves for uniaxial extension, respectively, using the deformation gradient for uniaxial extension and the isotropic hyperelastic constitutive model.^[34,41] Stress-stretch curves are more representative of the mechanical response of the tissue samples rather than the structural response provided by the load-elongation curves. Therefore, these curves are not representative of the tissue sample behavior during the experimental elongations but represent the response of the tissue to uniaxial extension. Because of the poor aspect ratio used in the experimental conditions, uniaxial extension was not achieved experimentally. Stress-stretch curves from $\lambda = 1.0$ to $\lambda = 1.3$ were discretized into twenty-one points and averaged across all tissue samples generating an average curve for the normal and injured AB-IGHL.^[12,34,41] This enabled a representation of how each tissue sample, with individual material properties, would behave in response to uniaxial extension.

Data Analysis

The initial stiffness of the toe region and the linear region stiffness, as well as the optimized material coefficients were compared between the normal and injured tissue states using paired t-tests as all data was normally distributed. Significance was set at $\alpha = 0.05$ for all statistical analyses which were performed using SPSS (IBM, Armonk, NY).

In order to verify that the simulated injury was successful (presence of nonrecoverable strain), the average maximum principal strain for each tissue sample in the *injured* and *nonrecoverable strain states* was determined. The coordinates of the markers at each strain state were input into a finite element software package (ABAQUS, Simulia, Providence, RI). The magnitude of the maximum principal strain at the centroid of each element was calculated and averaged over the four elements for the *injured* and *nonrecoverable strain states* for each tissue sample. This average maximum principal strain was then averaged again over all tissue samples at the *injured* and *nonrecoverable strain states*. The strains in the AB-IGHL midsubstance were an order of magnitude greater than the experimental repeatability of the strain calculations (0.25%).

Pearson's correlation coefficient was used to compare the average and peak maximum principal strain for the AB-IGHL in the *injured* and *nonrecoverable strain states* as well as to compare the average strain values to the linear region stiffness of the load-elongation curves and the change in stiffness between the normal and injured states. The critical value for Pearson's correlation coefficients for a sample size of $n = 6$ is $r = 0.8$.^[38] Therefore, correlations were considered statistically significant when $0.8 < |r| < 1.0$. Further, the correlation was considered moderate for $0.5 < |r| < 0.8$, weak for $0.0 < |r| < 0.5$.^[9] The initial

stiffness of the toe region and linear region was determined from the load-elongation curves of the normal and injured states for each tissue sample. The initial stiffness of the toe region was computed as the slope of the load-elongation curve between 0 and 0.5 mm of elongation. Since this region represents a small portion of the toe region and minimal load is taken up by the tissue during this elongation, a linear assumption is justified. The stiffness of the linear region was computed as the maximum slope of the entire load-elongation curve over a running window of 1% elongation, respectively.

A single statistical comparison of the optimized material coefficients may not be enough to reveal differences in the material properties of the normal and injured AB-IGHL as the constitutive model is nonlinear and the material coefficients are not linearly independent. Therefore, averaging the individual material coefficients does not yield the same stress-stretch response as averaging the individual stress-stretch curves and determining a set of average material coefficients. In order to determine if differences existed between the normal and injured stress-stretch curves two additional analyses were performed for 1) for statistical differences and 2) clinically significant differences. To determine statistical differences, a repeated measures ANOVA and paired t-test post-hoc analyses were used to compare the twenty-one discretized points of the stress-stretch curves. The average stress-stretch curves were then fit to the stress-stretch relationship for uniaxial extension using the non-linear Levenberg-Marquardt algorithm to obtain a set of material coefficients representing the average stress-stretch curves for each region. These material coefficients representing the average stress-stretch curves were compared as previously reported.^[34,41] To determine clinically significant differences, a sensitivity analysis was performed on the material coefficients of the average stress-stretch curves. Based on the analysis, differences greater than 0.30 for C_1 or greater than 3.0 for C_2 were found to be clinically significant. Differences in the material properties greater than these values resulted in a change in the material properties of the tissue. This change was quantitatively measured by a squared correlation coefficient of <0.9 when two stress-stretch curves were compared.

Further, the modulus of each tissue sample in the normal and injured states was determined as the slope of the Cauchy stress-stretch curves between a stretch of $\lambda = 1.2$ and $\lambda = 1.3$. As the moduli were normally distributed, a paired t-test was used to compare between the normal and injured AB-IGHL.

RESULTS

Experimentally, an elongation of 3.78 ± 0.70 mm was applied to simulate injury and cause nonrecoverable strain in each AB-IGHL. At the *injured strain state* (AB-IGHL elongated into plastic region of load-elongation curve), the average and peak maximum principal strain in the AB-IGHL were $12.7 \pm 3.2\%$ (Range: 8.3% to 16.9%) and $19.2 \pm 5.6\%$ (Range: 12.8% to 27.1%), respectively. The average and peak maximum principal nonrecoverable strain in the tissue midsubstance was $2.5 \pm 0.9\%$ (Range: 1.5% to 4.0%) and $5.4 \pm 1.8\%$ (Range: 3.6% to 7.7%), respectively. Further, the clamp-to-clamp distance with the preload applied (*reference state*) increased significantly ($p = 0.01$) following simulated injury by 1.7 ± 1.0 mm ($11.0 \pm 7.6\%$). The correlation coefficients between the average and peak maximum principle nonrecoverable strain and maximum principal strain at the *injured strain state* were 0.5 and 0.7, respectively (Figure 3).

The experimental load-elongation curves for each tissue sample in the normal and injured states exhibited the non-linear behavior typical of biologic tissues (Figure 4). No statistical difference was found when comparing the initial stiffness of the toe region of the load-elongation curves for the normal (5.7 ± 1.9 N/mm) and injured (6.1 ± 3.3 N/mm) AB-IGHL ($p = 0.70$). The linear region stiffness was significantly different between the normal and

injured AB-IGHL ($p = 0.03$). (Figure 5) The increase in linear region stiffness following simulated injury was computed for all AB-IGHL samples; the average increase was 47%. Linear region stiffness in both the normal and injured states was significantly correlated to the average (normal & injured: $r = 0.8$, $p = 0.04$, Figure 6A) maximum principal nonrecoverable strain in the tissue midsubstance. However, the change in linear region stiffness between the normal and injured states was negatively correlated to the average ($r = -0.7$, Figure 6B) maximum principal nonrecoverable strain in the tissue midsubstance.

No statistical difference was found when comparing the optimized material coefficients for the normal and injured AB-IGHL ($p = 0.59$ for C_1 ; $p = 0.07$ for C_2). The values of C_1 and C_2 were 0.71 ± 0.51 MPa (Range: 0.21 MPa to 1.36 MPa) and 3.2 ± 1.8 (Range: 1.4 to 6.3), respectively for the normal tissue samples. Following injury, the values of C_1 and C_2 were 0.56 ± 0.59 MPa (Range: 0.12 MPa to 1.45 MPa) and 5.0 ± 1.7 (Range: 3.3 to 7.6), respectively. When compared to the normal AB-IGHL, the value of C_1 was 20% lower (by 0.15 MPa) and the value of C_2 was 56% higher (by 1.8) in the injured AB-IGHL.

Computationally predicted stress-stretch curves for uniaxial extension were generated up to a stretch of $\lambda = 1.3$ using the optimized material coefficients for each AB-IGHL in the normal and injured states. Average curves were then generated representing the response of the normal and injured AB-IGHL to uniaxial extension. The average stress-stretch curves exhibited a non-linear shape, typical of soft tissues and are similar with a correlation coefficient between the curves of $R=0.998$. (Figure 7) No significant differences were found between the normal and injured AB-IGHL when comparing the stress values at each of the twenty-one discretized points of the stress-stretch curve for uniaxial extension ($p = 0.05$).

The values of C_1 and C_2 representing the average stress-stretch curves were 0.36 MPa and 4.6 for the normal tissue, and 0.39 MPa and 5.4 for the injured tissue. The values of C_1 and C_2 were 0.03 MPa and 0.7 higher in the injured than the normal AB-IGHL. Again, differences of greater than 0.3 for C_1 and greater than 3.0 for C_2 are considered clinically significant.^[34] Therefore, the coefficients representing the average stress-stretch curves are not different using this constitutive model. However, statistically significant differences were found between the modulus of the normal and injured AB-IGHL ($p = 0.04$). (Figure 5) The average increase in modulus between the normal and injured tissue states was 34.5 ± 31.7 MPa (128%).

DISCUSSION

This study examined the effects of simulated injury on the stiffness and material properties of the AB-IGHL during tensile deformations using a combined experimental and computational methodology. Simulated injury of the AB-IGHL resulted in nonrecoverable strain and increased tissue elongation thereby confirming that permanent tissue deformation occurred. The maximum principle strain produced in the tissue samples from the AB-IGHL in the *injured strain state* and *nonrecoverable strain state* compare well with other injury models.^[17–19] For example, varying degrees of subluxation of the glenohumeral joint resulted in 9% – 15% maximum principal strain during injury^[19] and 5% – 7% nonrecoverable strain^[17] in the anteroinferior glenohumeral capsule. As expected, strain values found in tissue samples extracted from the AB-IGHL in the current work were slightly lower than those reported in the intact joint due to the continuous structure and function of the glenohumeral capsule. Nonetheless, the simulated injury model developed in this work is capable of creating maximum principal strains in tissue samples from the glenohumeral capsule that are similar to those experienced by the intact capsule during joint subluxation.

In this injury model, tissue samples from the AB-IGHL were elongated into the plastic region of the load-elongation curve in order to consistently create nonrecoverable strain. Although this model consistently injures the AB-IGHL, the amount of damage is variable between tissue samples, as indicated by the standard deviation in the maximum principal strain in the tissue sample in the *injured strain state*. Future studies should base injury on strains applied to the tissue midsubstance rather than elongation in order to produce consistent damage to each tissue sample.

The positive correlation between the strain at the *injured strain state* and the resulting nonrecoverable strain in the AB-IGHL suggests that more severe injuries may result in higher amounts of nonrecoverable strain. Future experiments should evaluate this relationship under more complex loading conditions such as the amount of nonrecoverable strain in the capsule resulting from glenohumeral dislocation. Similar results have been reported when a univariate regression model was used to examine the relationship between the maximum principal strain at the *injured* and *nonrecoverable strain states* produced by applying varying degrees of glenohumeral subluxation ($R^2 = 0.35$, $r = 0.6$).^[18]

The increase in linear region stiffness following simulated injury found in this study compares well with a sub-failure injury model in which rabbit anterior cruciate ligaments were subjected to an 80% sub-failure loading and then loaded to failure.^[27] The correlations between the linear region stiffness and nonrecoverable strain suggests that in tissue samples where the fibers are only able to rotate a small amount between the normal and injured states (small change in linear region stiffness), damage is likely to occur at an earlier elongation compared to tissue samples where the fibers can rotate substantially (large change in linear region stiffness) to support applied loads. Therefore, tissue samples which are more aligned initially are more likely to be damaged during the simulated injury than those with dispersed fibers. Further examination of collagen fiber kinematics in the glenohumeral capsule during tissue elongation is necessary to investigate this hypothesis.

The material coefficients of the normal AB-IGHL compare well with those reported for other capsule regions.^[34] Despite differences in the material properties of the AB-IGHL following injury, no statistical differences were found in the constitutive model parameters or the computationally predicted stress values at each of the 21 discretized points. These findings suggest that the constitutive model used in this work is not able to detect changes in the material coefficients, for the small amount of nonrecoverable strain created. This could be attributed to the phenomenological form of the strain energy function, which does not distinguish between matrix and collagen fiber responses. Changes in the material coefficients affect the entire stress-stretch response, as opposed to the toe or linear regions independently, making changes in tissue microstructure, which primarily affect the linear region, difficult to detect. Therefore, a constitutive model which includes terms describing the contributions of the fibers and ground substance separately will likely describe the behavior of the tissue more appropriately following injury and will be utilized in the future.

In addition to a significant increase in clamp-to-clamp distance following simulated injury, an increase in the toe-region of the load-elongation curve was observed. However, it was not quantified as the same preload had to be applied to the normal and injured AB-IGHL samples in order to quantify the nonrecoverable strain produced during simulated injury. This concept should be examined in the future in order to fully characterize the properties of the injured AB-IGHL. In vivo, the capsule acts as a continuous sheet of tissue rather than a uniaxial ligament^[22] and thus experiences more complex loadings and strains than reported here. However, a low aspect ratio was used to simulate these complex loading conditions by creating inhomogeneous deformations during loading. Tissue properties were then determined from the theoretical case of uniaxial extension.

The simulated injury model examined here is capable of creating nonrecoverable strain in capsular tissue that is similar to damage experienced by the intact capsule during injury. Following permanent deformation, changes in the stiffness and material properties of the AB-IGHL were found. These changes could be due to permanent changes in the collagen fiber architecture following simulated injury. Since the tissue is stiffer, its reference state may contain more aligned collagen fibers following injury that shift the tissue towards anisotropy. Therefore, surgical repair procedures aimed at tightening the capsule should account for the changes in tissue properties following injury in order to fully restore normal capsule function. In the future, collagen fiber kinematics during simulated injury as well as a structural constitutive model will be examined to characterize microstructural changes in the capsule due to injury.

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Figure 1.
Overview of methodology used to determine the stiffness and material properties of the normal and injured AB-IGHL.

Figure 2. Mechanical testing setup for experimentally applied tensile elongations showing load cell and clamp relationships and 3×3 grid of strain markers on the surface of the tissue sample.

Figure 3. Correlations between the average (A) and peak (B) maximum principal strain collected experimentally in the *nonrecoverable* and *injured strain states*.

Figure 4. Experimental load-elongation curves for a representative normal and injured AB-IGHL during the nondestructive elongations.

Figure 5. Linear region stiffness calculated from the experimental load-elongation curves and modulus of the stress-stretch curves determined from the computationally predicted stress-stretch curves for uniaxial extension for the normal and injured AB-IGHL.

Figure 6. Correlations between experimental average maximum principal nonrecoverable strain and the A) linear region stiffness of the normal and injured AB-IGHL and B) change in linear region stiffness between the normal and injured AB-IGHL.

Figure 7.
Computationally predicted stress-stretch curves (mean $\pm$ SD) for the normal and injured AB-IGHL under uniaxial extension.