A novel cadaveric model for anterior-inferior shoulder dislocation using forcible apprehension positioning

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Abstract—A novel cadaveric model for anterior-inferior shoulder dislocation using forcible apprehension positioning is presented. This model simulates an in vivo mechanism and yields capsulolabral lesions. The scapulae of 14 cadaveric entire upper limbs (82 ± 9 years, mean ± standard deviation) were each rigidly fixed to a custom shoulder-testing device. A pneumatic system was used with pulleys and cables to simulate the rotator cuff and the deltoid muscles (anterior and middle portions). The glenohumeral joint was then positioned in the apprehension position of abduction, external rotation, and horizontal abduction. A 6-degree-of-freedom load cell (Assurance Technologies, Garner, North Carolina) measured the joint reaction force that was then resolved into three orthogonal components of compression force, anteriorly directed force, and superiorly directed force. With the use of a thrust bearing, the humerus was moved along a rail with a servomotor-controlled system at 50 mm/s that resulted in horizontal abduction. Force that developed passively in the pectoralis major muscle was recorded with an independent uniaxial load cell. Each of the glenohumeral joints dislocated anterior-inferior, six with avulsion of the capsulolabrum from the anterior-inferior glenoid bone and eight with capsulolabral stretching. Pectoralis major muscle force as well as the joint reaction force increased with horizontal abduction until dislocation. At dislocation, the magnitude of the pectoralis major muscle force, 609.6 N ± 65.2 N was similar to the compression force, 569.6 N ± 37.8 N. A cadaveric model yielded an anterior dislocation with a mechanism of forcible apprehension positioning when the appropriate shoulder muscles were simulated and a passive pectoralis major muscle was included. Capsulolabral lesions resulted, similar to those observed in vivo.

Key words: cadaver, dislocation, model, shoulder.

INTRODUCTION

The goal in treating glenohumeral instability is not only simply to eliminate pain but is also to restore normal function. Knowledge of shoulder anatomy, biomechanics, and operative techniques has yielded low rates of instability after treatment [1–6]. However, the return-to-normal function, gauged by the ability to return to the same level of sporting activities, remains inadequate [1,7].

Abbreviations: ANOVA = analysis of variance, DOF = degree of freedom, IGHL = inferior glenohumeral ligament, SD = standard deviation, SLAP = superior labrum from anterior to posterior.

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The Bankart lesion is an injury of the anterior-inferior capsulolabral structures that includes the glenoid insertion site of the anterior band of the inferior glenohumeral ligament (IGHL) [8], which is important in preventing anterior joint dislocation [9]. One of the challenges for accurately evaluating surgical reconstruction following anterior-inferior shoulder dislocation is that no cadaveric model exists that uses an in vivo injury mechanism to produce a pathoanatomy that is seen in vivo. In a prior study, joint pathoanatomy that occurs after anterior glenohumeral dislocation, such as the Bankart lesion, was simulated with the lesion being surgically created [10]. A linear force was applied to the humeral head, and translation on the glenoid was measured. Such studies found the inferior glenohumeral ligament to be the primary static restraint to anterior glenohumeral instability [9,11,12], and after the ligament was repaired, normal translation of the joint was restored [13]. A critical size of the glenoid rim defect that may occur after dislocation in vivo was also found [14]. The humeral head translated with significantly less force when the width of the osseous defect was at least 21 percent of the glenoid length (average width = 6.8 mm) [15]. Elimination of normal negative pressure by the venting of the joint to the atmosphere or simulation of a superior labrum from anterior to posterior (SLAP) lesion by incision of the superior labrum from anterior to posterior was also associated with anterior instability in cadaveric study [16–18].

Another method of cadaveric study was for the researchers to test the joint in tension to determine the biomechanical properties of the soft tissues. Large failure loads resulted and the anterior-inferior capsulolabrum was the initial structure to fail [19,20]. A small amount of anterior-inferior capsulolabrum stretching was found with an initial instability episode [21–23]. Stretching occurred along the length of the involved soft tissues regardless of the failure mode [24,25].

It has long been known that symptoms after unidirectional anterior glenohumeral joint dislocation are specific to the shoulder position. Only rarely does dislocation occur in vivo from a mechanism like that of prior cadaveric studies. Joint distraction or the application of an external force, as from the hand of another individual being applied to the posterior shoulder, is rare. Instead, patients commonly assert that dislocation occurred while the arm was behind the body with the shoulder abducted, externally rotated, and in horizontal abduction. When the arm is subsequently placed in this position, there is pain and uneasiness that dislocation will recur. This “behind the body” motion is also part of the apprehension test that aids in diagnosis of anterior glenohumeral instability [26]. If joint instability does occur, the patient may be apprehensive that the joint will dislocate.

Prior cadaveric models of glenohumeral joint dislocation often lacked simulation of appropriate shoulder muscle forces despite these muscles being responsible not only for movement but also for joint stability. The orientation of the shoulder muscles to the glenohumeral joint is such that the joint reaction force has a large component that acts perpendicular to the glenoid fossa to compress the concave humeral head into the glenoid fossa [25,27]. This phenomenon was coined “concavity-compression” by Lippett and coworkers and has been shown to maintain anterior joint stability over a large range of shoulder motion [27–29]. Likewise, simulated inactivity of the supraspinatus and subscapularis muscles yielded an 18 and 17 percent decrease in the force to dislocate the joint, respectively [30]. Also prior models that simulated shoulder muscles failed to include large muscles that originate on the thorax [30–33].

We hypothesized that a cadaver model will yield anterior-inferior dislocation with a mechanism of forcible apprehension positioning of abduction, external rotation, and horizontal abduction when the appropriate shoulder muscles were simulated and a passive pectoralis major muscle was included. If capsulolabral lesions similar to those observed in vivo resulted, the model would also be valuable in evaluating the optimal surgical repair technique. Therefore, the objective of this study was to quantitatively assess and determine if a novel cadaveric shoulder dislocation model that simulates appropriate shoulder muscles will result in shoulder dislocation when the shoulder is forcibly moved from the apprehension position into further horizontal abduction.

**MATERIALS AND METHODS**

We used 14 fresh frozen shoulders (age $= 82 \pm 9.2$ years, mean [$M$] ± standard deviation [$SD$]) without any evidence of rotator cuff tear and degenerative joint disease. These specimens were carefully selected via radiographic and visual examinations. The shoulders were then carefully dissected, leaving the glenohumeral joint and the humerothoracic muscles intact. The muscles of the anterior and middle portions of the deltoid and the
pectoralis major were resected and their tendons preserved. The rotator cuff muscles were also resected from the scapula to expose their tendons. Thereafter, we vented each glenohumeral joint using a 22-gauge needle for consistency. Venting affects glenohumeral joint stability when the shoulder muscles are not active, which occurs when the arm is hanging at the side [16]. However, the effect of venting on stability is small compared to that of concavity-compression when the muscles are active as was simulated in this study [28].

A custom shoulder testing system was developed to be used for shoulder dislocation (Figure 1). The glenoid was positioned parallel to the medial border of a custom aluminum scapula box and fixed with plaster of Paris. It was then mounted onto a custom joint-loading frame that enabled individual control of simulated muscles. The glenohumeral joint reaction force was measured with a 6-degree-of-freedom (DOF) load cell (Assurance Technologies, Garner, North Carolina) and resolved into three orthogonal components of compression force, anterior-directed force, and superior-directed force. The tendons of the rotator cuff (subscapularis, supraspinatus, and infraspinatus and teres minor muscles) and the deltoid (anterior and middle portions) were then clamped, and a pulley and cable system was used to simulate the action of these muscles as prior studies have shown each to be important in glenohumeral abduction [31,34–36] (Figure 2). The infraspinatus and teres minor muscles were combined because they have similar direction and function. The direction of each rotator cuff muscle force vector was defined as the line from the tendon insertion to the centroid of the muscle [37]. The anterior portion of the deltoid was defined as that anterior to the anterolateral corner of the acromion, and the middle portion was defined as that between the anterolateral and the posterolateral corners of the acromion. The direction of the anterior portion of the deltoid force was defined as the line from the anterior lateral corner of the acromion, to the anterior third of the deltoid tendon insertion on the humerus. The direction of the middle portion of the deltoid force was defined as the line from the point midway between the anterolateral and posterolateral corners of the acromion to the middle third of the deltoid tendon insertion. We combined anterior and the middle deltoid to produce one resultant force vector to simulate both of these muscle forces.

**Figure 1.**
Shoulder testing system. Individual pneumatic cylinders were used to simulate individual muscle forces. A 6-DOF load cell was used to measure glenohumeral joint forces. Servomotor was used to horizontally abduct shoulder. Pectoralis major rod and uniaxial load cell are not shown.
groups. We accomplished the simulation of the muscle forces by using LabView/personal computer (PC)-controlled pneumatic cylinders and a custom pulley system incorporated into the shoulder testing system. The pneumatic system permits the application of a specific force magnitude of each muscle and the custom pulley system permits the control of muscle force direction.

The ratio of load applied to the supraspinatus and the deltoid may vary but still result in full glenohumeral abduction [36]. We applied an equal load (40 N) to each of the subscapularis and the infraspinatus and teres minor tendons to balance the anterior-posterior force couple. The same load was then applied to the supraspinatus tendon. We applied a load double (80 N) to that of each rotator cuff tendon to the combined anterior and middle portions of the deltoid to simulate the force necessary to achieve shoulder abduction of 90°. This ratio of muscle loads was similar to previous studies and was kept constant throughout testing [31,36].

The custom aluminum scapula box was abducted 30° in the scapular plane on the custom joint-loading frame, the glenohumeral joint was abducted 60°, and the humerus was externally rotated 90° (Figure 3). We then clamped the pectoralis major tendon to a solid rod attached to a uniaxial load cell (Omega Engineering Co., Stamford, Connecticut) to measure the passive force that developed with testing. The accuracy of this load cell is ±0.13 N.

Based on a preliminary study in our laboratory, the pectoralis major muscle force was directed 60° anterior to the plane of the custom aluminum scapula box and 60° inferior to its superior margin [38], simulating the maximum excursion length of the pectoralis major with the glenohumeral joint in the apprehension position of anterior-inferior instability. The pneumatic system was not involved in the simulation of the pectoralis major muscle in a passive state. These positions were fixed rigidly throughout.

Figure 2.
(a) Muscle force simulation from anterior view. Infraspinatus and teres minor are not shown, since it is on the posterior aspect of shoulder. (b) Muscle force simulation from superior view.

Figure 3.
Shoulder apprehension position (90° shoulder abduction, 90° external rotation.) Custom aluminum scapula box was abducted 30° in scapular plane on custom joint-loading frame, glenohumeral joint was abducted 60°, and humerus was externally rotated 90°.
testing. The humerus was secured in a thrust bearing that prevented distraction forces and allowed only horizontal abduction of the shoulder (Figure 1). Then the humerus was positioned 10° anterior to the plane of the scapula, in horizontal adduction. Testing began with the start of a servomotor-controlled system that moved the humerus in horizontal abduction at a rate of 50 mm/s (Figure 4). Preliminary study in our laboratory with such “forcible apprehension positioning” did not yield joint dislocation unless the pectoralis major tendon was clamped to a solid rod attached to a uniaxial load cell to simulate passive force in this muscle. Study in our laboratory of the direction of the pectoralis major muscle with the shoulder in the apprehension position indicated the pectoralis major muscle force was directed 60° anterior to the plane of the custom aluminum scapula box and 60° inferior to its superior margin.

Testing stopped when the joint dislocated or it moved beyond 45° of horizontal abduction. The joint reaction force was recorded throughout testing and resolved into three orthogonal components: (1) force perpendicular to the glenoid (compression force), (2) force directed anterior to the glenoid (anteriorly directed force), and (3) force directed superior to the glenoid (superiorly directed force). The force in the pectoralis major tendon was also recorded. At the end of testing, we made an incision in the posterior capsule and meticulously examined each joint for intracapsular pathology. We used a one-way analysis of variance (ANOVA) with a p value of 0.05 to compare the forces between the two types of failure modes.

**RESULTS**

All 14 glenohumeral joints dislocated in an anterior-inferior direction. All the force components increased until shoulder dislocation occurred (Figure 5). The individual force data for each individual specimen are shown in Tables 1 and 2. In six shoulders, the anterior-inferior capsulolabrum was avulsed from the glenoid bone (Figure 6). A small shell of glenoid bone was also avulsed in each and remained attached to the capsulolabrum. Little variability was found in its extent, from the 2 o’clock to the 6 o’clock position in each, involving only a very small portion of the articular surface, never greater than a few millimeters. Also, stripping of the capsulolabral structures occurred from the anterior-inferior rim and neck of the glenoid bone. In eight joints, intracapsular inspection revealed no avulsion or tearing of the capsulolabrum, rotator cuff tear, or articular surface damage. Instead, the anterior-inferior capsulolabrum was stretched and patulous compared to that before testing. The amount of capsulolabral stretch was not quantified in this study.

The glenohumeral joint forces showed significant differences in the anterior force (p < 0.03) and inferior force (p < 0.03) between the capsulolabrum avulsion (Bankart) group and the capsulolabrum stretching group (non-Bankart). (Tables 1 and 2, Figure 7(a) and (b)) The compression force showed similar trend but was not statistically significant (p > 0.05) (Tables 1 and 2, Figure 7(c)). The force developed in the pectoralis major during anterior-inferior shoulder dislocation also showed a significantly greater magnitude for the capsulolabrum avulsion group when compared to the capsulolabrum stretching group (p < 0.03) (Tables 1 and 2, Figure 7(d)). For both groups combined, at dislocation, the magnitude of the pectoralis major muscle force, 609.6 ± 65.2 N, was similar to the compression force, 569.6 ± 37.8 N.

**DISCUSSION**

This cadaver model yielded anterior dislocation with a mechanism of forcible apprehension positioning when the appropriate shoulder muscles were simulated and a passive pectoralis major muscle was included. Anterior dislocation occurred partly because the joint was placed forcibly in the apprehension position. It has long been known that symptoms of anterior glenohumeral joint dislocation are specific to the shoulder position of abduction,
external rotation, and horizontal abduction, known as the anterior apprehension position. In prior studies with cadaveric models, the joint was placed gently in the apprehension position and not forced to exceed the normal range of joint motion, so dislocation did not occur. Although active shoulder muscles were simulated [25,39] and no force was applied to the humeral head, joint dislocation did not occur even when the Bankart lesion was created by incision of the anterior-inferior capsulolabral structures. Also, the joint reaction force was not abnormal [25]. Only after division of the entire joint capsule (including both the anterior the posterior aspects) was dislocation found and then in only two of the nine joints studied [39].

The mechanism of forcible apprehension positioning as described in this study may account for the range of shoulder motion being increased in those with chronic instability [40]. The anterior-inferior capsulolabrum is

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**Figure 5.**
Typical glenohumeral joint force components and force developed in pectoralis (pec) major versus horizontal abduction during anterior-inferior shoulder dislocation.

**Table 1.**
Capsular stretch failures.

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<td>222.1</td>
<td>156.5</td>
<td>526.8</td>
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<td>24.3</td>
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<td>31.0</td>
<td>29.7</td>
<td>29.0</td>
<td>28.7</td>
<td>2.5</td>
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SEM = standard of error of mean
normally responsible not only for stabilizing but also for limiting extremes of glenohumeral joint motion [41]. Injury to the anterior-inferior capsulolabrum may occur from either a traumatic dislocation of the joint because of impact, or a rotation, such as horizontal abduction, that exceeds the normal range of motion.

The other reason this model resulted in glenohumeral dislocation was because the pectoralis major muscle was included. Muscles that originate on the scapula and insert on the humerus have been commonly included in prior cadaveric models and rightly so [30,31], because rehabilitation of these muscles is an important part of treatment [2]. But additional shoulder muscles can affect glenohumeral stability and have origin on the thorax. A prior study implicated the pectoralis major muscle in inducing anterior glenohumeral dislocation. After finding a Bankart lesion in a patient with concurrent pectoralis major tendon rupture and anterior glenohumeral dislocation, Arciero and Cruser theorized eccentric lengthening of the pectoralis had led to the dislocation [42]. They theorized that active contraction of the pectoralis major was not necessary for the muscle to contribute to anterior glenohumeral instability. Sinha and

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**Table 2.**
Capsulolabral avulsion (Bankart) failures.

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<td>65.9</td>
<td>63.2</td>
<td>77.2</td>
<td>1.9</td>
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SEM = standard of error of mean

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**Figure 6.**
Glenohumeral joint with a capsulolabrum avulsion (Bankart lesion) following anterior-inferior shoulder dislocation.
coworkers also reported the pectoralis muscle as having a role in glenohumeral instability after an irreducible dislocation was successfully treated with paralysis of the muscle with botulinum toxin injection [43]. The pectoralis major as a humerothoracic muscle not only can cause stability as one of the glenohumeral joint stabilizers but also instability as well. The anterior-inferior shoulder dislocation mechanism described in this study demonstrates the magnitude of the force generated in the pectoralis major to be similar to the compression force on the glenoid. This anterior-inferior shoulder dislocation in forcible apprehension positioning could not be achieved without significant contribution of the pectoralis major.

The idea that a muscle may contribute to, rather than aid in, preventing joint stability is somewhat novel. Yet, the pectoralis major muscle may contribute to anterior glenohumeral dislocation not from active contraction but from tension that develops with passive lengthening beyond the normal muscle-tendon length. The pectoralis major tendon is a broad sheet from two distinct muscle heads, and the lowermost fibers of the sternal head have the most proximal insertion on the humerus. When the shoulder is placed in horizontal abduction, similar to the apprehension position, these lowermost fibers of the sternal head can be stretched to an extreme [44]. The pectoralis major muscle has ideal orientation to pull the

Figure 7.
(a) Shoulder histogram showing anteriorly directed force developed in glenohumeral joint during anterior-inferior shoulder dislocation by forcible apprehension positioning. (b) Shoulder histogram showing inferiorly directed force developed in glenohumeral joint during anterior-inferior shoulder dislocation by forcible apprehension positioning. (c) Shoulder histogram showing compression force developed in glenohumeral joint during anterior-inferior shoulder dislocation by forcible apprehension positioning. (d) Shoulder histogram showing pectoralis major force developed during anterior-inferior shoulder dislocation by forcible apprehension positioning.
humeral head anterior-inferior. Since anterior dislocation may also occur from forcible horizontal abduction of the shoulder, the humeral head can be pulled out of the glenoid by passive tension in the pectoralis major. In a prior study, the pectoralis major muscle resulted in a significant increase in the anteriorly directed force at the gleno-humeral joint in the apprehension position [27]. In this study, a large force developed in the pectoralis major muscle that may have levered the humeral head out of the glenoid. The role of the pectoralis major and other shoulder muscles that have origin on the thorax should be considered in future study of anterior glenohumeral dislocation.

Because of their orientation to the gleno-humeral joint, muscles that move the shoulder contribute to anterior joint stability through concavity-compression. Coined by Lippett and coworkers, this term refers to the component of the joint reaction force that acts perpendicular to the glenoid fossa [28], compressing the humeral head. Concavity-compression was previously reported as being important in maintaining anterior joint stability at the mid-range of shoulder motion when the static restraints are lax [28,45]. This study indicated concavity-compression was also quite important at an end-range of motion, the apprehension position. In this position, the muscle forces were great, maintaining the joint in abduction [36], so concavity-compression was also large. This finding was supported by the joint compressive force being significantly greater than that of anterior force and inferior force. The force component that represents compression to the glenoid surface along with the glenoid concavity provides concavity-compression when the muscles are active as was simulated in this study [28,46]. Prior biomechanical studies of simulated shoulder muscle weakness demonstrated the force to dislocate the joint was diminished [30,32]. Also, there was interplay between the active posterior rotator cuff muscles and the anterior-inferior static restraints [47]. Since the shoulder muscles are crucial to joint stability, treatment should include their rehabilitation.

While lesions in vivo are varied and include capsulolabral avulsion, tears, and stretching and articular surface fracture and rotator cuff tears [48], in this study two types of capsulolabral lesions were observed. The first was capsulolabral avulsion that is similar to the Bankart lesion described in vivo and consistent with anatomic study of the insertion site of the anterior band of the inferior glenohumeral ligament that showed a direct attachment to the labrum and an indirect attachment to the bone of the glenoid neck [24]. The second was dislocation that resulted in no avulsion or tearing of the capsulolabral structures but instead resembled stretching that has been reported in prior biomechanical studies and after instability in vivo [1–3,7,20,21,24,49–51].

Seventeen functional muscle groups contribute to shoulder function [52]. The cadaveric model presented in this study included only the major muscles that contribute to glenohumeral stability and did not include all the muscles that contribute to shoulder function. All cadaveric models of glenohumeral instability to date do not completely recreate the synergy of joint stabilizing systems that are present in vivo. Joint proprioception contributes to stability, mediated by joint mechanoreceptors by initiating muscle activity [53]. This feedback coordinates the shoulder muscles [54,55]. Cadaveric models also simplify complex muscle behavior as a single line of action from the centroid of the muscle to the tendon insertion and fail to include all the shoulder muscles that may contribute to stability [31,36]. The results of this study may have been influenced by the elderly age of the glenohumeral joints used. Lee et al. in 1999 showed that the biomechanical properties of the anterior band of the IGHL are strongly influenced by their age [49]. In the younger group (age = 38.5 ± 0.5 years), disruption of the complex most often occurred at an insertion site. This site was either the glenoid-labrum region of the glenoid insertion site, which represented the Bankart lesion, or the humeral insertion site. In the older group (age = 74.8 ± 5.3 years), disruption most often occurred at the subjacent region but tended to be closer to the glenoid insertion site than the humeral insertion site. In addition, the biomechanical properties were significantly inferior in the older group when compared to the younger group. The ultimate load and the stress at failure in the anterior band of the inferior glenohumeral ligament of the glenoid-soft-tissue-humerus complex showed that the older group was only 61 percent and 46 percent of the younger group, respectively [49]. Therefore, the force to dislocate the joint may have been greater if younger specimens had been used. Likewise, Bankart lesions occur more often in younger than older individuals [6]. Therefore, we believe that the mode of failure would also have been influenced and a higher incidence of capsulolabral avulsions would be observed in younger cadaveric joints.
CONCLUSION

A cadaveric model that included forcible apprehension positioning and the pectoralis major muscle resulted in anterior-inferior glenohumeral dislocation. This resulted in capsulolabral avulsion that resembled the Bankart lesion and capsulolabral stretching similar to lesions observed in vivo. While current treatments aim to restore the interaction between the dynamic and static restraints, the role of shoulder muscles with origin on the thorax, such as the pectoralis major, should be evaluated further. Future cadaveric models may allow quantitative evaluation of surgical repair techniques that lead to improvements in treatment after anterior glenohumeral dislocation.

REFERENCES

MCMAHON et al. Model for anterior-inferior shoulder dislocation


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