

Posterior Capsular Plication Constrains the Glenohumeral Joint by Drawing the Humeral Head Closer to the Glenoid and Resisting Abduction

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Background: Shoulder pain is a common problem, with 30% to 50% of the American population affected annually. While the majority of these shoulder problems improve, there is a high rate of recurrence, as 54% of patients experience persistent symptoms 3 years after onset.

Purpose: Posterior shoulder tightness has been shown to alter glenohumeral (GH) kinematics. Clinically, posterior shoulder contractures result in a significant loss of internal rotation and abduction (ABD). In this study, the effect of a posterior capsular contracture on GH kinematics was investigated using an intact cadaveric shoulder without violating the joint capsule or the rotator cuff.

Study Design: Controlled laboratory study.

Methods: Glenohumeral motion, humeral load, and subacromial contact pressure were measured in 6 fresh-frozen left shoulders during passive ABD from 60° to 100° using an automated robotic upper extremity testing system. Baseline values were compared with the experimental condition in which the full thickness of posterior tissues was plicated without decompressing the joint capsule.

Results: Posterior soft tissue plication resulted in increased compression between the humeral head and the glenoid (axial load) at 90° of ABD. Throughout ABD, the posterior contracture increased the anterior and superior moment on the humeral head, but it did not change the GH kinematics in this intact model. As a result, there was no increase in the subacromial contact pressure during ABD with posterior plication.

Conclusion: In an intact cadaveric shoulder, posterior contracture does not alter GH motion or subacromial contact pressure during passive ABD. By tightening the soft tissue envelope posteriorly, there is an increase in compressive load on the articular cartilage and anterior/superior force on the humeral head. These findings suggest that subacromial impingement in the setting of a posterior soft tissue contracture may result from alterations in scapulothoracic motion, not changes in GH kinematics.

Clinical Relevance: This investigation demonstrates that posterior capsular plication increases the axial load on the shoulder joint during ABD. While a significant difference from baseline was observed in the plicated condition, posterior capsular plication did not change GH motion or subacromial contact pressure significantly.

Keywords: shoulder biomechanics; motion analysis; subacromial space; plication; glenohumeral motion

Shoulder pain is a common problem, affecting 30% to 50% of the American population annually.¹⁹ While the majority of these shoulder problems improve, there is a high rate of recurrence, as 54% of patients experience persistent symptoms 3 years after onset.¹⁹ In 2008, rotator cuff pain and

injuries resulted in approximately 2 million consultations, and more than 53,000 surgical repairs were performed in the United States alone.^{40,42}

Subacromial impingement is the most common disorder of the shoulder, accounting for 44% to 65% of all shoulder complaints.⁴⁵ As a condition, it encompasses various pathologies, including partial-thickness rotator cuff tears, rotator cuff tendinosis, calcific tendinitis, and subacromial bursitis, with associated pain resulting in the loss of

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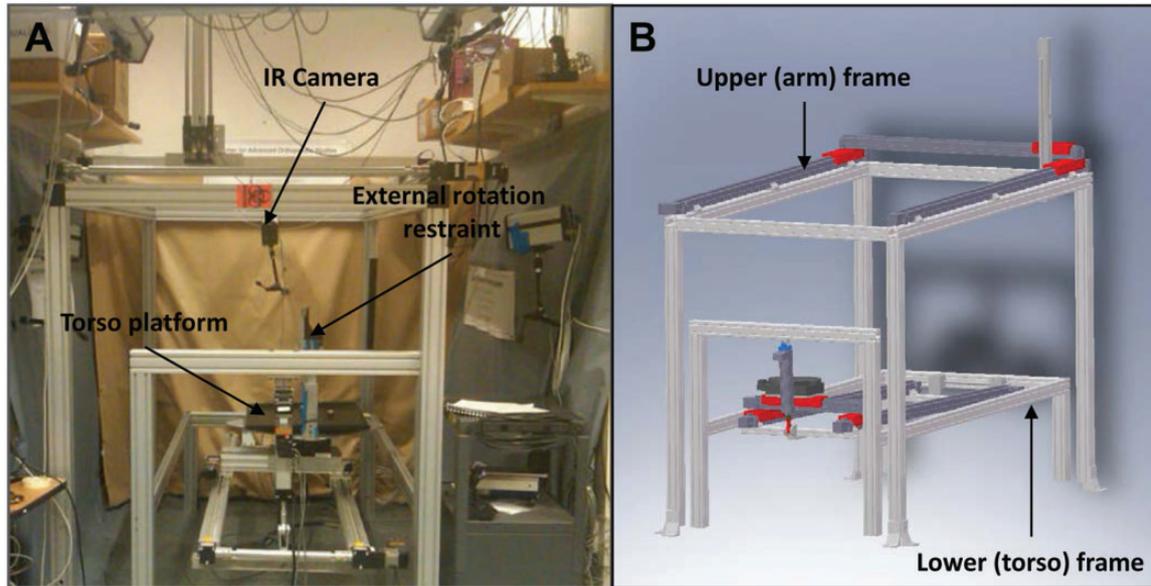


Figure 1. Illustrations of the actual testing system with the (A) infrared (IR) cameras and (B) lower and upper frames.

function and disability.^{18,22} In recalcitrant cases, a posterior capsule contracture has been found to result in altered shoulder kinematics, leading to dysfunction and disability.²⁹

Posterior contracture results in a significant loss of internal rotation and abduction (ABD).^{10,29,34,38,39} Harryman et al¹² demonstrated an increase in anterior and superior glenohumeral (GH) translation during passive forward flexion after tightening of the posterior capsule. Clinically, this problem leads to increased subacromial contact pressure by altering both GH motion and scapulothoracic (ST) motion. In a cadaveric study, Grossman et al¹¹ found a posterolateral shift in the humerus after simulating a posterior capsular contracture. Similarly, Clabbers et al⁵ and Anderson et al (Anderson K, Deng X-H, Johnson T, Altchek DW. "Biomechanical Analysis of Posterior Capsular Contracture of the Shoulder." Presented at American Orthopaedic Society for Sports Medicine, 2001) reported that posterior contracture contributes to changes in the overhead athlete's motion. Additionally, Anderson et al, in a cadaveric study, demonstrated that changes in the overhead athlete can be attributed to the posterior capsule. In patients with persistent disability, surgical release of the posterior capsule has been shown to relieve pain.⁴⁴

In prior studies, GH and ST motions were not quantified. Evidence suggests that impingement results from dysfunction in both the GH and ST joints.^{14,24,43} Biomechanical investigations have shown that the position of the scapula is as important as soft tissue mobility in subacromial impingement.³⁰

It is important to understand the interplay between scapulothoracic motion and rotator cuff pathology because it has important clinical implications as a result of the high incidence of shoulder-related injury and morbidity. Similarly, its treatment and prevention have significant economic consequences. No previous study has clearly demonstrated the cause-effect relationship between posterior capsular contracture and subacromial impingement because of improper methodology or experimental setting. Unfortunately, investigations well-positioned to research this problem have inconclusive results because by violating the joint capsule when manipulating the rotator cuff, the negative pressure within the GH joint is released, which disrupts the GH ligaments and thus alters GH and scapulothoracic kinematics.^{4,15,16,25,28} Not only does this methodology alter the intricate interaction of the GH ligaments during motion, it decompresses the stabilizing benefit of

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concavity compression and it subjugates the GH motion with the effects of gravity. Additionally, manual manipulation of the shoulder during testing can introduce variability, and discontinuous motion analysis can lead to significant artifact and error.

In this investigation, the effect of posterior capsular contracture during passive ABD of an intact cadaveric shoulder was investigated through a simulated plication. GH kinematics, humeral load, and subacromial contact pressure were evaluated in normal and diseased states using a validated robotic apparatus with 7 degrees of freedom (DoF)^{8,41} to mediate reproducible motion trajectories of the upper extremity. Using this system, we hypothesized that a posterior capsular contracture would increase humeral load, increase subacromial contact pressure, and shift the center of GH motion anteriorly and superiorly.

METHODS

Testing Apparatus

A previously validated upper extremity testing system was used to evaluate each specimen.^{8,41} This automated apparatus has precise control and renders a highly reproducible pattern of motion with 7 DoF while recording GH motion, humeral load, and subacromial contact pressure continuously.⁸ The system consists of a lower frame that houses the cadaveric specimen and an upper frame to which the limb is attached (Figure 1). In this investigation, the lower frame rigidly fixed the scapula while the upper frame was programmed to create passive ABD. All axes employ actuator motion and can be programmed using a central controller to generate any motion trajectory within the system's limits.^{8,41}

Cadaveric Torsos

Six fresh-frozen left shoulders were obtained (Medcure Anatomical Tissue Bank) and inspected for rotator cuff integrity and associated morbidities. The specimens originated from white males, 61 ± 5 years of age, with a mean height of 180 ± 10 cm and mean body mass index of 20.14 ± 5.35 kg/m². All specimens were thawed at room temperature 2 days prior to testing. On the day of testing, the skin, subcutaneous fat, and deltoid muscle were removed, as described by Hartzler et al.¹³ Each specimen was mounted rigidly through the body of the scapula. Retroreflective marker clusters were rigidly mounted in the humeral shaft and the spine of the scapula using a validated protocol.^{8,41}

Testing Procedures

For each specimen, ABD was tested in the plane of the scapular body from 60° to 100° (40° range) (Figure 2). Three repetitions of ABD were performed to establish a baseline for GH motion, humeral load, and subacromial contact pressure. The posterior capsule was plicated, and 3 additional repetitions of ABD were performed to model the plicated state. Throughout testing, the specimens

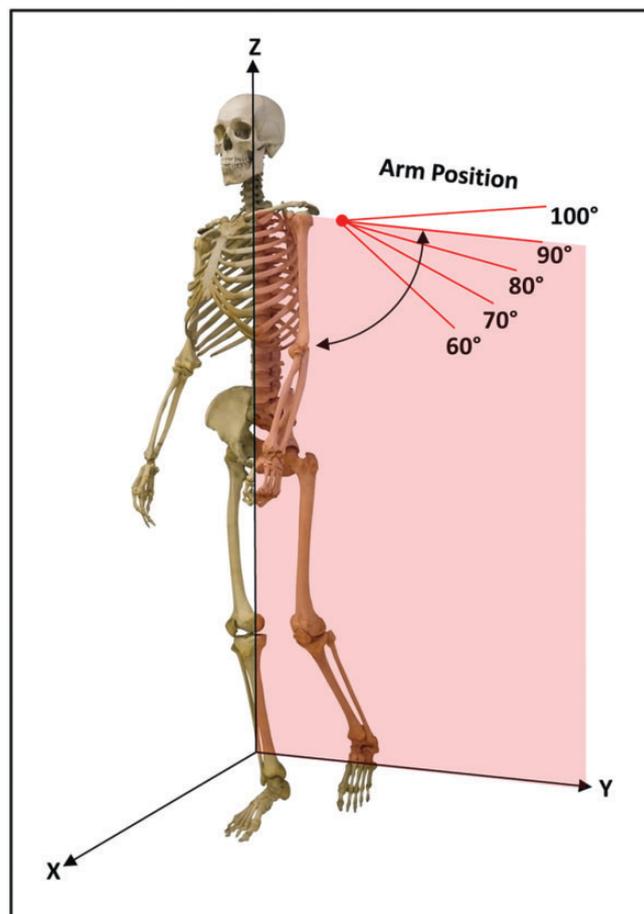


Figure 2. Range of motion of the abduction motion segment.

were kept moist with physiologic 0.9% saline. No resting time was present between repetitions to limit hysteresis.

Posterior Plication

A posterior plication was created using the technique described by Muraki et al.³⁴ In brief, a 3×3 -cm square was created on the posterior shoulder approximately 1 cm medial to the tendon's insertion and 1 cm below the posterior edge of the acromion by internally and externally rotating the humerus. With the arm in neutral rotation, the capsule was imbricated using a No. 5 Ethibond suture (Ethicon US) with a 48-mm conventional cutting sternal needle.³³

Motion Analysis

Five Qualisys Pro Reflex (Qualisys AB) high-speed cameras (120 Hz) were used to record the motion of the retroreflective, bone-embedded marker clusters as previously reported.^{31,32} Prior to testing, the cameras underwent a multiaspect calibration resulting in a spatial resolution of 0.043 m. Anatomical landmarks were used to calibrate the reference frame with respect to the technical (bone-embedded) marker clusters using a pointed wand in accordance with International Society of Biomechanics (ISB)

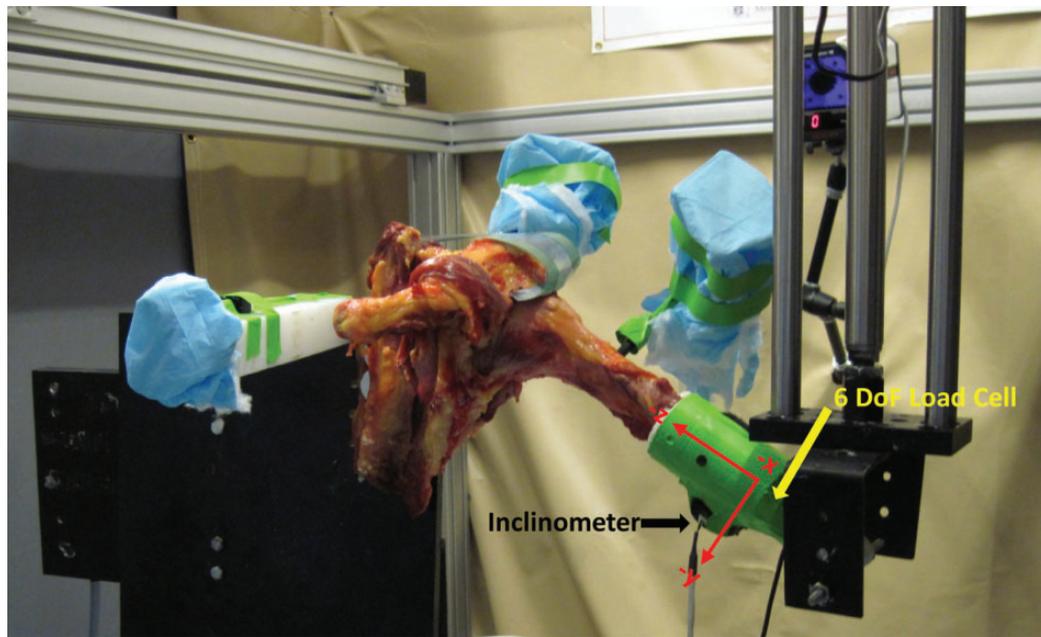


Figure 3. Orientation of the specimens and the load cell coordinates (the axes for the 6-degree of freedom [DoF] load cell with the negative x -axis going into the plane). The load cell axes are shown in the negative direction, as they highlight forces in compression (tension in positive direction).

guidelines.⁴⁹ The calibrated scapular landmarks were calculated as outlined by Meskers et al²⁶ to determine the instant center of rotation of the GH joint (COR_{GH}) within the scapular reference system. The 0 point was defined as the location of COR_{GH} with the arm hanging at the torso's side. The exact angle of shoulder ABD (arm position) was recorded as an independent variable using a digital inclinometer (US Digital).

Humeral Load and Subacromial Contact Pressure Analysis

A nano-25, 6-DoF load cell (ATI Industrial Automation) was mounted between the cut end of the humerus and the actuator arm (upper frame) to measure the humeral load in all 3 axes (Figure 3). Data acquisition was performed using LabView (National Instruments) at a sampling rate of 10 Hz.

A pressure transducer film (model 5051; Tekscan Inc) was used to measure contact pressure in the subacromial space. The film was calibrated and then placed on the undersurface of the coracoacromial arch from the coracoid process anteriorly to the posterior edge of the acromion. The film was inserted from the lateral aspect of the acromion (50-yard line) and was sutured to the coracoacromial ligament and the periosteum on the posterior edge of the acromion.

Statistical Analysis

The Shapiro-Wilk test was used to assess the data for normality. GH translation, humeral load, and contact pressure were recorded continuously throughout the 3 repetitions of

ABD for baseline and plicated. The average GH translation was plotted over time to calculate the total translation and the area under the curve (AUC) during each motion segment. The motion was divided into five 10° segments (60°, 70°, 80°, 90°, and 100°) to facilitate statistical analysis. A linear mixed model analysis of variance was conducted for x -, y -, and z -axis translations, where the sample was the repeated effect and the group and angle were fixed. AUC was calculated for each condition on each axis using the trapezoidal rule to appropriately assess the path-dependent motion (Matlab version 12; MathWorks). The Wilcoxon signed-rank test was used to compare AUCs between conditions. Statistical analysis was conducted using SPSS (version 21.0; IBM). Two-tailed P values of $<.05$ were considered statistically significant.

RESULTS

Humeral Load (Load Cell)

Posterior plication decreased the posterior and increased the anterior force on the humerus when the arm was abducted to 90° compared with baseline ($P = .04$) (Figure 4A and Table 1). At all other positions, the anteroposterior moment showed no statistical difference ($P > .05$ at 60°, 70°, 80°, and 100°) (Figure 4A and Table 1). In comparison with the normal shoulder, the posterior suture plication increased the superior load on the humerus throughout ABD ($P < .001$ at 60°, 70°, 80°, 90°, and 100°) (Figure 4B and Table 1). In the load cell z -axis (medial-lateral), the force on the humerus was significantly lower at 70°, 80°, and 90° of ABD when compared with baseline ($P < .001$) (Figure 4C and Table 1).

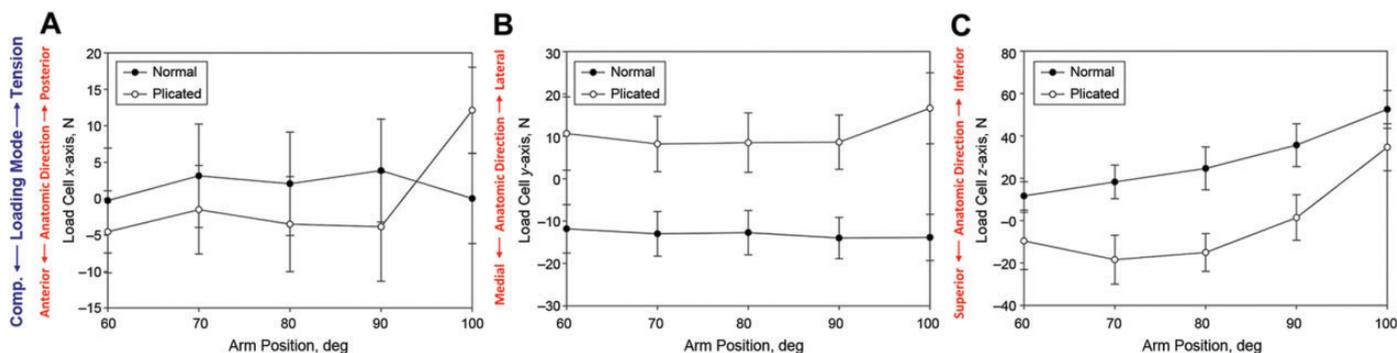


Figure 4. Load cell versus arm position for (A) x, (B) y, and (C) z planes between 60° and 100° of abduction. Comp., compression.

TABLE 1
Load Cell, Tekscan Contact Pressure, and Glenohumeral Center of Rotation Displacement Data for the Control and Plicated Groups^a

	Normal, Mean ± SD	Plicated, Mean ± SD	Delta	P Value
Load cell x, N				
60	-0.27 ± 7.20	-4.56 ± 5.63	-4.29	>.05
70°	3.13 ± 7.09	-1.53 ± 6.08	-4.65	>.05
80°	2.04 ± 7.09	-3.51 ± 6.52	-5.55	>.05
90°	3.84 ± 7.07	-3.87 ± 7.47	-7.72	.04
100°	0.01 ± 6.19	12.12 ± 5.88	12.11	>.05
Load cell y, N				
60°	-11.84 ± 5.72	10.67 ± 8.65	22.50	<.001
70°	-13.00 ± 5.27	8.2 ± 6.52	21.20	<.001
80°	-12.73 ± 5.23	8.50 ± 7.01	21.23	<.001
90°	-14.00 ± 4.89	8.63 ± 6.40	22.63	<.001
100°	-13.86 ± 5.46	16.62 ± 8.38	30.48	<.001
Load cell z, N				
60°	11.66 ± 6.69	-9.57 ± 13.51	-21.23	>.05
70°	18.29 ± 7.94	-18.41 ± 11.56	-36.70	<.001
80°	24.65 ± 10.11	-15.02 ± 8.93	-39.67	<.001
90°	35.65 ± 10.09	1.41 ± 10.71	-34.24	<.001
100°	52.50 ± 8.92	34.64 ± 11.12	-17.86	>.05
CONT press, N				
60°	1.03 ± 0.19	1.03 ± 0.21	0.01	>.05
70°	0.83 ± 0.14	0.75 ± 0.13	-0.08	>.05
80°	0.69 ± 0.10	0.62 ± 0.08	-0.07	>.05
90°	0.81 ± 0.12	0.76 ± 0.12	-0.05	>.05
100°	0.88 ± 0.11	0.88 ± 0.17	0.00	>.05
COR _{GH} , x, mm				
60°	-0.00 ± 0.31	-0.04 ± 0.25	-0.04	>.05
70°	-1.10 ± 0.53	-1.20 ± 0.57	-0.10	>.05
80°	-1.14 ± 0.56	-1.74 ± 0.90	-0.61	>.05
90°	-1.56 ± 0.99	-2.05 ± 0.95	-0.49	>.05
100°	-1.82 ± 1.01	-1.99 ± 1.07	-0.18	>.05
COR _{GH} , y, mm				
60°	-0.42 ± 0.47	-0.51 ± 0.37	-0.09	>.05
70°	-0.58 ± 1.12	-0.72 ± 1.12	-0.15	>.05
80°	-1.13 ± 2.00	-2.47 ± 1.88	-1.34	>.05
90°	-1.68 ± 2.61	-2.96 ± 2.48	-1.28	>.05
100°	-1.71 ± 2.68	-2.56 ± 2.65	-0.85	>.05
COR _{GH} , z, mm				
60°	-0.28 ± 0.10	-0.29 ± 0.39	-0.01	>.05
70°	-2.98 ± 0.74	-3.20 ± 1.11	-0.22	>.05
80°	-5.14 ± 1.07	-5.33 ± 1.89	-0.19	>.05
90°	-7.03 ± 1.74	-7.61 ± 2.50	-0.58	>.05
100°	-0.80 ± 1.82	-8.53 ± 2.65	-0.73	.01

^aCONT press, Tekscan contact pressure; COR_{GH}, glenohumeral center of rotation.

Subacromial Contact Pressure (Tekscan)

There was no change in the subacromial contact pressure during ABD following posterior plication in the intact shoulder (Figure 5 and Table 1).

Glenohumeral Kinematics (QUALYSIS Motion Capture System)

The posterior soft tissue plication did not significantly alter GH kinematics during passive ABD when examining an intact shoulder (Figure 6, A and B, and Table 1). The plication did result in a small increase (<1 mm) in superior translation that was statistically different at 100° of ABD ($P = .01$) (Figure 6C and Table 1). However, given the effect size of this change, its clinical relevance is uncertain.

Using an AUC analysis, plication did not affect the motion trajectory when compared with the baseline condition for all 3 axes ($P = .26, .84,$ and $.88$ for x -, y -, and z -axes, respectively) (Table 2).

All data were normally distributed, as assessed by the Shapiro-Wilk test ($P > .05$ for all cases).

DISCUSSION

Subacromial impingement is the most common disorder of the shoulder, accounting for 44% to 65% of all shoulder complaints.⁴⁵ When a posterior capsular contracture is present, internal rotation and ABD are limited. This change is believed to increase anterior and superior GH translation resulting in an increase in the subacromial contact pressure. We sought to quantify the effect of a posterior capsular contracture on subacromial contact pressure and GH kinematics during passive ABD of an intact cadaveric shoulder using an automated testing apparatus.^{12,20,23}

While prior cadaveric studies have described an increase in subacromial contact during passive shoulder ABD and external rotation,^{2,3,9} these changes were not observed in an intact cadaveric shoulder. This discrepancy may highlight the importance of the joint capsule's integrity and the joint's negative intra-articular pressure during cadaveric assessment of GH motion. Additionally, the difference may result from our use of an automated robotic system with continuous, real-time data collection. Interestingly, the humeral loading and kinematic data correspond to the in vivo behavior of the normal shoulder at baseline^{1,47} and the changes in humeral loading after the posterior soft tissue plication mirror that has been described during active joint motion.⁴⁸ Werner et al⁴⁶ found that these changes did not significantly alter GH translations during ABD.

Our results do not demonstrate a significant change in GH motion or subacromial contact pressure following posterior plication. In contrast to the results published by Poitras et al,³⁹ Mihata et al,²⁹ and Peltier et al,³⁸ our study showed a significant decrease in the superior displacement in the sagittal plane at 100° of ABD, while the center of contact and the magnitude of the contact pressure in the subacromial space before and after capsular plication demonstrated no discernible changes.

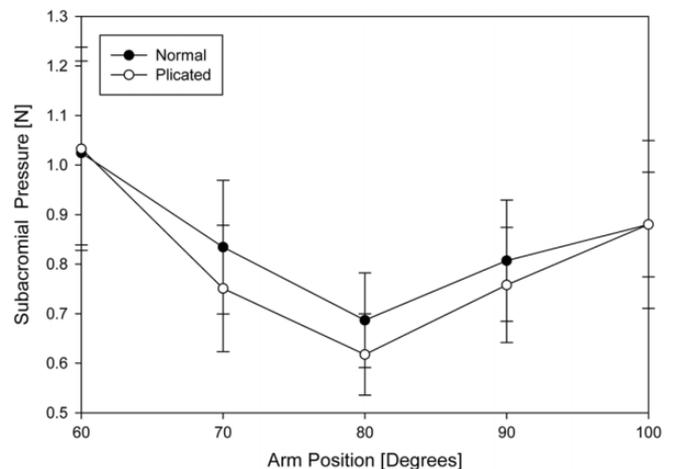


Figure 5. Subacromial pressure versus arm position between 60° and 100° of abduction.

The forces experienced at the load cell are measurements of the end-effector forces that are intrinsically related to the capsular integrity of the GH joint and thus provide a representation of the forces experienced at the humeral head. Our results demonstrate significant decrease in anterior and lateral forces measured at the load cell in the anterior-posterior and medial-lateral directions during ABD, suggesting increased compression, or load, at articulation (Figure 4, A-C). Plication resulted in a significant increase in axial load throughout ABD (Figure 4B).

Measuring subacromial contact pressure during ABD is an effective proxy for simulating shoulder impingement.^{17,35,37,39,46,50,51} Interestingly, the posterior capsular plication resulted in no significant difference in subacromial contact pressure. While there was a maximum at 60° and 100° of ABD—the beginning and end of the motion—the contact pressure decreased until it reached a minimum at 80° before climbing again to 100°. This trend may illustrate the interplay of the humeral head's ovoid geometry the ABD angle and subacromial pressure.

Similarly, Poitras et al³⁹ found that tightening the posterior capsule did not increase the subacromial contact pressure when loaded in discrete static steps. While our data were captured continuously, the findings are consistent with their claim that increased tightness of the posterior capsule does not cause an increase in subacromial contact pressure when the scapula is fixed (rigid).

As with any cadaveric model, this study has limitations. First, 6 shoulders represents a relatively small sample size. Despite this shortcoming, the observed trends in contact pressures and compressional forces were present throughout, as was the absolute displacement relative to arm position. In this study, 6 healthy, male, left shoulders were evaluated, but comparisons were not made to the contralateral limb. In the literature, 6 specimens have provided adequate insight.^{1,7,36}

Second, the precision of the measurements of the GH displacement depends on the reliability of the specimen's calibration. Because this process highlights individual anatomic landmarks on each specimen, there is inherent

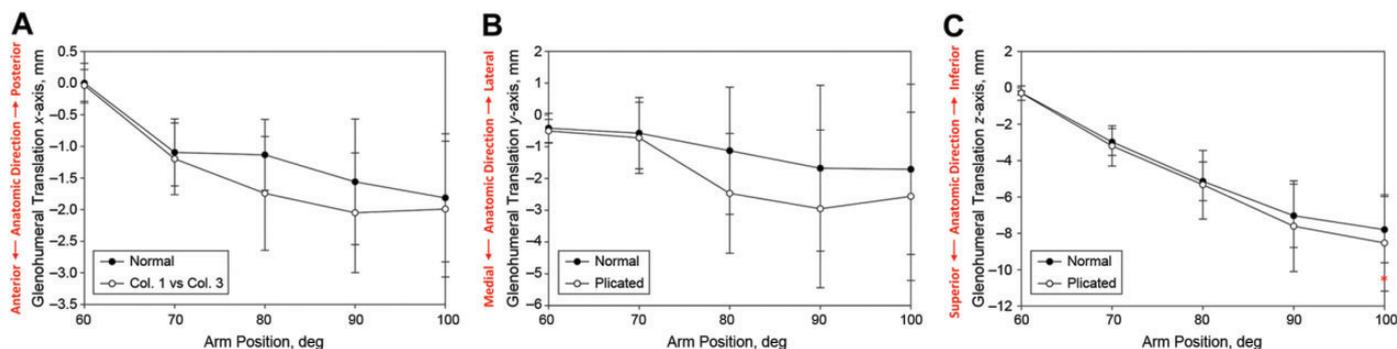


Figure 6. Glenohumeral center of rotation versus arm position for (A) x, (B) y, and (C) z planes between 60° and 100° of abduction.

TABLE 2
Area Under the Curve Data for Motion Segments
in the x, y, and z Directions

Motion Segment	Area Under the Curve, mm, Mean ± SD			
	x-Axis	y-Axis	z-Axis	
60°-70°	Control	1.168 ± 0.934	3.922 ± 2.927	2.699 ± 1.528
	Plicated	1.25 ± 0.924	4.035 ± 2.838	2.843 ± 1.444
70°-80°	Control	1.506 ± 1.273	3.896 ± 1.886	2.194 ± 1.052
	Plicated	1.151 ± 0.958	3.595 ± 1.938	2.123 ± 1.008
80°-90°	Control	2.456 ± 1.55	3.297 ± 1.745	2.276 ± 1.69
	Plicated	2.552 ± 1.474	3.237 ± 1.558	2.499 ± 1.604
90°-100°	Control	1.705 ± 1.371	1.551 ± 1.668	1.498 ± 0.829
	Plicated	1.862 ± 1.06	1.817 ± 1.624	1.584 ± 0.795

variability because they are landmarks rather than discrete points.⁶ The cadaveric shoulders offer a representation of the material and mechanical characteristics observed in vivo.²⁷

With the scapula rigidly fixed, each specimen’s range of motion was limited 40° of ABD. While the model affords the opportunity to examine various motion patterns, this investigation focused on ABD to best understand the interplay of GH motion, scapular kinematics, and subacromial impingement. Moreover, the effects of rotator cuff forces were not replicated in this passive model. Their relevance in extreme ranges of motion has not been questioned.²¹ The results and conclusions of this study should be interpreted with caution. Our system is capable of simulating gross motion through elevation, rotation, and spatial translation of anatomical landmarks. However, in vivo ABD represents a complex interaction of muscles acting over various joints in concert, making the individual contributions of various factors difficult to predict. However, this testing system offers a model of ABD that accurately represents the current clinical test for impingement.

Future investigations would benefit from dynamic rotator cuff loading during ABD as well as controlled scapulothoracic motion. These additions may help provide a comprehensive understanding of shoulder kinematics by more accurately simulating in vivo motion.

In this study, the scapula was rigidly held to isolate GH motion during ABD of the humerus. Posterior soft tissue

plication did not alter GH motion and, as a result, no significant change in subacromial contact pressure was observed. Suture plication constrained the joint’s motion, drawing the humeral head closer to the glenoid, increasing the load at the articulation (Figure 4C). These findings suggest that a posterior capsular contracture may cause increased subacromial contact pressure (impingement) due to compensatory change in scapulothoracic motion rather than a change in GH motion. Future studies with an intact shoulder and mobile scapula are needed to address this concern.

This investigation demonstrates that posterior capsular plication increases the axial load on the shoulder joint during ABD. While a significant difference from baseline was observed in the plicated condition, posterior capsular plication did not change GH motion or subacromial contact pressure significantly. In future studies, the effects of physiologically relevant peripheral musculoskeletal forces may offer greater insight into how a posterior capsular plication changes the shoulder’s motion to increase subacromial forces. Clinicians will benefit from this understanding of GH mechanics, which will help patients with shoulder pain and outlet impingement.

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