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ORIGINAL ARTICLE

Effects of asymptomatic rotator cuff pathology on in vivo shoulder motion and clinical outcomes

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Background: The incidence of asymptomatic rotator cuff tears has been reported to range from 15% to 39%, but the influence of asymptomatic rotator cuff pathology on shoulder function is not well understood. This study assessed the effects of asymptomatic rotator cuff pathology on shoulder kinematics, strength, and patient-reported outcomes.

Methods: A clinical ultrasound examination was performed in 46 asymptomatic volunteers (age: 60.3 ± 7.5 years) with normal shoulder function to document the condition of their rotator cuff. The ultrasound imaging identified the participants as healthy (n = 14) or pathologic (n = 32). Shoulder motion was measured with a biplane x-ray imaging system, strength was assessed with a Biodex (Biodex Medical Systems, Inc., Shirley, NY, USA), and patient-reported outcomes were assessed using the Western Ontario Rotator Cuff Index and visual analog scale pain scores.

Results: Compared with healthy volunteers, those with rotator cuff pathology had significantly less abduction ($P = .050$) and elevation ($P = .041$) strength, their humerus was positioned more inferiorly on the glenoid ($P = .018$), and the glenohumeral contact path length was longer ($P = .007$). No significant differences were detected in the Western Ontario Rotator Cuff Index, visual analog scale, range of motion, or acromiohumeral distance.

Conclusions: The differences observed between the healthy volunteers and those with asymptomatic rotator cuff pathology lend insight into the changes in joint mechanics, shoulder strength, and conventional clinical outcomes associated with the early stages of rotator cuff pathology. Furthermore, these findings suggest a plausible mechanical progression of kinematic and strength changes associated with the development of rotator cuff pathology.

Level of evidence: Basic Science Study; Kinesiology

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Rotator cuff tears affect approximately about 40% of individuals aged older than 60 years^{31,41,65} and account for an economic burden of \$3 to \$5 billion per year in the United States alone.^{53,59} Asymptomatic rotator cuff tears are

especially common. Specifically, the prevalence of asymptomatic rotator cuff tears in the general population has been reported to range from 15% to 39%, depending on age and specific imaging modality.^{41-43,47,49,52,55} Asymptomatic tears are estimated to be approximately twice as common as symptomatic tears.⁴²

Several studies have investigated the natural history of asymptomatic rotator cuff tears.^{23,24,38,44,63} These studies have been conducted by identifying subjects with an asymptomatic rotator cuff tear, monitoring those subjects longitudinally for several years, and then comparing characteristics of those who became symptomatic with those who remained asymptomatic. These studies have reported that approximately 25% to 50% of asymptomatic rotator cuff tears will become symptomatic during the course of 2 to 3 years.^{23,38,44,63} Compared with subjects whose rotator cuff tear is symptomatic, subjects with an asymptomatic rotator cuff tend to have higher functional scores (eg, American Shoulder and Elbow Surgeons, simple shoulder test),^{25,46} equal or greater range of motion (ROM) and strength during certain shoulder motions (eg, abduction, flexion, external rotation [ER]),^{25,32,43,64} less tear enlargement,^{23,38} less muscle degeneration,⁴⁴ and lower muscle activation levels during overhead activities.²⁷

In contrast to what is known about the effect of asymptomatic rotator cuff tears on conventional clinical outcomes (eg, ROM, strength, functional scores), relatively little is known about the effects of asymptomatic rotator cuff pathology on shoulder kinematics. Several previous studies have documented subtle changes in glenohumeral joint motion in patients whose asymptomatic rotator cuff tear became symptomatic,^{26,38,62} but studies focusing on differences in joint motion between subjects with an asymptomatic rotator cuff tear and healthy control subjects are less common. Yamaguchi et al⁶² have reported that patients with a rotator cuff tear have approximately 1 to 2 mm of superior humeral migration compared with healthy control subjects. More recently, Kijima et al³⁰ used biplanar fluoroscopy and 3-dimensional (3D)/2D image registration techniques to measure glenohumeral joint and scapular motion in healthy control subjects, patients with a symptomatic rotator cuff tear, and patients with an asymptomatic rotator cuff tear. This study reported that patients with a symptomatic rotator cuff tear had more anterior scapular tilt than healthy control subjects. However, no differences in glenohumeral joint or scapular motion were detected between the control subjects and patients with an asymptomatic tear, which may have been a result of sample size limitations.³⁰

The primary objective of this study was to assess the effects of asymptomatic rotator cuff pathology on shoulder kinematics, strength, and conventional patient-reported outcomes. We hypothesized that subjects with asymptomatic rotator cuff pathology would have lower strength, less ROM, and altered glenohumeral and scapulothoracic kinematics compared with individuals with an intact rotator cuff.

Materials and methods

This was a retrospective case-control study investigating the effects of pathology on the function and motion of asymptomatic shoulders.

Subject selection

The study enrolled 46 volunteers (22 men, 24 women) who were an average age of 60.3 ± 7.5 years (range, 42-77 years) and who denied any history of shoulder injury, surgery, or pain. A standard clinical ultrasound examination was performed to document the condition of the rotator cuff of each study participant. Two fellowship-trained musculoskeletal radiologists (M.v.H., D.S.S.) with significant experience in musculoskeletal imaging interpreted the ultrasound images.^{18,19,41,57,58,61} These images were used to separate the subjects into 2 groups: 14 control (CNTL) subjects with a healthy rotator cuff (average age, 58.0 ± 8.1 years; range, 42-72 years; 8 men, 6 women) and 32 subjects with rotator cuff pathology (PATH; average age, 61.4 ± 7.1 years; range, 51-77 years; 14 men, 18 women). Subjects were not informed of the ultrasound findings.

Subject testing

In vivo shoulder kinematics were measured using a biplane x-ray imaging system during frontal-plane abduction, starting with the subject's arm at his or her side and ending at approximately 120° of abduction. Subjects performed this shoulder motion while holding a 1-pound hand weight. Three trials were performed, with a minimum of 2 minutes between trials. Radiographic images of the shoulder were acquired at 60 Hz with the biplane x-ray system.⁶⁻¹⁰

A computed tomography scan of the humerus, scapula, and upper thorax was also acquired from each study participant. Scans were performed on a LightSpeed16 system (GE Medical Systems, Waukesha, WI, USA) with 1.25 mm slice spacing, approximately 30 cm field of view, and 512×512 pixel image size. The computed tomography images were manually segmented and reconstructed into 3D bone models for the humerus, scapula, and ribs 3 and 4 using Mimics 16.0 software (Materialise, Inc., Leuven, Belgium). The 3D locations of major anatomic landmarks were identified and used to define anatomic coordinate systems for the humerus, scapula, and thorax.⁶⁻⁹

Shoulder motion

The 3D position and orientation of the humerus, scapula, and ribs 3 and 4 were measured from the biplane x-ray images using model-based tracking, which has been shown to be accurate to within ± 0.4 mm and 0.5° .⁶⁻¹¹ Using these data, we measured conventional humerothoracic, glenohumeral, and scapulothoracic kinematics (ie, translations and rotations of the distal bone relative to the proximal bone).⁸ Humerothoracic and glenohumeral motions were expressed as the plane of elevation, the amount of elevation, and the amount of humeral rotation. Scapulothoracic motion was expressed as anterior/posterior (A/P) tilting, upward/downward rotation, and internal rotation (IR)/ER of the scapula relative to the thorax.²⁰⁻²² Scapulothoracic and glenohumeral ranges of motion were calculated using data from 20° to 105° of humerothoracic elevation. As a result of recent advances in our biplane x-ray image acquisition

protocol and analytic techniques,⁴ scapulothoracic kinematics were available for only 25 (9 CNTL and 16 PATH) of the 46 subjects.

Glenohumeral joint (GHJ) contact patterns were estimated by combining the joint motion data measured from biplane x-ray images with the patient-specific bone models.^{1-3,16,33,39,45} Briefly, the joint contact center was estimated by calculating the centroid of the minimum distance between the humerus and glenoid bone model surfaces for each frame of data and expressing the contact center position relative to the glenoid. By repeating this process for each frame, these calculations result in a contact path (ie, a time-series of GHJ contact center data). To account for differences in subject size, these estimates of the joint contact center were normalized relative to the glenoid height and width as determined from the patient-specific bone models. We used these joint contact center data to determine the dynamic contact location—that is, an estimate of the average position of the humerus on the glenoid during shoulder abduction—by calculating the average anterior/posterior (A/P) contact center and the average superior/inferior (S/I) contact center over each trial.

To assess dynamic joint excursion (ie, the amount of GHJ translation that occurred during shoulder motion), we calculated the A/P contact center range, the S/I contact center range, and the contact center path length over each trial.^{7,9} The technique for estimating GHJ contact patterns was also used to determine the acromiohumeral distance (AHD). Specifically, we calculated the shortest distance between the humeral and acromial surfaces for every frame of data and then computed the average distance over the entire trial.⁶ The GHJ contact center data and acromiohumeral data were determined using data from 20° to 70° of glenohumeral elevation.

Clinical outcomes

For each study participant, isometric shoulder strength was measured during coronal-plane abduction at 30° of abduction, sagittal-plane elevation at 30° of elevation, IR at 15° of frontal-plane elevation and 0° of humeral rotation, and ER at 15° of frontal-plane elevation and 0° of humeral rotation with a Biodex System 2 isokinetic dynamometer (Biodex Medical Systems, Inc., Shirley, NY, USA).⁹ Three trials were performed at each position. In addition, patient-reported outcomes were assessed using the Western Ontario Rotator Cuff Index (WORC) score and a 10-cm visual analog scale (VAS) for pain. Lastly, active and passive ROM were manually measured with a goniometer for frontal-plane abduction, sagittal-plane elevation, IR, and ER. The IR and ER measurements were performed from a starting position of 90° of frontal-plane abduction and the forearm parallel to the ground.

Statistical analysis

The effects of rotator cuff pathology on the measures of shoulder motion, strength, and ROM were assessed with an unpaired *t* test. Significance was set at $P \leq .05$.

Results

The study revealed several interesting differences the PATH subjects were compared with the CNTL subjects (Table I). Specifically, the PATH subjects had significantly less abduction ($P = .050$) and elevation ($P = .041$) strength (Fig. 1). In

addition, the PATH subjects' GHJ contact center path length was significantly longer ($P = .007$; Fig. 2, A, Table I), and their average S/I GHJ contact center was positioned more inferiorly on the glenoid ($P = .018$; Fig. 2, B, Table I).

For the conventional clinical outcome measures, no significant differences were detected between the CNTL and PATH subjects in age ($P = .162$), composite WORC score ($P = .165$), or VAS score ($P = .465$). No significant differences were detected between the CNTL and PATH subjects in any of the measures of active or passive ROM ($P > .324$, Table I) or in ER ($P = .106$) or IR ($P = .083$) strength.

Results of joint kinematics showed the PATH subjects' scapula was more anteriorly tilted than the CNTL subjects' (PATH: $13 \pm 8^\circ$, CNTL: $19 \pm 11^\circ$), but this difference did not achieve statistical significance ($P = .158$, Fig. 3). No significant differences were detected between CNTL and PATH subjects in scapulothoracic IR/ER ROM ($P = .470$), scapulothoracic A/P tilt ROM ($P = .768$), or scapulothoracic upward rotation ROM ($P = .690$). Similarly, no significant difference was detected between the CNTL and PATH subjects in the average scapulothoracic IR/ER ($P = .559$) or average scapulothoracic upward rotation ($P = .926$). No significant difference was detected between the CNTL and PATH subjects in the glenohumeral elevation ROM ($P = .645$) or the average glenohumeral elevation ($P = .484$).

The arthrokinematic data indicated that the humerus' path of contact on the glenoid moved primarily in the S/I direction during shoulder elevation for both the CNTL and PATH subjects (Fig. 2, A). No statistically significant differences were detected between the 2 subject populations in S/I contact center range ($P = .238$), the average A/P contact center location ($P = .699$), or the A/P contact center range ($P = .335$). The PATH subjects' average AHD was slightly less than that of the CNTL subjects' (PATH: 3.8 ± 1.4 mm, CNTL: 4.2 ± 1.7 mm), but this difference did not reach statistical significance ($P = .093$; Fig. 4, Table I). Lastly, no significant difference was detected between the CNTL and PATH subjects in the range of AHD ($P = .492$; Fig. 4, Table I).

Discussion

The primary objective of this study was to assess the effects of asymptomatic rotator cuff pathology on shoulder kinematics, strength, and patient-reported outcomes. The study failed to detect statistically significant differences between the 2 asymptomatic subject groups for many of the outcome measures, but subjects with asymptomatic rotator cuff pathology were found to have (1) lower abduction and elevation strength (Fig. 1), (2) a more inferiorly positioned humerus on the glenoid (Fig. 2), and (3) a longer GHJ contact center path length compared with subjects with an intact rotator cuff.

Previous research indicates that decreases in shoulder strength occur secondary to pain,^{5,48,60} which makes the findings from the current study of decreased strength in the shoulders of the asymptomatic PATH subjects particularly

Table I Clinical outcome measures

Category ^a	Outcome measure	CNTL	PATH	P value [†]
Age, y	Average	58.0 ± 8.1	61.4 ± 7.1	.162
	Range	42-72	51-77	—
Patient-reported outcomes	WORC	97.0 ± 4.1	93.1 ± 9.7	.165
	VAS	0.0 ± 0.0	0.1 ± 0.3	.465
Active ROM, °	Coronal-plane abduction	180 ± 0	180 ± 0.0	1.000
	Sagittal-plane elevation	180 ± 0	180 ± 0.0	1.000
	ER	103 ± 7	101 ± 10	.662
	IR	66 ± 13	64 ± 9	.607
Passive ROM, °	Coronal-plane abduction	180 ± 0	180 ± 0	1.00
	Sagittal-plane elevation	180 ± 0	180 ± 0	1.00
	ER	104 ± 4	103 ± 14	.799
	IR	55 ± 11	61 ± 13	.324
Strength, ft-lb	Coronal-plane abduction	30 ± 14	21 ± 11	.050
	Sagittal-plane elevation	31 ± 17	22 ± 13	.041
	ER	19 ± 8	15 ± 6	.106
	IR	26 ± 10	20 ± 8	.083
Scapulothoracic motion, °	IR/ER ROM	12 ± 3	11 ± 6	.470
	Average IR/ER	-42 ± 9	-44 ± 6	.559
	A/P tilt ROM	24 ± 6	25 ± 5	.768
	Average A/P tilt	19 ± 11	13 ± 8	.158
	Upward rotation ROM	27 ± 3	26 ± 7	.690
	Average upward rotation	22 ± 9	22 ± 6	.926
GHJ motion, °	Elevation ROM	43 ± 8	44 ± 6	.645
	Average elevation	55 ± 6	53 ± 6	.484
	Average S/I position	9.9 ± 6.1	3.1 ± 9.5	.018
GHJ contact center, % of glenoid height or width	S/I range	11.7 ± 5.4	14.0 ± 6.0	.238
	Average A/P position	-7.2 ± 5.1	-6.1 ± 10.0	.699
	A/P range	8.5 ± 3.8	10.1 ± 5.4	.335
	Path length	28.0 ± 10.3	37.1 ± 9.4	.007
	Average Distance	4.2 ± 1.7	3.8 ± 1.4	.093
Acromiohumeral distance, mm	Average Range	3.7 ± 1.3	3.8 ± 1.5	.492

A/P, anterior/posterior; CNTL, control group; ER, external rotation; GHJ, glenohumeral joint; IR, internal rotation; PATH, group with shoulder pathology; ROM, range of motion; S/I, superior/inferior; VAS, visual analog scale; WORC, Western Ontario Rotator Cuff Index.

* Data are shown as the mean ± standard deviation unless indicated otherwise.

† Bold numbers indicate statistical significance ($P \leq .05$).

interesting. Kim et al³² studied subjects with an asymptomatic rotator cuff tear and reported mixed findings regarding shoulder strength. Specifically, they reported no difference in abduction and ER strength between asymptomatic shoulders with a unilateral partial- or full-thickness tear and the intact contralateral shoulder, but significantly lower abduction strength in asymptomatic shoulders with a large/massive rotator cuff tear compared with the intact contralateral shoulder. The findings from the current study tend to suggest that decreased shoulder strength, independent of pain, may be one of the early functional changes associated with the development of rotator cuff pathology. In addition, in contrast to the Kim et al³² study, the findings from the current study would suggest that small tears or even tendinosis alone can have an appreciable effect on shoulder strength.

The GHJ contact paths (Fig. 2, A) and average joint contact centers (Fig. 2, B) are consistent with previous studies that have reported GHJ arthrokinematic data^{9,12,40} and provide insight into functional deficits associated with the early, as-

ymptomatic stage of rotator cuff pathology. Previous research has shown that the supraspinatus stabilizes the humerus against the glenoid during shoulder motion and, in particular, is an important stabilizer against inferior GHJ translation.⁵⁴ Consequently, it is perhaps not surprising that the average GHJ contact center of the PATH subjects was positioned lower on the glenoid compared with the CNTL subjects (Fig. 2, B).

We recognize that this finding of inferior translation of the humerus relative to the glenoid is in stark contrast to the common clinical presentation of superior humeral migration in patients with a rotator cuff tear. However, we believe that the clinical presentation of superior humeral migration represents a more advanced stage of rotator cuff pathology consistent with larger full-thickness tears. In contrast, the findings reported here represent the initial functional changes in GHJ mechanics indicating that joint stabilizing function of the supraspinatus has been compromised despite the absence of symptoms.

Scapular dyskinesia—in particular, anterior tilting of the scapula—is frequently reported in patients with rotator cuff

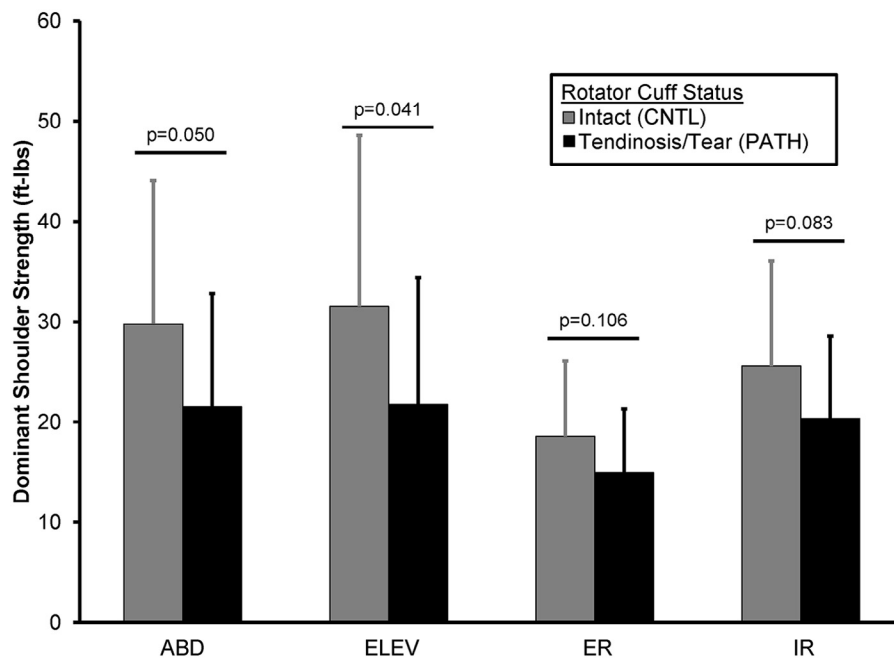


Figure 1 Subjects with asymptomatic rotator cuff pathology (*black bars*) had significantly lower strength in coronal-plane abduction (*ABD*; $P = .05$) and sagittal-plane elevation (*ELEV*; $P = .041$) than subjects with an intact rotator cuff (*gray bars*). The error bars show the standard deviation. *ER*, external rotation; *IR*, internal rotation.

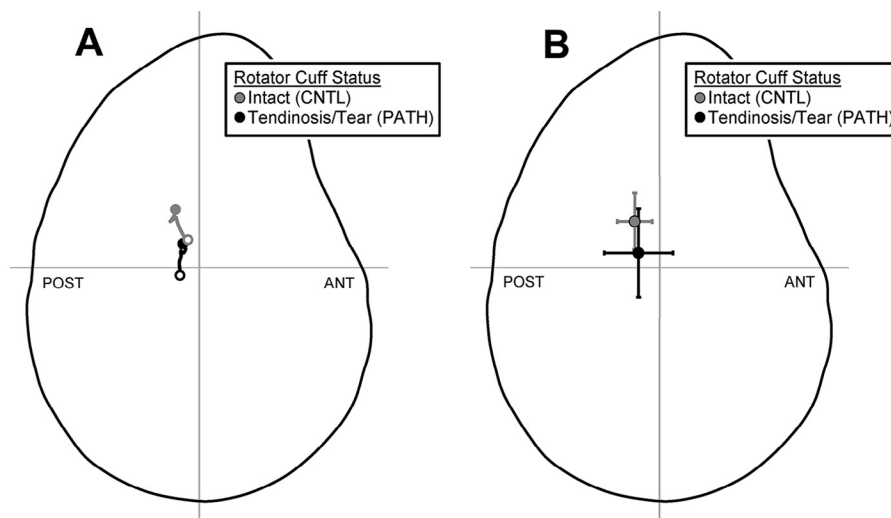


Figure 2 (A) The lines superimposed on the glenoid indicate the path of joint contact during shoulder elevation for the control subjects (*CNTL*) and for the subjects with asymptomatic rotator cuff pathology (*PATH*). In each figure, the *open circle* (○) indicates the center of contact at 20° of glenohumeral elevation and the *closed circle* (●) indicates the center of contact at 70° of glenohumeral elevation. (B) Compared with the *CNTL* subjects, the average joint contact center of the *PATH* subjects was positioned more inferiorly on the glenoid ($P = .018$). *ANT*, anterior; *POST*, posterior

pathology.^{15,30,34-36} The current study found the scapula was anteriorly tilted approximately 6° more in the *PATH* subjects than in the *CNTL* subjects, although this finding did not achieve statistical significance ($P = .10$, Fig. 3). Increased anterior tilt is consistent with the work of Ludewig and Cook,³⁴ who reported that differences in scapular A/P tilt between subjects with and without impingement symptoms ranged from approximately 2° to 6°, depending on the elevation angle. However,

the clinical significance of a 6° difference in A/P tilt between *PATH* and *CNTL* subjects is unclear. Previous research has suggested that 3° of scapular rotation is the minimal clinically important change,^{13,14,37} but Uhl et al⁵⁶ reported that scapular asymmetry—defined as a difference of 7° to 9°, depending on the specific rotation direction—is present in 71% to 77% of subjects. Moreover, the prevalence of scapular asymmetry did not differ between symptomatic and asymptomatic subjects.⁵⁶

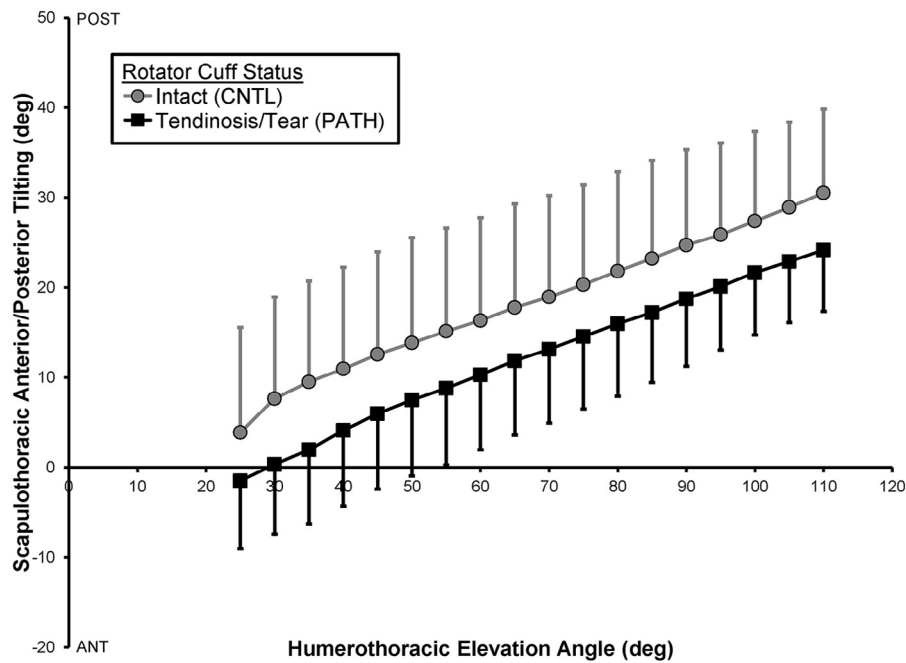


Figure 3 The scapula tilted posteriorly with increasing elevation angle for control subjects (*CNTL*) and for the subjects with rotator cuff pathology (*PATH*). There was a statistical trend for the average scapulothoracic position to be more anteriorly tilted in the *PATH* subjects ($13^\circ \pm 8^\circ$) compared with the *CNTL* subjects ($19^\circ \pm 11^\circ$, $P = .158$). The *error bars* show the standard deviation.

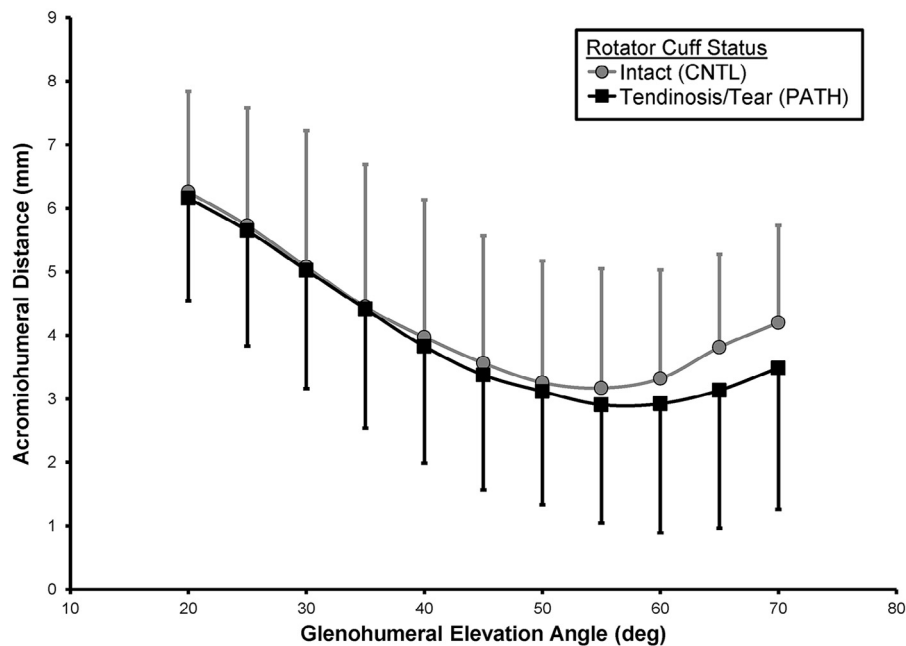


Figure 4 No significant difference in acromiohumeral distance was detected between the subjects with an intact rotator cuff (*CNTL*) and subjects with a rotator cuff tear or tendinosis (*PATH*). The *error bars* show the standard deviation.

Consequently, it remains unclear whether a 6° difference in A/P tilt would even be detected during a clinical examination or whether that difference would be interpreted as being outside the normal range of intershoulder variability.

Initially, it was surprising that the study failed to detect a statistically significant difference in AHD between the *PATH* and *CNTL* subjects ($P = .093$, Fig. 4). The *PATH* subjects'

humerus was positioned 6.8% of the glenoid height (or approximately 2.6 mm assuming a glenoid height of 39 mm¹⁷) more inferiorly on the glenoid than the *CNTL* subjects' average GHJ contact center (Fig. 2, B), which should have increased the AHD. However, the *PATH* subjects' scapula was tilted anteriorly about 6° compared with the *CNTL* subjects, and previous research has shown that anterior tilting

of the scapula will decrease the AHD.^{37,50,51} In particular, the data reported by Seitz et al^{50,51} indicates that a 5° change in A/P tilt of the scapula results in about a 2-mm change in the AHD. Taken together, the increased AHD caused by the humerus being positioned more inferiorly on the glenoid may be offset by the increase in anterior tilt of the scapula. However, it is important to acknowledge that the AHD is likely influenced by more than just the A/P tilt of the scapula and that the precise association between AHD and scapular kinematics is still not fully understood.

Although the study failed to detect statistically significant differences between the 2 asymptomatic populations for many of the outcome measures, the differences that were detected suggest a plausible progression of rotator cuff pathology. First, regardless of the specific initiating event(s), intrinsic supraspinatus tendon degeneration—typically identified clinically as tendinosis—appears to be the earliest indication of rotator cuff pathology. In turn, this tendon degeneration (or a small tear) likely leads to functional deficits that we suspect are related. Initially, we suspect that the degenerated/torn supraspinatus tendon is structurally compromised in a way that prevents it from fully transmitting muscle forces, thus contributing to a decrease in shoulder strength (Fig. 1).

Next, the decrease in supraspinatus tendon forces leads to subtle alterations in GHJ motion, as evidenced by the humerus positioned more inferiorly on the glenoid (Fig. 2, B). These subtle changes in GHJ motion further exacerbate the issue of decreased shoulder strength, because the GHJ is no longer in an optimal configuration to provide a stable base of support necessary to generate high muscle forces. At some point during the progression of rotator cuff pathology, the scapula becomes tilted more anteriorly (Fig. 3), although whether abnormal scapular motion contributes to the development of rotator cuff pathology or is the result of rotator cuff pathology is still unknown.^{28,29} There is evidence that anterior tilting of the scapula decreases the AHD and, presumably, increases the likelihood of symptoms due to subacromial impingement,^{37,50,51} but the more inferior positioning of the humerus on the glenoid appears to preserve the AHD (Fig. 4). Consequently, the ongoing biologic processes associated with rotator cuff tendon degeneration or tear propagation, or both, are able to proceed in the absence of impingement-related symptoms.

This mechanism is speculative but may help to explain why such a high number of rotator cuff tears remain asymptomatic.⁴² This mechanism would also suggest that exercises designed to restore normal scapular motion may increase the AHD sufficiently to minimize or eliminate impingement-related symptoms and the need for surgery. Furthermore, it is possible that some measure of scapular kinematics may be the best predictor of the presence/absence of symptoms in individuals with a rotator cuff tear. Clearly, further research is needed to more precisely understand the relationships between tendon pathology, GHJ motion, scapulothoracic motion, AHD, and symptoms.

As a secondary analysis aimed at lending further insight into the progression of asymptomatic rotator cuff pathology,

the PATH subjects were subdivided into those with tears ($n = 16$; partial-thickness: $n = 11$, full-thickness: $n = 5$) and those with tendinosis ($n = 16$) and compared using an unpaired t test. Interestingly, the only difference between these 2 groups was that active ER ROM of the subjects with tendinosis ($96^\circ \pm 7^\circ$) was significantly less than that of the subjects with a rotator cuff tear ($109^\circ \pm 7^\circ$, $P = .003$). For comparison, the CNTL subjects with a healthy rotator cuff had an active ER ROM of $103^\circ \pm 7^\circ$, which is almost exactly midway between the tendinosis and rotator cuff tear subjects. How increasing levels of asymptomatic rotator cuff pathology would first be associated with a loss of ER and then a subsequent and substantial increase in ER ROM is not obvious. An asymptomatic rotator cuff tear may destabilize the GHJ in a way that preferentially affords an increase in ER, but further research would be necessary to confirm this speculation. Alternatively, this finding may be a statistical anomaly caused by the limited sample size of each group.

This study has several limitations. First, the sample sizes were relatively small, although several statistically significant differences and trends were detected between the subject populations. Second, the data were acquired at only a single assessment, and therefore, estimating disease progression for the subjects with asymptomatic rotator cuff pathology was not possible. Third, the study failed to assess strength of the lower scapular stabilizing muscles (eg, rhomboids, serratus anterior, lower trapezius), and therefore, this study is unable to comment on the extent to which the strength of these muscles is associated with altered joint motion or rotator cuff pathology.

In summary, this study demonstrated that individuals with asymptomatic rotator cuff pathology have significantly lower abduction and elevation strength, a more inferiorly positioned humerus on the glenoid, and a longer path of contact between the humerus and glenoid compared with age-matched individuals with an intact rotator cuff. The findings from the study lend insight into the changes in joint mechanics, shoulder strength, and conventional clinical outcomes associated with the early stages of rotator cuff pathology.

Conclusions

The differences observed between the healthy subjects and subjects with asymptomatic rotator cuff pathology lend insight into the changes in joint mechanics, shoulder strength, and conventional clinical outcomes associated with the early stages of rotator cuff pathology. Specifically, in the PATH subjects, functional deficits in strength were observed along with changes in humeral contact on the glenoid, all in the absence of pain. Additional research is necessary to more fully understand the relationship between in vivo measures of shoulder function and the development of symptoms in individuals with rotator cuff pathology.

Disclaimer

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