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Effects of Asymptomatic Rotator Cuff Pathology On In-Vivo Shoulder Motion and Clinical Outcomes

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Abstract

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

Methods—46 asymptomatic volunteers (age: 60.3±7.5) with normal shoulder function underwent a clinical ultrasound exam to document the condition of their rotator cuff. The ultrasound imaging identified subjects 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, and patient-reported outcomes were assessed using the Western Ontario Rotator Cuff Index and VAS pain scores.

Results—Compared to healthy subjects, the subjects with rotator cuff pathology had significantly less abduction (p=0.050) and elevation (p=0.041) strength, their humerus was positioned more inferiorly on the glenoid (p=0.018), and the glenohumeral contact path length was longer (p=0.007). No significant differences were detected in terms of WORC, VAS, ROM, or acromiohumeral distance.

Discussion—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

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DISCLAIMER: none

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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

Keywords

Shoulder; Rotator cuff; Biomechanics; Motion Analysis; Clinical outcomes; Asymptomatic

Rotator cuff tears affect approximately about 40% of individuals over age 60^{31, 41, 65} and account for an economic burden of \$3–5 billion per year in the U.S. 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 15–39% depending on age and specific imaging modality^{41–43, 47, 49, 52, 55}. It has been estimated that asymptomatic tears are approximately twice as common as symptomatic tears⁴².

The natural history of asymptomatic rotator cuff tears has been investigated in several studies^{23, 24, 38, 44, 63}. These studies have been conducted by identifying subjects with an asymptomatic rotator cuff tear, following 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 over the course of 2 to 3 years^{23, 38, 44, 63}.

Compared to subjects whose rotator cuff tear is symptomatic, subjects with an asymptomatic rotator cuff tend to have higher functional scores (e.g., ASES, SST)^{25, 46}, equal or greater ROM and strength during certain shoulder motions (e.g., abduction, flexion, external rotation)^{25, 32, 43, 64}, less tear enlargement^{23, 38}, less muscle degeneration⁴⁴, and lower muscle activation levels during overhead activities²⁷.

In contrast to what is known about the impact of asymptomatic rotator cuff tears on conventional clinical outcomes (e.g., 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 and colleagues have reported that patients with a rotator cuff tear have approximately 1–2 mm of superior humeral migration compared to healthy control subjects⁶². More recently, Kijima and colleagues used biplanar fluoroscopy and 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 due to sample size limitations³⁰.

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 when compared to individuals with an intact rotator cuff.

MATERIALS AND METHODS

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

Subject Selection

Following IRB approval, 46 volunteers (average age: 60.3 ± 7.5 , range: 42–77, 22 male, 24 female) who denied any history of shoulder injury, surgery, or pain enrolled in the study. A standard clinical ultrasound exam was performed to document the condition of the rotator cuff of each study participant. Interpretation of the ultrasound images was performed by two fellowship-trained musculoskeletal radiologists (M.v.H., D.S.S.) with significant experience in musculoskeletal imaging^{18, 19, 41, 57, 58, 61}. Based on these images, the subjects were separated into two groups: control (CNTL) subjects with a healthy rotator cuff ($n=14$, average age: 58.0 ± 8.1 , range: 42–72, 8 male, 6 female) and subjects with rotator cuff pathology (PATH, $n=32$, average age: 61.4 ± 7.1 , range: 51–77, 14 male, 18 female). 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/her side and ending at approximately 120° of abduction. Subjects performed this shoulder motion while holding a 1-pound hand weight. Three trials were acquired, with a minimum of two minutes between trials. Radiographic images of the shoulder were acquired at 60 Hz with the biplane x-ray system^{6–10}. A CT 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) with 1.25 mm slice spacing, approximately 30 cm FOV and 512×512 pixel image size. The CT images were manually segmented and reconstructed into 3D bone models for the humerus, scapula, and ribs 3 and 4 (Mimics 16.0, Materialise, Inc., Leuven, Belgium). The 3D locations of major anatomical landmarks were identified and used to define anatomical 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 (i.e., translations and rotations of the distal bone relative to the proximal bone)⁸. Humerothoracic and glenohumeral motions were expressed in terms of the plane of elevation, the amount of elevation, and the amount of humeral rotation. Scapulothoracic motion was expressed in

terms of anterior/posterior (A/P) tilting, upward/downward rotation, and internal/external rotation 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. Due to recent advances in our biplane x-ray image acquisition protocol and analytical techniques⁴, scapulothoracic kinematics were available for only 25 of the 46 subjects (CNTL: n=9, PATH: n=16).

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 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, i.e., 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. Using these joint contact center data, we determined 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 (i.e., 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. 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 trial⁶. 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 (ABD) at 30° of abduction, sagittal-plane elevation (ELEV) at 30° of elevation, internal rotation (IR) at 15° of frontal-plane elevation and 0° of humeral rotation, and external rotation (ER) at 15° of frontal-plane elevation and 0° of humeral rotation with an isokinetic dynamometer (Biodex System 2)⁹. Three trials were acquired at each position. In addition, patient-reported outcomes were assessed using the Western Ontario Rotator Cuff (WORC) index and a 10 cm visual-analog scale for pain. Lastly, active and passive ROM were manually measured with a goniometer for frontal-plane ABD, sagittal-plane ELEV, 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 $p = 0.050$.

RESULTS

The study revealed several interesting differences when comparing the subjects with rotator cuff pathology to the healthy control subjects (Table 1). Specifically, the subjects with rotator cuff pathology had significantly less ABD ($p=0.050$) and ELEV ($p=0.041$) strength (Figure 1). In addition, the PATH subjects' glenohumeral joint contact center path length was significantly longer ($p=0.007$, Figure 2A, Table 1), and their average superior/inferior glenohumeral joint contact center was positioned more inferiorly on the glenoid ($p=0.018$, Figure 2B, Table 1). In terms of conventional clinical outcome measures, no significant differences were detected between the CNTL and PATH subjects in terms of age ($p=0.162$), composite WORC score ($p=0.165$), or VAS score ($p=0.465$). No significant differences were detected between the CNTL and PATH subjects in any of the measures of active or passive ROM ($p>0.324$, Table 1). No significant difference in ER ($p=0.106$) or IR ($p=0.083$) strength was detected between the CNTL and PATH subjects.

In terms of joint kinematics, 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 ($p=0.158$, Figure 3). No significant differences were detected between CNTL and PATH subjects in scapulothoracic internal/external rotation ROM ($p=0.470$), scapulothoracic A/P tilt ROM ($p=0.768$), or scapulothoracic upward rotation ROM ($p=0.690$). Similarly, no significant difference was detected between the CNTL and PATH subjects in the average scapulothoracic internal/external rotation ($p=0.559$) or average scapulothoracic upward rotation ($p=0.926$). No significant difference was detected between the CNTL and PATH subjects in the glenohumeral elevation ROM ($p=0.645$) or the average glenohumeral elevation ($p=0.484$).

The arthrokinematic data indicated that the humerus' path of contact on the glenoid moved primarily in the superior/inferior direction during shoulder elevation for both the CNTL and PATH subjects (Figure 2A). No statistically significant differences were detected between the two subject populations in terms of the S/I contact center range ($p=0.238$), the average A/P contact center location ($p=0.699$), or the A/P contact center range ($p=0.335$). The PATH subjects' average acromiohumeral distance 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 the level of statistical significance ($p=0.093$, Figure 4, Table 1). Lastly, no significant difference was detected between the CNTL and PATH subjects in the range of acromiohumeral distance ($p=0.492$, Figure 4, Table 1).

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 two asymptomatic subject groups for many of the outcome measures, but subjects with asymptomatic rotator cuff pathology were found to have: 1) lower ABD and ELEV strength (Figure 1), 2) a more inferiorly positioned humerus on the glenoid (Figure 2), and 3) a longer glenohumeral joint contact center path length when compared to 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 interesting. Kim and colleagues studied subjects with an asymptomatic rotator cuff tear and reported mixed findings regarding shoulder strength³². Specifically, Kim et al. reported no difference in ABD and ER strength between asymptomatic shoulders with a unilateral partial- or full-thickness tear and the intact contralateral shoulder, but significantly lower ABD strength in asymptomatic shoulders with a large/massive rotator cuff tear when 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 al study, the findings from the current study would suggest that small tears or even tendinosis alone can have an appreciable impact on shoulder strength.

The GHJ contact paths (Figure 2A) and average joint contact centers (Figure 2B) are consistent with previous studies that have reported GHJ arthrokinematic data^{9, 12, 40} and provide insight into functional deficits associated with the early, asymptomatic 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 translation⁵⁴. Consequently, it is perhaps not surprising that the average GHJ contact center of the PATH subjects is positioned lower on the glenoid when compared to the CNTL subjects (Figure 2B). 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 dyskinesis – in particular, anterior tilting of the scapula – is frequently reported in patients with rotator cuff pathology (e.g.,^{15, 30, 34–36}). The current study found the scapula to be anteriorly tilted approximately 6° more in the PATH subjects than in the CNTL subjects, although this finding did not achieve statistical significance ($p=0.10$, Figure 3). Increased anterior tilt is consistent with the work of Ludewig and Cook who reported that differences in scapular A/P tilt between subjects with and without impingement symptoms ranged from about 2–6°, depending on elevation angle³⁴. 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 and colleagues reported that scapular asymmetry – defined as a difference of 7° to 9°, depending on the specific rotation direction – is present in 71%–77% of subjects⁵⁶. Moreover, the prevalence of scapular asymmetry was not found to differ between symptomatic and asymptomatic subjects⁵⁶. Consequently, it remains unclear if a 6° difference in A/P tilt would even be detected during a clinical exam or if that difference would be interpreted as being outside the normal range of inter-shoulder variability.

Initially, it was surprising that the study failed to detect a statistically significant difference in AHD between the PATH and CNTL subjects ($p=0.093$, Figure 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 (Figure 2B), which should have increased the AHD. However, the PATH subjects' scapula was tilted anteriorly about 6° compared to 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 and colleagues indicates that a 5° change in A/P tilt of the scapula results in about a 2 mm change in AHD^{50, 51}. Taken together, it appears that the increased AHD due to 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 two 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 (Figure 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 (Figure 2B). These subtle changes in GHJ motion further exacerbate the issue of decreased shoulder strength, since 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 (Figure 3), though it is still unknown if abnormal scapular motion contributes to the development of rotator cuff pathology or is the result of rotator cuff pathology^{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 (Figure 4). Consequently, the on-going biologic processes associated with rotator cuff tendon degeneration and/or tear propagation are able to proceed in the absence of impingement-related symptoms. Although this mechanism is speculative, it may help to explain why such a high number of rotator cuff tears remain asymptomatic⁴². 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 sub-divided 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 two groups was that active ER ROM of the subjects with tendinosis ($96 \pm 7^\circ$) was significantly less than that of the subjects with a rotator cuff tear ($109 \pm 7^\circ$, $p=0.003$). For comparison, the CONT subjects with a healthy rotator cuff had an active ER ROM of $103 \pm 7^\circ$, i.e., almost exactly midway between the tendinosis and rotator cuff tear subjects. It's not obvious 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. It may be that an asymptomatic rotator cuff tear destabilizes the glenohumeral joint 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 due to the limited sample size of each group.

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

In summary, this study demonstrated that individuals with asymptomatic rotator cuff pathology have significantly lower ABD and ELEV strength, a more inferiorly positioned humerus on the glenoid, and a longer path of contact between the humerus and glenoid compared to 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 pathologic 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.

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Figure 1.

Subjects with asymptomatic rotator cuff pathology had significantly lower strength in coronal-plane abduction ($p=0.050$) and sagittal-plan elevation ($p=0.041$) than subjects with an intact rotator cuff.

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 to the CNTL subjects, the average joint contact center of the PATH subjects was positioned more inferiorly on the glenoid ($p=0.018$).

Figure 3.

The scapula tilted posteriorly with increasing elevation angle for both the CNTL and PATH subjects. There was a statistical trend for the average scapulothoracic position to be more anteriorly tilted in the PATH subjects ($13 \pm 8^\circ$) compared to the CNTL subjects ($19 \pm 11^\circ$, $p=0.158$).

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).

Table I

Mean±standard deviation of clinical outcome measures.

Category	Outcome Measure	CNTL	PATH	p-value
age	average	58.0 ± 8.1	61.4 ± 7.1	0.162
	range	42 – 72	51 – 77	--
patient-reported outcomes	WORC	97.0 ± 4.1	93.1 ± 9.7	0.165
	VAS	0.0 ± 0.0	0.1 ± 0.3	0.465
active ROM (degrees)	ABD	180 ± 0	180 ± 0.0	1.000
	ELEV	180 ± 0	180 ± 0.0	1.000
	ER	103 ± 7	101 ± 10	0.662
	IR	66 ± 13	64 ± 9	0.607
passive ROM (degrees)	ABD	180 ± 0	180 ± 0	1.00
	ELEV	180 ± 0	180 ± 0	1.00
	ER	104 ± 4	103 ± 14	0.799
	IR	55 ± 11	61 ± 13	0.324
strength (ft-lb)	ABD	30 ± 14	21 ± 11	0.050
	ELEV	31 ± 17	22 ± 13	0.041
	ER	19 ± 8	15 ± 6	0.106
	IR	26 ± 10	20 ± 8	0.083
scapulothoracic motion (degrees)	int/ext ROM	12 ± 3	11 ± 6	0.470
	average int/ext rotation	−42 ± 9	−44 ± 6	0.559
	ant/post tilt ROM	24 ± 6	25 ± 5	0.768
	average ant/post tilt	19 ± 11	13 ± 8	0.158
	upward rotation ROM	27 ± 3	26 ± 7	0.690
	average upward rotation	22 ± 9	22 ± 6	0.926
GHJ motion (degrees)	elevation ROM	43 ± 8	44 ± 6	0.645
	avg. elevation	55 ± 6	53 ± 6	0.484
GHJ contact center (% of glenoid height or glenoid width)	average S/I position	9.9 ± 6.1	3.1 ± 9.5	0.018
	S/I range	11.7 ± 5.4	14.0 ± 6.0	0.238
	average A/P position	−7.2 ± 5.1	−6.1 ± 10.0	0.699
	A/P range	8.5 ± 3.8	10.1 ± 5.4	0.335
	path length	28.0 ± 10.3	37.1 ± 9.4	0.007
acromiohumeral distance (mm)	average	4.2 ± 1.7	3.8 ± 1.4	0.093
	range	3.7 ± 1.3	3.8 ± 1.5	0.492

VAS = visual analog scale, WORC = Western Ontario Rotator Cuff index, ABD = coronal-plane abduction, ELEV = sagittal-plane elevation, ER = external rotation, and IR = internal rotation. Bold numbers indicate $p < 0.050$.