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Original Article

Participants with restricted dominant shoulder internal rotation range of motion demonstrate no side-to-side difference in humeral head translation; and no difference before and after joint mobilization: a pilot study

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Abstract. [Purpose] To compare humeral head translation (HHT) during shoulder elevation between dominant and non-dominant shoulders in participants with limited dominant shoulder internal rotation range of motion (ROM). To determine if joint mobilization alters HHT, and if relationships exist between the bicipital forearm angle and HHT. [Participants and Methods] Fifteen (9 female) participants (age 25.7 ± 6.8 years) with a minimum 15-degree dominant shoulder internal rotation ROM deficit compared to the opposite shoulder participated. All participants underwent bicipital forearm angle (BFA) measurements and ultrasound imaging to measure acromiohumeral and posterior glenohumeral distances in 3 positions: Resting, 90 degrees of shoulder flexion, and 60 degrees of shoulder abduction with full external rotation. Ultrasound images were used to calculate HHT. Participants' dominant shoulders underwent posterior glide mobilization, followed immediately by repeated ultrasound images and ROM measures. [Results] There was no dominant to non-dominant shoulder, or before and after mobilization HHT differences. No correlations existed between bicipital forearm angles and HHT or ROM gains after mobilization. [Conclusion] Participants with internal rotation ROM loss demonstrated symmetrical HHT. Joint mobilization increased ROM, but HHT was unchanged. No relationships existed between BFA and HHT.
Key words: Humeral retroversion, Joint translation, Joint mobilization

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INTRODUCTION

Shoulder injuries in overhead athletes including baseball, softball, handball, and water polo players occur at rates up to 1.8 per one-thousand hours of participation[1]. Many risk factors have been investigated, but consensus is lacking. Overhead

athletes frequently demonstrate glenohumeral internal rotation deficit, a loss of passive internal rotation range of motion (ROM) in the dominant compared to non-dominant shoulder[2]. This ROM deficit has been associated with shoulder injuries[3–5], but the causes of shoulder injury in these athletes is multifactorial. A recent systematic review concluded that rotator cuff weakness, previous injury, and an excess or deficit in shoulder range of motion (ROM) were important risk factors[6].

Using ultrasound imaging and shear wave elastography, thicker and stiffer posterior glenohumeral joint capsules and stiffer infraspinatus muscles were found in overhead athletes with reduced internal rotation ROM[7–11]. It has been proposed that posterior glenohumeral capsule stiffness leads to shoulder injury by increasing posterior superior humeral head translation (HHT) in abducted and externally rotated positions[5]. Cadaveric studies found increased superior and/or posterior HHT during abduction and external rotation after plicating the posterior inferior glenohumeral joint capsule[12–14]. Other studies found altered anterior translation during flexion after posterior capsule plication[15, 16].

However, decreased internal rotation ROM in overhead athletes' dominant shoulders are due not only to soft tissue changes, but also increased humeral head retroversion[17–19]. The humeral head retroversion angle is measured with radiographs and CT scans, but these measures expose participants to ionizing radiation and may not be readily available to physical therapists and athletic trainers. The bicipital forearm angle is inversely related to the retroversion angle and is used to indirectly assess retroversion. Previous investigators demonstrated the validity and reliability of ultrasound imaging and goniometric measurements to determine the bicipital forearm angle and found smaller angles in the dominant compared to non-dominant shoulders in overhead athletes[17–19].

In vivo studies measured HHT during arm elevation with 3D electromagnetic motion capture devices in participants with limited shoulder ROM and found conflicting results. One study found participants with posterior stiffness demonstrated significantly less posterior HHT during abduction in the scapular plane compared to participants with anterior stiffness[20]. Rosa et al. found that anterior and superior HHT during active shoulder flexion was not different in participants with and without posterior shoulder tightness estimated with the low flexion test[21]. These studies did not measure the bicipital forearm angle, did not report internal rotation ROM, and did not assess abduction and external rotation combined, which is an important contributor to shoulder injury[5]. Therefore, it is not known if limited shoulder internal rotation or humeral retroversion is related to HHT.

Previous authors found ultrasound imaging a reliable and valid method to measure HHT *in vivo*. By measuring the distance between the humeral head and scapular boney landmarks before and after application of external forces, valid and reliable measurements were obtained for anterior and posterior HHT[22]. Reliable measures for anterior, posterior, and inferior HHT were reported by other studies[23, 24].

Limited shoulder internal rotation ROM in overhead athletes improved after various stretching and manual therapy interventions[25–30]. However, none of these studies assessed HHT or the relationship between bicipital forearm angle and ROM changes. Therefore, it is not known if interventions to improve internal rotation ROM change HHT during arm elevation. Also, how humeral retroversion is related to ROM gains following intervention has not been studied.

We aimed to assess relationships between internal rotation ROM loss, the bicipital forearm angle, and HHT in current and former overhead athletes. We used ultrasound imaging to assess: (1) How HHT varied between dominant and non-dominant shoulders during active shoulder abduction and full external rotation (AbER) and active flexion; (2) Correlations between the dominant shoulder bicipital forearm angle and HHT during active AbER and active flexion; 3) How posterior glide mobilizations to the dominant shoulder affected internal rotation ROM and HHT during active (AbER) and active flexion; 4) Correlations between the bicipital forearm angle and changes resulting from posterior glide mobilizations in internal rotation ROM and HHT during active AbER and active flexion. We hypothesized that HHT would differ significantly between the dominant and non-dominant shoulders, and in the dominant shoulder before and after posterior glide mobilizations. Finally, we hypothesized that internal rotation ROM gains and HHT changes after joint mobilization would demonstrate weak correlations with the bicipital forearm angle.

PARTICIPANTS AND METHODS

The Texas Tech University Health Science Center's Institutional Review Board approved the study (IRB-L# L20-038). Inclusion criteria were: 1) adults under age 55 years; 2) at least 15-degrees less dominant shoulder passive internal rotation ROM compared to the non-dominant shoulder[4]; 3) upper arm circumference measured at the axilla less than forty cm to assure quality ultrasound images. Exclusion criteria were: (1) previous shoulder surgery or fracture (2) shoulder pain that prevented measurements or intervention completion; (3) less than 90 degrees passive abduction ROM in either shoulder.

A convenience sample of 15 participants with a mean age of 25.7 (± 6.8) years met the inclusion criteria, provided informed consent, and completed the study. Nine participants were female and 6 were male. All participants participated in overhead sports for at least 6 months in the previous 10 years. Their sports included volleyball (5); baseball (4); softball (2), tennis (2), and multiple sports (2).

Five participants had greater internal rotation passive ROM (range: 1–3 degrees) in their dominant shoulder on the day of data collection compared to the day of recruitment. This resulted in 12−14 degrees less internal rotation passive ROM on the dominant compared to non-dominant shoulder. These participants were not excluded from the study because previous research in overhead athletes demonstrated variable shoulder internal rotation ROM depending on pre-measurement activ-

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ity[31]). In our study, pre-measurement activity was not controlled on the day of recruitment, but participants were not allowed to perform any upper extremity exercise on the day of data collection.

Three investigators completed measurements and ultrasound images with all participants in the same order. A digital inclinometer (Baseline Inclinometer, Fabrication Enterprises Inc., Irvington, NY, USA) measured bilateral shoulder ROM and bicipital forearm angles with participants lying supine. Passive shoulder internal and external ROM were measured in the right followed by the left shoulder. Investigators were blinded to the goniometer's digital reading. Next, the bicipital forearm angle was measured with participants lying supine as described by previous authors[17, 19]. A commercially available Supersonic diagnostic US Explorer (Axe-En-Provence, France, Version 10) using 2–10 linear transducer visualized the bicipital tuberosities during bicipital forearm angle measurement.

Next, participants sat without back support in neutral spine posture with feet on the floor. Investigator 4 (JK) a physiotherapist with 7 years of clinical experience and 2 years of ultrasound imaging experience captured acromiohumeral and posterior glenohumeral ultrasound images using Supersonic diagnostic US Explorer (Version 10) using a 15-4 linear transducer in three positions: (1) resting, the upper arm at the participant's side and the ulnar border of the hand resting on the ipsilateral thigh (Fig. 1a); (2) 90 degrees active shoulder flexion with full elbow extension and thumb pointing up (Fig. 1b); (3) 60 degrees active shoulder abduction in the frontal plane with 90 degrees elbow flexion, full external rotation, and forearm supination with the thumb pointing backwards (Fig. 1c). Investigator one (JH) measured joint angles using a goniometer and monitored participants' posture and positions.

To capture acromiohumeral distance images the ultrasound transducer was placed transversely on the superior acromion halfway between the posterior-lateral and anterior-lateral corners. The location was marked on the skin with a permanent marker for reliability. The acromial-humeral distance was the vertical distance on the image between the acromion (medial of any hook or downslope) and a line projected from the humeral head[23]. (Fig. 2a.) To capture posterior glenohumeral images, the transducer was placed parallel to the infraspinatus and flared slightly superiorly or inferiorly to obtain a hyperechoic humeral head and posterior glenoid edge image. The posterior glenohumeral distance was the vertical distance on the image between the posterior most humeral head and a line projected from the glenoid rim[31] (Fig. 2b).

Superior HHT was calculated by subtracting the acromiohumeral distance in the flexed (or abducted and externally rotated position) from the distance at rest. Anterior HHT was calculated by subtracting the posterior glenohumeral distance in the flexed position from that at rest[23, 24]. Investigator one measured acromiohumeral and posterior glenohumeral distances from randomized images using Image J software version 1.52 (Fiji) with a laptop computer several days after data collection. Participant identity, shoulder position, and day of measurement were concealed so the investigator was blinded.
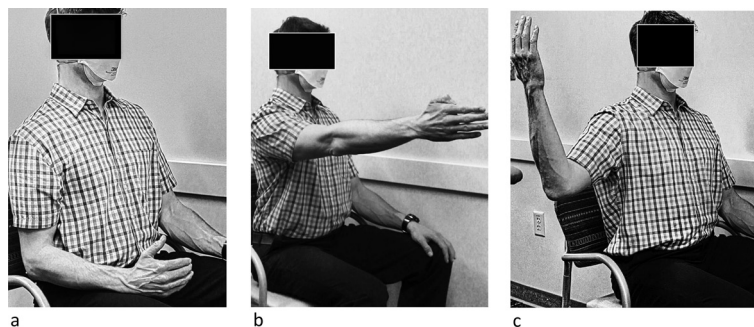

Fig. 1. Resting, flexed, abducted positions.
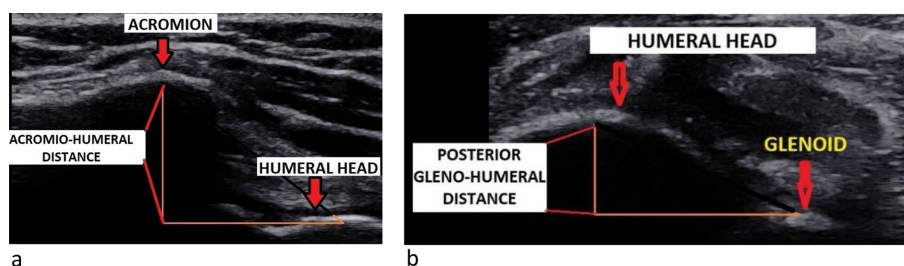a. Resting position, b. Flexed position, c. Abducted position.

Fig. 2. Acromiohumeral and posterior glenohumeral distances.
a. Acromiohumeral distance, b. Posterior glenohumeral distance.

Immediately after ultrasound imaging, investigator five (BD) performed six posterior glide mobilizations to all participants' dominant shoulders using 40N of force (measured with a hand-held dynamometer) with a 30 second rest between mobilizations[30]. Mobilizations were performed supine with a folded towel posterior to the scapular spine and acromion for stabilization. The shoulder was abducted 90 degrees and internally rotated to end range. Mobilizations were directed in a posterior and lateral direction to respect the glenoid fossa's orientation (Fig. 3)

Immediately after mobilization, internal and external rotation ROM was measured bilaterally and recorded on new forms to maintain blinding. Finally, dominant shoulder acromiohumeral and posterior glenohumeral ultrasound images were repeated as before in the resting, flexed, and abducted and externally rotated positions.

Twelve participants volunteered for two assessments at least one day apart to conduct intra-rater reliability for acromiohumeral distance, posterior glenohumeral distance, and bicipital forearm angle measurements. Five of these twelve participants met the inclusion criteria and were enrolled in the study. Statistical analysis was performed with Excel (Microsoft, 2016) and SPSS version 23 for Windows (Armonk, NY, USA: IBM Corp).

All ultrasound images, ROM, HHT, and bicipital forearm angle measurements were repeated three times and the mean of the three measurements was used for data analysis. The Shapiro–Wilk's test assessed data normality and Levene's test assessed homogeneity of variance. Paired t-tests assessed the differences between dominant and non-dominant shoulder internal rotation ROM, bicipital forearm angle, and anterior HHT during shoulder flexion. Paired t-tests assessed internal rotation ROM, and anterior HHT during shoulder flexion before and after mobilizations to the dominant shoulder. A two (side within participants: dominant vs. non-dominant shoulder) × two (motion within participants: flexion vs. AbER) repeated measures ANOVA compared superior HHT. A second two (time within participants: pre- vs. post-mobilization) × two (motion within participants: flexion vs. AbER) repeated measures ANOVA assessed HHT before and after joint mobilization to the dominant shoulder. Intra-class correlation coefficients (3,3) assessed intra-rater reliability of the measurements. Pearson product correlation coefficients determined linear relationships between the bicipital forearm angle, HHT, and internal rotation ROM gains. Significance was set at alpha ≤ 0.05; Bonferroni adjustments were applied to repeated contrasts.

RESULTS

Because of poor ultrasound image quality, acromiohumeral distance in abduction and external rotation data was excluded for two participants. Anterior HHT during flexion in the non-dominant shoulder for one participant was missing. As a result, inferential statistics involving superior HHT include thirteen participants and for anterior HHT 14 participants.

Intra-rater reliability was excellent for acromiohumeral distance ($ICC_{3,3}$: 0.89; CI_{95}: 0.81, 0.93), and posterior glenohumeral distance ($ICC_{3,3}$: 0.85; CI_{95}: 0.71, 0.92) and bicipital forearm angle ($ICC_{3,3}$: 0.92; CI_{95}: 0.73, 0.95) measurements. The assumptions of normality were met for internal rotation range of motion, humeral head translation measurements, and bicipital forearm angle ($p > 0.05$). Likewise, data variances were not significantly different ($p > 0.05$).

Dominant shoulder internal rotation ROM was significantly less than the non-dominant side (mean 18.1 ± 4.4 degrees; CI_{95}: 15.6, 20.6; $p < 0.01$; effect size=1.88) (Table 1). The dominant shoulder bicipital forearm angle was significantly less than the non-dominant side (mean 14.4 ± 6.9 degrees; $p < 0.001$; effect size=1.34) (Table 1). However, significant differences between sides did not exist for anterior HHT during flexion (mean difference 0.3 mm; $p=0.677$; effect size=0.15) (Table 2). Likewise, mean differences in superior HHT during flexion (0.6 mm) and AbER (0.5 mm) were not significant between dominant and non-dominant shoulders, ($F (1,12)=0.501$; $p=0.493$) (Table 3). No significant correlations between bicipital forearm angles and superior or anterior HHT during flexion or AbER in the dominant shoulder existed ($r=0.1$–0.15; $p > 0.59$).

Dominant shoulder internal rotation ROM increased significantly from 41.8 (± 9.8; CI_{95}: 36.4, 47.2) to 52.2 (± 12.2; CI_{95}: 45.4, 58.9) degrees after mobilizations ($p < 0.001$; effect size=0.9) (Table 1). No significant differences in superior HHT were found after mobilizations during flexion and AbER ($F (1,12)=0.058$, $p=0.814$). Similarly, no significant difference existed in anterior HHT during flexion after joint mobilization ($p=0.796$) mm (Table 3).

Fig. 3. Posterior glide mobilization.

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Table 1. Shoulder passive range of motion and bicipital forearm angle

	DOM IR		NON-DOM IR	Bicipital forearm angle	
	PRE	POST	PRE	DOM	NON-DOM
			DEGREES		
Mean ± SD	41.8 ± 9.8*#	52.2 ± 12.2#	59.9 ± 8.9*	10.3 ± 9.6**	24.7 ± 11.3**
Lower 95% CI	36.4	45.4	52.6	4.4	18.5
Upper 95% CI	47.2	58.9	67.8	15.9	31.8
Minimum	21.3	26.2	42.3	−9.4	1.8
Maximum	57.3	67.4	74.5	28.5	42.3
Number	14	14	14	14	14

SD: standard deviation; CI: confidence interval; IR: internal rotation; ER: external rotation; Pre: pre-mobilization; Post: post-mobilization; DOM: dominant shoulder; NON-DOM: non-dominant shoulder; *p<0.01; **p<0.001; #p<0.001.

Table 2. Humeral head translation during shoulder elevation: bilateral shoulders (mm)

	Dominant shoulder			Non-dominant shoulder		
	Superior		Anterior		Superior	Anterior
	Flexion	Abduction and external rotation	Flexion	Flexion	Abduction and external rotation	Flexion
Mean ± SD	3.4 ± 2.2	0.1 ± 1.9	−1.5 ± 2.0	4.0 ± 2.1	0.6 ± 2.5	−1.2 ± 1.8
Lower 95% CI	2.2	−1.1	−2.6	2.8	−0.9	−2.2
Upper 95% CI	4.6	1.2	−0.3	5.2	2.2	−0.2
Number	13	13	14	13	13	14

SD: standard deviation; CI: confidence interval.

Table 3. Humeral head translation dominant shoulder pre-post mobilization (mm)

	Superior translation				Anterior translation	
	Flexion		Abduction and external rotation		Flexion	
	Pre	Post	Pre	Post	Pre	Post
Mean ± SD	3.4 ± 2.2	3.2 ± 2.9	0.1 ± 1.9	0.3 ± 2.3	−1.5 ± 2.0	−1.3 ± 2.9
Lower 95% CI	2.2	1.6	−1.1	−1.1	−2.6	−3.0
Upper 95% CI	4.6	4.8	1.2	1.1	−0.3	0.4
Number	13	13	13	13	14	14

SD: standard deviation; CI: confidence interval; Pre: pre-mobilization; Post: post-mobilization.

A weak non-significant correlation was found between dominant shoulder bicipital forearm angle and changes in superior HHT during AbER after mobilizations (r=0.25; p=0.42). There were no significant correlations between the dominant shoulder bicipital forearm angle and gains in internal rotation ROM (r<−0.06; p=0.87), or changes in superior or anterior HHT during shoulder flexion after posterior glide mobilizations (r<0.1; p>0.78)

DISCUSSION

To our knowledge, this is the first study that assessed HHT during both active shoulder flexion and active AbER in current and former overhead athletes with internal rotation ROM loss. Contrary to our hypothesis, no significant differences in HHT during flexion to 90 degrees or (AbER) existed between the dominant and non-dominant shoulders. Our results contradict previous cadaveric studies[12–16] but are consistent with Rosa et al. who found no difference in HHT during flexion in participants with and without posterior capsular tightness[21]. Additionally, a cadaveric study that simulated rotator cuff muscle activity found no change in HHT after posterior capsular plication[32]. In our study and these previous studies muscle activity may control HHT to a greater extent than capsular stiffness

Internal rotation ROM loss results from soft tissue adaptations and increased humeral head retroversion. To our knowledge no previous study assessed relationships between retroversion and HHT during arm elevation in those with internal rotation loss. We found no correlation between dominant shoulder bicipital forearm angles and HHT during flexion or AbER

in dominant shoulders (r=0.1–0.15; p>0.59) refuting our hypothesis. Despite significantly different bicipital forearm angles between dominant and non-dominant shoulders (mean difference 14.4 ± 6.9 degrees, p<0.001; effect size=1.4), contrary to our hypothesis there were no significant differences in HHT. These findings further suggest the neuromuscular system controlled HHT to a greater extent than soft tissue length, stiffness, or retroversion angles in our participants.

To our knowledge, this study is the first to assess HHT before and after an intervention to improve internal rotation ROM in participants with internal rotation ROM loss. Posterior glide mobilizations improved internal rotation ROM by 10.3 (± 6.7) degrees (p<0.001; effect size 0.9), but HHT during arm elevation did not change significantly contrary to our hypothesis. Acute gains in ROM after treatment result from decreased soft tissue stiffness or decreased muscle sensitivity to stretching. Bailey et al. found instrument assisted soft tissue mobilization and stretching in participants with internal rotation ROM loss decreased infraspinatus stiffness and this was moderately associated with increased internal rotation ROM (r=0.35)[33, 34]. Presumed soft tissue changes from posterior glide mobilizations in our participants did not alter HHT.

There were no significant correlations between the bicipital forearm angle and internal rotation ROM gains or HHT changes during shoulder flexion or shoulder AbER after posterior glide mobilizations (r=0.01 −0.24; p>0.41) refuting our hypothesis. Participants with internal rotation ROM loss may respond similarly to soft tissue interventions despite their degree of humeral head retroversion.

Six end range forty-second sustained posterior glide mobilizations with 40N force increased internal rotation ROM significantly. Witt and Talbott used forces up to 140N for grade 3 glenohumeral inferior glide mobilizations with shoulders positioned in a resting position[23]. Our results were consistent with a cadaveric study that used 40N forces during simulated oscillatory mobilization to the glenohumeral joint and concluded this force is adequate to decrease joint stiffness[37]. Forty newton mobilization forces performed at the end of available ROM may be adequate to improve motion in participants with internal rotation ROM loss.

There are several limitations to our study. First, our participants had small internal rotation ROM loss (mean 18.1 ± 4.4 degrees; range 12.9−25.2) compared to a previous study of current overhead athletes (mean side to 23.8 degrees)[28]. However, a recent meta-analysis found a mean internal rotation ROM loss of 13.8 (± 5.6) degrees in a group of injured overhead athletes[4]. Non-significant HHT differences during flexion and abduction elevation existed between shoulders before mobilizations (0.3–0.5 mm), and in the dominant shoulder after mobilizations (0.2 mm); our small sample size may have resulted in type 2 errors. Future research should include more participants.

Although 5 (33%) participants had experienced dominant shoulder pain during 3 months prior to testing, none sought treatment or reported disability; so, ours was a healthy cohort. Previous *in vivo* research demonstrated altered HHT in participants with various shoulder pathologies[21, 35, 36]. Future studies should assess HHT in participants with internal rotation ROM loss and shoulder pathology.

Our study was not designed to assess differences between male and female participants. Nor was it designed to assess differences between the athletes' preferred sport. Future research should assess whether differences in HHT during shoulder elevation, or the response to posterior glide mobilizations are affected by participants' sex or sport.

Our humeral head translation values represent combined rotational and translatory motion. By controlling the amount of angular motion during arm elevation and performing within participants' comparisons we believe our HHT measures provide meaningful comparisons. Our participants did not elevate their shoulders to end range positions because we could not obtain ultrasound images in those positions. We measured the immediate effects of posterior glide mobilizations; results may differ with longer term follow up including self-stretching. Finally, our HHT values were calculated using static ultrasound images; results may differ during active motions. Also, we did not assess scapular mobility on the thorax, which would have direct consequences on HHT as we measured it. We recommend further research should assess HHT through full arcs of active motion in participants with internal rotation ROM loss and shoulder injury.

Participants with internal rotation ROM loss demonstrated no difference in HHT between dominant and non-dominant shoulders during active shoulder flexion or AbER. Posterior glenohumeral joint mobilization significantly increased internal rotation ROM in the dominant shoulders but did not alter HHT. The bicipital forearm angle did not correlate with humeral head translation before or after joint mobilizations in the dominant shoulder.

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