



# Review of Shoulder Range of Motion in the Throwing Athlete: Distinguishing Normal Adaptations from Pathologic Deficits

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## Abstract

**Purpose of Review** Overhand (OH) throwers demonstrate a unique motion profile of the shoulder joint complex. This manuscript reviews normal adaptations in the OH thrower and contrast findings with pathologic motion deficits.

**Recent Findings** Multiple adaptations in range of motion have been associated with increased risk for arm injury. The use of a more conservative cutoff value for glenohumeral internal rotation deficit and horizontal adduction in younger throwers may help reduce injury risk. Deficits in glenohumeral internal rotation, total range of motion, shoulder flexion, and external rotation insufficiency have all been proposed as means to identify OH throwers at risk for arm injury, but conflicting evidence exists.

**Summary** Understanding normal adaptation due to repetitive stress of throwing is essential to effective management of these athletes. Adaptive change in bone and soft tissues is normal and contributes to the unique motion profiles expected in throwers. The causative link between normal adaptation and shoulder and elbow injury remains uncertain.

**Keywords** Range of motion · Shoulder · Overhead athlete · Normal adaptation · Motion deficit · Injury risk

## Introduction

Injuries to the dominant arm are extremely common in the overhand (OH) throwing motion of baseball. Shoulder and elbow injuries represent 50–67% of injuries reported in professional pitchers [1–3] and are common in younger players as well [4, 5]. Thus, reduction of shoulder and elbow injuries in these populations is paramount. OH throwing is the fastest recorded human movement with peak rotational velocities reaching nearly 7000 degrees per second (°/sec) [6]. Extreme ranges of motion and forces at the shoulder and elbow have been observed in the OH throwing athlete [6, 7].

Numerous studies have examined glenohumeral (GH) joint motion in the OH thrower [8–12] (Figs. 1, 2, 3, and 4). Sub-optimal shoulder range of motion (ROM) has been identified as a risk factor for both shoulder and elbow injury [13–18, 19, 20]. The prevailing thought is that the OH athlete's shoulder adapts to the repetitive stress of throwing, resulting in a unique motion profile. Thus, what is “normal” for a non-thrower may be pathologic in the throwing athlete. The purpose of this review is to describe normal adaptations that occur in the throwing shoulder and the effects of these adaptations on ROM. A second purpose is to describe pathologic deficits in the thrower's shoulder motion in order to provide a framework for clinical evaluation and treatment, and to potentially reduce the risk of injury.

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## “Normal” adaptation in the overhand thrower

OH throwers are often observed to achieve supraphysiologic shoulder motion. Shoulder mobility is achieved through a combination of spine, scapulothoracic joint, sternoclavicular joint, acromioclavicular joint, and GH joint motion [21–24]. Each articulation contributes to linking of the “kinetic chain” to optimize acceleration of the distal segments prior to ball release [25–28]. A comprehensive review of the unique



**Fig. 1** Passive glenohumeral internal rotation range of motion measurement, scapula stabilized

contributions of each of these joints is beyond the scope of this paper. We will focus on the bony, soft tissue, and motion adaptations of the GH joint.

Adaptations of the shoulder over time due to the stress of throwing have been well-documented [7, 13, 29–36, 37, 38–40]. The traditional motion profile of the OH thrower demonstrates increased GH external rotation and a decrease in GH internal rotation of the dominant (throwing) arm [29]. Both bony and soft-tissue adaptations contribute to this change in motion [35, 37, 41–47, 48]. A brief review of bone and tissue mechanics is helpful for one to understand the physiologic basis for the observed changes.

## Bone

Bone is a dynamic tissue that responds to a variety of stimuli and is regulated by a number of homeostatic controls [49]. Mechanical stimulation provides a unique driver of bone



**Fig. 2** Passive glenohumeral external rotation range of motion measurement



**Fig. 3** Passive glenohumeral horizontal adduction range of motion measurement

development and alteration. Wolff's law states that bony adaptation will occur in response to repeated load [50]. Wolff also proposed that this remodeling—in the setting of a particular mechanical stimulus—would result in an improvement in the bone's ability to withstand that particular stimulus. This process of bony response to subjected loads contributes to alterations in humeral retrotorsion (HRT) and glenoid retroversion (GRV) observed in throwers. The humeral head displays a position of retrotorsion at birth. During maturation, the humeral head rotates into relative antetorsion [51]. The majority of this process is completed by 8 years of age but continues slowly until 16 years of age [37]. The forces experienced during the OH throw can strain the epiphysis which may produce epiphyseal changes that limit natural antetorsion during maturation [37, 42, 52, 53]. This decrease in antetorsion, over time, results in relative retrotorsion when compared to the non-dominant arm. The association between HRT and GRV is poorly understood. Recently, the relationship was observed to be a 2:1 “throwers’ ratio” in the dominant arm in a group of professional baseball pitchers—suggesting HRT and GRV may be coupled during development [35].



**Fig. 4** Passive glenohumeral flexion range of motion measurement

HRT adaptation was initially thought to occur at a later age due to prolonged, repetitive OH throwing. This theory came under scrutiny as HRT was shown to be consistent among high school baseball players over the course of 1 year [54]. In addition, a recent study on a group of 8–12 year-old youth baseball players suggests that HRT may occur even earlier than originally proposed [37•]. Diagnostic ultrasound was used to determine HRT and posterior capsular thickness using reliable and valid biomechanical methods [46, 55, 56]. HRT variance between dominant and non-dominant shoulders was found to be similar in the 8–12-year-old group to HRT variance reported in previous studies of high school, collegiate, and professional athletes [30, 46, 54, 57]. This result is in contrast to a previously reported study that observed a much smaller HRT variance in elite elementary and middle school-aged baseball athletes with less playing experience [37•, 53]. This new finding has important implications when evaluating the youth OH thrower. At this time, research suggests that we may expect similar HRT increases in the dominant arm of youth athletes as early as 8 years of age when compared to older and more experienced OH throwing athletes (high school, collegiate, and professional). Increased HRT and GRV contribute, in part, to the observed increase in throwing arm GH external rotation and decreased GH internal rotation in a 90° abducted position. Thus, increased GH external rotation and decreased GH internal rotation should be considered normal when comparing the dominant and non-dominant arms of nearly all OH throwers.

### Soft tissue

In addition to HRT and GRV, there has been investigation into scapular position, rotator cuff, and posterior capsule adaptations. The scapula functions as part of the scapulothoracic “pseudojoint” to stabilize the upper extremity to the axial skeleton. Primary rotational motions include upward/downward rotation, internal/external rotation, and anterior/posterior tilting. Composite translational motions of elevation/depression and protraction/retraction around the curved trunk are also used to describe scapular movement. The scapula retracts, posteriorly tilts, and upwardly rotates to achieve the late-cocking position of an OH throw. It then protracts with arm acceleration and follow-through [6, 58]. Patients with subacromial pain have been reported to have less scapular upward rotation and posterior tilt during arm raising tasks [59]. Further, subjects with a shortened resting pectoralis minor have exhibited altered scapular kinematics similar to those seen in patients with shoulder impingement [60]. There have been differences in kinematics described between the dominant and non-dominant arms in pitchers when compared to the general population [43]. Differences also exist between pitchers and position players [61, 62]. However, changes in scapular position, orientation, and dynamic

motion—also known as scapular dyskinesis—have been observed in both healthy and injured throwers [43–45, 63]. In a recent prospective trial of 246 high school baseball players, no significant difference existed in injury rates between those with “normal” scapular function and those with scapular “dysfunction” [64]. Shitara et al. also did not find scapular dyskinesis as a statistically significant risk factor for shoulder or elbow injury in a prospective study of high school baseball pitchers [65•]. Alterations in dominant arm scapular position and orientation—including anterior tilt and internal rotation at rest; and increased upward rotation, internal rotation, and retraction during dynamic arm elevation—may represent chronic adaptations due to throwing. These alterations have not been definitively shown to contribute to or result from injury. Therefore, clinical evaluations should consider minor scapular position and orientation changes to be normal in the OH thrower and proceed with caution when attempting to implicate minor scapular alterations as a primary cause of throwing shoulder pathology.

Posterior capsular change and increased rotator cuff stiffness are also recognized as changes occurring about the GH joint in response to throwing. Muscle and other soft tissues are adaptable based on a variety of mechanical stimuli [66]. The posterior capsule has been shown to thicken over time in response to OH throwing. This is likely due to repetitive injury resulting in reactive scar tissue formation [16, 67•, 68]. Posterior capsule thickness increase has now been demonstrated in the dominant arm of both older and younger throwers via diagnostic ultrasound [37•, 46]. Further, increased posterior capsule thickness has been correlated to GH internal rotation [47]. Confirmation of increased posterior capsular thickness and loss of GH internal rotation in younger and older throwers suggests that the capsule does contribute to this motion deficit and is a normal response to repetitive OH throwing. Thus, posterior capsular thickening may be both normal and pathologic given the relationship of GH internal rotation deficit (GIRD) to upper extremity injury in OH throwers [15, 16, 69, 70•].

OH throwing can further create a response at the cellular level resulting in muscle shortening in addition to capsular thickening [71]. The particular response of muscle to chronic strain is termed thixotropy—increased muscle stiffness based on exposure history of the muscle [41, 72–74]. Shoulder ROM changes have been demonstrated to occur after an acute bout of throwing, over the course of a season, and over a 1-year period [10, 41, 54, 75–77]. This suggests that alterations in the ROM profile of the OH thrower are multifactorial and not related to bony or posterior capsule changes in isolation. Further, these changes are acutely amenable to intervention including stretching and soft tissue mobilization [48•74, 78–81]. A recent study demonstrated that an acute intervention of instrument-assisted soft tissue mobilization plus self-stretching demonstrated improved GH internal rotation, total

arc of motion, horizontal adduction, and decreased posterior rotator cuff stiffness compared to self-stretching alone [48•]. The authors agree with Bailey et al. that changes in rotator cuff stiffness contribute, in part, to the normal ROM profile observed in OH throwers.

### “Normal” motion

An understanding of typical variations in observed motion is important in evaluation of the OH thrower. Some of the most robust studies to date include passive ROM assessment of asymptomatic youth (age 8–16) baseball players, youth (age 8–18) pitchers, high school (age 14–18) pitchers, and professional baseball pitchers [11, 12, 20•, 70•, 82]. These studies utilized reliable ROM measurement procedures (Figs. 1, 2, 3, and 4) [83]. Whereas Meister, Shanley, and Hurd et al. specified asymptomatic participants, this was not explicitly stated by Wilk or Camp et al. GH joint motion group means are summarized in Table 1 [11, 12, 20•, 70•, 82]. In summary, the total ROM (TROM) is expected to fall between 160 and 210° in the OH thrower. Side-to-side differences of 10–15° are considered normal when examining GH external rotation and GH internal rotation individually; however, the physiologic TROM has consistently been found within 5° on bilateral comparison [11, 12, 20•, 41, 70•, 82, 84]. Horizontal adduction may fall between 3 and 50° and shoulder flexion ranges from 165° to greater than 180°. These measures provide general ranges to guide clinical examination. Although these ranges are large and describe a large age array, there is great overlap between the youth and adult thrower. Based on reported evidence regarding early adaptation to throwing, there is perhaps more similarity than previously thought. This allows for a more consistent general expectation of the thrower’s ROM among all age groups.

### Pathologic deficit

Pathologic change should be discussed with the thrower’s normal motion profile in mind. Research has focused on a variety of specific motion characteristics including glenohumeral internal rotation deficit (GIRD), total rotational motion (TRM)/TROM, GH flexion, GH horizontal adduction, external rotation gain/surplus, and external rotation insufficiency summarized in Table 2 [15–18, 19•, 20•, 65•, 69, 70•, 85, 86••]. Deficits and gains refer to differences between the dominant and non-dominant shoulders. For clarification purposes, GIRD will be defined as a > 20° deficit on bilateral comparison [16]. TRM and TROM can be used synonymously as they both refer to the sum of GH external rotation and GH internal rotation measured in 90° of abduction. External rotation insufficiency is defined as having less than a 5° increase in GH external rotation of the dominant compared to non-dominant shoulder. For the purpose of this review, these

motions are measured passively in supine (Figs. 1, 2, 3, and 4) unless otherwise stated. A recent meta-analysis and systematic review examined whether adaptations in GH ROM lead to upper extremity injuries (shoulder or elbow) in OH athletes [86••]. Keller et al. found that the pooled results of the review did not reach statistical significance for any shoulder motion measurement and its correlation to shoulder or elbow injury. They did find a trend toward injury in OH athletes with GIRD, TRM loss, and GH external rotation gain [86••]. However, this study included all OH athletes. These motion characteristics will be reviewed with respect to OH throwers with particular focus on studies performed in the past 5–10 years.

GIRD was originally proposed as pathologic in the throwing shoulder by Burkhart et al. [32, 41, 67•, 88]. Over time, its definition has changed slightly and is now commonly accepted as a  $\geq 20^\circ$  loss of GH internal rotation in the throwing shoulder compared to the non-dominant arm [16]. GIRD has been shown to relate to changes in the posterior capsule and HRT [47, 89]. Several studies have demonstrated an increased risk of shoulder/arm injury in OH throwers with GIRD [14–16, 69, 70•]. However, there have also been multiple studies showing no correlation between GIRD and shoulder [19•] or elbow [17, 18, 20•, 86••] injury. Conversely, one study found that high school pitchers without GIRD were at increased risk for injury compared to peers with GIRD as defined as > 20° loss of GH internal rotation [85]. The limits to GH external rotation and GH internal rotation motion measurements are primarily due to soft tissue restriction and not bony approximation [90•]. GIRD as a measure in isolation primarily evaluates posterior capsule restriction as a cause for decreased GH internal rotation ROM [47, 90•]. Further, in a study of 193 major and minor league pitchers, a statistically significant association was found between TROM, decreased shoulder strength, and GIRD [91]. The presence of GIRD may potentially be more relevant in risk factor identification for younger throwers [69, 70•] but, given its association with decreased shoulder strength [91], should not be ignored in the older thrower [15].

TROM is proposed as a more complete evaluation of the throwers’ shoulder motion. Bilateral comparison allows for an indirect assessment of HRT based on the gain in external rotation and respective loss of internal rotation which have been shown to correlate with an increase in HRT [31, 34]. Wilk et al. demonstrated that when TROM deficits are  $\geq 5^\circ$ , there is an associated 2.5× and 2.6× increased risk for shoulder and elbow injury, respectively [16, 18]. Others have shown a relationship between TROM deficits comparing injured and uninjured players [14, 17, 65•, 92]. However, there have also been multiple studies finding no correlation between TROM deficits and upper extremity injury [19•, 20•, 70•]. TROM should be evaluated with the understanding that GH external rotation will be increased and GH internal rotation will be decreased on bilateral comparison. Despite several

**Table 1** Glenohumeral range of motion in baseball

Reference	Population	Measurement	Primary findings
Meister [82]	Youth baseball players	GHER; GHIR; TRM; Flexion	*D GHER: 142.9° ± 13.1 *ND GHER: 136.6° ± 12.7 *D GHIR: 35.9° ± 9.8 *ND GHIR: 41.8° ± 8.6 D TRM: 178.7° ± 16.5 ND TRM: 178.3° ± 16.5 D Elevation: 177.4° ± 5.8 ND Elevation: 177.9° ± 5.1
Shanley [70•]	Youth (8–12) and adolescent (13–18) pitchers	GHER; GHIR; TRM; HA; Side-to-side differences	<i>Dominant shoulder motion:</i> GHER (youth): 135° ± 13 GHER (adolescent): 130° ± 14 GHIR (youth): 43° ± 10 GHIR (adolescent): 47° ± 13 HA (youth): 19° ± 16 HA (adolescent): 17° ± 18 TRM (youth): 179° ± 15 TRM (adolescent): 177° ± 15 <i>Side-to-Side Differences</i> GHIR (youth): 10° ± 10 GHIR (adolescent): 12° ± 12 HA (youth): 11° ± 16 HA (adolescent): 13° ± 16 TRM (youth): 5° ± 14 TRM (adolescent): 2° ± 12
Hurd [11]	High school baseball pitchers	GHER; GHIR; TRM	*D GHER: 130° ± 11 *ND GHER: 120° ± 10 *D GHIR: 60° ± 11 *ND GHIR: 75° ± 11 *D TRM: 190° ± 15 *ND TRM: 195° ± 15
Wilk [12]	Professional pitchers	GHER; GHIR; TRM; HA	*D GHER: 132° ± 11 *ND GHER: 127° ± 11 *D GHIR: 52° ± 12 *ND GHIR: 63° ± 12 *D TRM: 184° *ND TRM: 190° *D HA: 42° ± 8 *ND HA: 44° ± 8
Camp [20•]	Professional pitchers	Dominant to non-dominant: Flexion deficit; ER gain; IR deficit; HA deficit; TROM deficit	Flexion deficit: 4.1° (–10 to 20°) ER gain: 7.8° (–15 to 26°) IR deficit: 13.2° (–15 to 40°) HA deficit: 4.5° (–20 to 25°) TROM deficit: 4.7° (–20 to 31°)

Mean reported in degrees

*GHER* glenohumeral external rotation, *GHIR* glenohumeral internal rotation, *TRM* total rotational motion, *HA* horizontal adduction, *D* dominant arm, *ND* non-dominant arm, *TROM* Total range of motion

\*Statistical significance

studies that showed no increased risk of injury [19•, 20•, 70•], compelling evidence exists that special attention should be paid to TROM deficits [14, 16–18, 65•, 92]. The authors suggest a cut-off value of > 5° on bilateral comparison, as this may place the thrower at increased risk for dominant arm injury.

GH horizontal adduction and flexion measurements are also being performed more regularly to attempt to identify at-risk shoulders in OH throwers. Garrison et al. found no group differences in

horizontal adduction motion between high school and collegiate baseball players diagnosed with an ulnar collateral ligament injury and those without injury [17]. Conversely, Shanley et al. found a 4× greater risk of developing an overuse arm injury in adolescent baseball pitchers with > 15° difference in horizontal adduction [70•], whereas a previous study of high school baseball and softball players was unable to establish a risk criterion for horizontal adduction [69]. Posterior shoulder tightness was

**Table 2** Pathologic change in glenohumeral motion

Reference	Population	Measurement	Primary finding
Scher [15]	57 professional baseball players	GHIR; GHER; GIRD	Non-pitchers with history of shoulder injury had > ER and < IR of the shoulder than non-pitchers with no shoulder injury history GIRD was greater in both pitchers ( $-10.1^\circ \pm 9.0$ ) and non-pitchers ( $13.5^\circ \pm 8.8$ ) with a history of shoulder injury compared to pitchers ( $3.1^\circ \pm 11.2$ ) and non-pitchers ( $4.2^\circ \pm 13.8$ ) with no history of shoulder injury
Wilk [16]	122 professional pitchers (170 pitcher-seasons)	GHER; GHIR; injuries tracked prospectively	Players with GIRD were nearly twice as likely to be injured but without statistical significance ( $p = .17$ ; OR: 1.9) TRM deficit $> 5^\circ$ were $\times 2.5$ more likely to sustain a shoulder injury ( $p = .03$ )
Shanley [69]	246 high school softball and baseball players	GHER; GHIR; HA; injuries tracked prospectively	Significant decrease in HA ( $p = .05$ ) and IR ( $p = .04$ ) for all injured players TRM of the dominant arm in injured baseball players displayed significant decrease ( $8.0^\circ \pm 0.1$ ; $p = .05$ ) compared to the dominant shoulder of uninjured baseball players Players with a decrease of $\geq 25^\circ$ of IR were at 4x greater risk of arm injury compared with those with $< 25^\circ$ decrease in IR, particularly baseball players 1.5-2x increased risk of injury for 10–20° loss in TRM, risk estimates were not statistically significant ( $p > .05$ )
Garrison [17]	60 high school and collegiate baseball players with diagnosed ulnar collateral ligament tear	GHIR; GHER; HA	Players with UCL tear had greater deficits in TRM compared with healthy controls ( $-6.67^\circ \pm 11.82$ vs. $0.93^\circ \pm 9.91$ ) No group differences present for GIRD ( $p = .4761$ ) or HA ( $p = .860$ ) Pitchers with UCL tear had significantly > deficits in TRM ( $-6.96^\circ \pm 11.2$ vs. $1.29^\circ \pm 8.33$ ) and dominant shoulder ER than those without ( $112.04^\circ \pm 14.35$ vs. $121.85^\circ \pm 9.46$ )
Wilk [18]	296 major and minor league pitchers in a single organization over 8 years	GHER; GHIR; Flexion; injuries tracked prospectively	No correlation between GIRD or GHER insufficiency and elbow injury Pitchers with TRM deficit $\geq 5^\circ$ in the throwing shoulder had $\times 2.6$ greater risk for elbow injury ( $p = .007$ ) Pitchers with deficit of $\geq 5^\circ$ in flexion of the throwing shoulder had a $\times 2.8$ greater risk for elbow injury ( $p = .008$ )
Tyler [85]	101 high school pitchers over 4 seasons	GHER; GHIR; HA <sup>a</sup> ; injuries tracked prospectively	Pitchers with no GIRD were at increased risk compared to with pitchers with $> 20^\circ$ loss ( $p = .04$ ) Other ROM measures were not related to injury risk
Shanley [70]	115 youth (8–12) and adolescent (13–18) baseball pitchers	GHER; GHIR; injuries analyzed retrospectively	HA difference $> 15^\circ$ resulted in $\times 4$ greater risk of arm injury GIRD $> 13^\circ$ resulted in $\times 6$ greater risk of arm injury No statistically significant difference in TRM between injured and non-injured pitchers
Wilk [19]	296 major and minor league pitchers in a single organization over 8 years	GHER; GHIR; flexion; injuries tracked prospectively	GIRD, TRM deficit, and flexion deficit were not significantly related to shoulder injury or surgery Pitchers with GHER insufficiency ( $< 5^\circ$ greater ER in the throwing shoulder) were $\times 2.2$ more likely to be placed on the disabled list for a shoulder injury ( $p = .014$ ) and were 4x more likely to require shoulder surgery ( $p = .009$ )
Camp [20]	All pitchers invited to Major League Baseball spring training for a single organization	GHER; GHIR; flexion, HA; injuries analyzed retrospectively	Most significant categorical risk factor associated with increased elbow injury rates was the presence of shoulder flexion deficit $> 5^\circ$ (OR 2.83; $p = .042$ ) The risk of elbow injury increased by 7% for each degree of increased shoulder ER deficit (OR 1.07; $p = .030$ ) and for 9% for each degree of decreased shoulder flexion (OR 1.09; $p = .017$ )
Shitara [65]	105 high school baseball pitchers (15–17)	GHER; GHIR; HA; injuries tracked prospectively	No measures significantly correlated with shoulder injuries GHIR ( $35.7^\circ \pm 14.5$ vs. $42.7^\circ \pm 11.9$ ; $p = .02$ ) and TRM ( $139.8^\circ \pm 13.6$ vs. $146.4^\circ \pm 13.3$ ; $p = .04$ ) of the dominant shoulder were significantly less than those in the uninjured group Decreased GHIR in the dominant shoulder was significantly associated with injury ( $p = .02$ )
Keller [86]	2195 OH athletes	Shoulder ROM and UEX injury	Pooled results did not reach statistical significance for any shoulder motion measurement and its correlation to shoulder or elbow injury Results favored injury in OH athletes with GIRD, TRM loss, and ER gain

GHER glenohumeral external rotation, GHIR glenohumeral internal rotation, TRM total rotational motion, HA horizontal adduction

<sup>a</sup> HA measured in sidelying as distance from medial epicondyle to treatment table described by Tyler et al. [87]

significantly different on bilateral comparison in throwing athletes with internal impingement [13], though this was measured in a separate, reliable manner [87, 93] than demonstrated in Fig. 3. Shoulder flexion deficits  $> 5^\circ$  have been shown to lead to  $2.8 \times$  greater risk of dominant arm elbow injury in professional baseball pitchers ( $p = .008$ ; OR 2.83 and  $p = .042$ , respectively) [18, 20]. Flexion has not been shown to correlate to shoulder injury in professional pitchers [19]. It has yet to be documented as a risk factor for young throwers to the authors' knowledge. Based on best available prospective research, horizontal adduction deficits  $> 15^\circ$  may identify increased risk of developing an overuse arm injury in adolescent baseball pitchers [70]. There is no evidence to establish a cut-off value in older throwers at this time. Multiple prospective studies demonstrate that shoulder flexion deficits  $> 5^\circ$  increase the risk for dominant arm elbow injury in professional pitchers and other older throwers and should therefore be included in injury risk assessments [18, 20].

As with TROM, horizontal adduction, and GH flexion, GH external rotation motion changes in isolation present the clinician with a unique predicament. There is conflicting evidence whether GH external rotation insufficiency ( $< 5^\circ$  increased external rotation in the dominant compared to non-dominant shoulder) may increase the risk of shoulder injury [19] and elbow injury [20]. In a group of high school and collegiate baseball pitchers with diagnosed ulnar collateral ligament tears, significant deficits in dominant arm external rotation were observed compared to those without injury [17]. In their recent systematic review and meta-analysis, Keller et al. indicated that external rotation gain favored injury in OH athletes [86]. However, this result was skewed by inclusion of tennis and handball athletes. When evaluating baseball throwers alone, strong evidence suggests that GH external rotation insufficiency increases the risk of dominant arm injury in high-level throwers [19, 20]. Careful attention should be paid to excess external rotation gain as maximum elbow varus torque is produced near the moment of maximum GH external rotation placing significant stress across the medial elbow [7]. It is the opinion of the authors that external rotation gain in the dominant arm should be  $\geq 5^\circ$ . There is no conclusive evidence to suggest a cut-off where ER gain moves from a protective adaptation to a pathologic change.

The authors acknowledge the conflicting nature of evidence with regard to normal adaptation compared to pathologic changes in the OH athlete [86]. For example, increased HRT has recently been suggested as protective of the shoulder and detrimental to the elbow [94]. Because throwing shoulder ROM is affected by both bony and soft tissue adaptation, the authors caution the clinician in prescribing treatment based on one measurement characteristic alone. Further, it has been demonstrated that risk stratification based solely on GIRD, TROM, or HRT alone is not in agreement [90]. The authors recommend a comprehensive evaluation of passive ROM in the OH thrower to include GIRD, TROM, flexion, horizontal

adduction, and external rotation gain/insufficiency, as these have all been linked to upper extremity injury [16, 17, 19, 20, 65, 70, 92]. The use of a more conservative cutoff value for GIRD of  $\geq 13^\circ$  in younger throwers and the more traditional  $\geq 20^\circ$  in older throwers may assist with reduction of arm injury risk. TROM and shoulder flexion deficits  $> 5^\circ$  on bilateral comparison may identify OH throwers at risk for dominant arm injury. The dominant shoulder should have  $\geq 5^\circ$  increased external rotation on bilateral comparison. Lastly, a horizontal adduction difference value of  $> 15^\circ$  in adolescent throwers may identify those at risk.

## Conclusion

Injuries to the dominant arm are common in OH throwers of all ages [1–5]. Physiologic adaptation of bone and soft tissue in the OH thrower occur, and in most instances do not result in significant pathology. Instead these adaptations contribute to the unique motion profile observed in the dominant arm of the OH thrower. Multiple motion changes have been proposed to increase risk of shoulder and/or elbow injury. However, there is conflicting evidence regarding the causative relationship between these changes and injury risk. It is the opinion of the authors that in general, asymmetry in ROM should not be universally considered pathologic. Additional research is needed to more clearly define the normal and pathologic extent of asymmetry in various motions of the OH thrower's shoulder.

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