Glenohumeral Internal Rotation Deficit: Pathogenesis and Response to Acute Throwing

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Abstract: Overhand throwing places high loads and stresses on the joints and tissues of the shoulder and arm. As a result, throwing athletes regularly demonstrate altered shoulder internal and external ranges of motion where internal rotation (IR) is decreased and external rotation is increased in the dominant arm when compared with the nondominant arm. This alteration can exist as a result of alterations to the bones (humeral retroversion), capsule (posterior thickening), or muscle (passive stiffness known as thixotropy). When the amount of IR or total arc of motion difference reaches a certain threshold (typically 20 or more degrees of IR or 8 degrees total arc difference), it is known as glenohumeral internal rotation deficit or total arc of motion deficit. Glenohumeral internal rotation deficit and total arc of motion deficit can cause alterations in biomechanics such as scapular "wind-up" or alteration of glenohumeral joint kinematics, which can in turn lead to clinical findings of impingement and labral pathology. This study will review the causes of motion alteration, effects of altered motion on the throwing motion, provide definitions for the various types of rotation deficits, and how to evaluate and treat rotational deficits.

Key Words: GIRD, shoulder, stiffness, throwing

(*Sports Med Arthrosc Rev* 2012;20:34–38)

PRECISION OF GLENOHUMERAL (G-H) BALL AND SOCKET KINEMATICS

The overhand throwing or serving motion produces large loads and forces on the joint tissues, as a result of the high velocities and large range of motions. A high degree of arthrokinematic precision is required to accomplish this task efficiently for maximal performance and minimal injury risk.

One of the key factors in G-H arthrokinematics is to allow optimal G-H rotation, which includes both internal rotation (IR) and external rotation (ER). The proper balance of IR and ER allows the humeral head to remain centered in the glenoid fossa[1] and maximizes concavity compression.[2] Optimal ER also contributes to maximal hand and ball velocity[3,4] and decreases the valgus loads at the elbow.[4,5]

The amount of developed IR and ER is dynamic and is continually adapting to the high G-H forces and loads. Multiple studies have documented that in the dominant arm, compared with the nondominant arm, the magnitude of glenohumeral internal rotation (GIR) is decreased and the magnitude of glenohumeral external rotation is increased in most throwing athletes.[6–12] In many throwers, although there is a shift in motion, the total arc of motion (TAM, the sum of IR and ER) is the same bilaterally.[12] The increase in ER is thought to be advantageous for achieving the maximal arm position for the cocking phase of throwing and creating maximum ball velocity.[6,13]

However, maladaptations can also occur that alter the arthokinematics and may increase injury risk. Because of repetitive throwing, IR and TAM in the dominant shoulder may be decreased in magnitude compared with the nondominant shoulder. This decreased motion results in altered G-H arthokinematics, which causes a shift in the humeral head instant center of rotation to an anterosuperior position on the glenoid during forward flexion[1] and a posterosuperior position with ER and cocking.[6,14–16] These arthokinematic alterations have theoretical implications for injury to the G-H and elbow joint structures due to increased compression and tension forces, altered joint kinematics, and altered interactive moments.[6,17] Studies have confirmed these theoretical implications showing associations of decreases in IR and TAM with labral injury[6,18,19] and elbow injuries,[20] and these changes have also been shown to be predictive of labral injury, indicating increased injury incidence at the shoulder.[21]

These maladaptive changes have been expressed as deficits of G-H motion compared with the nondominant arm—glenohumeral internal rotation deficit (GIRD) and total arc of motion deficit (TAMD). Since GIRD was first reported,[22] multiple studies have been conducted further describing it. GIRD is known to increase and IR to decrease with years of throwing exposure,[23,24] throughout a competitive season,[25,26] and acutely after a throwing exposure.[27,28] IR and GIRD have also been shown to be different in the amount of change over 1 season between starters and relievers.[26] This demonstrates that IR, TAM, GIRD, and TAMD are dose or exposure dependent. However, it seems that GIRD is one of the most important factors influencing injury risk in the arm.

WHAT ARE GIRD AND TAMD?

IR is a value that represents the amount of IR, and GIRD represents the side-to-side difference in IR that is considered to have significantly adverse side effects on the biomechanics of the G-H joint during activities and may be predictive of injury. Similarly, TAMD represents a side-to-side difference of the total arc of G-H motion that is of concern and may predict injury. TAMD seems to be mainly influenced by the amount of GIRD, as altered IR is the largest single alteration in G-H motion.[28] GIRD and TAMD are seen in both sexes, and although no definitive

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Disclosure: The authors declare no conflict of interest.

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studies have been carried out, the magnitude and frequency of these alterations are anecdotally reported to be similar.[25,29] The addition of GIRD and TAMD in conjunction with repetitively high loads, high forces, extremes of motion, and high velocities can place ultraphysiological strains on the shoulder and arm, creating injury and decreased performance.

WHAT GIRD AND TAMD ARE NOT?

These entities are not injuries themselves, nor are they specifically related to 1 type of injury. They are not predictive of any 1 specific injury, especially in individuals who do not expose their shoulders and arms to repetitive overhead motions of high intensity or long duration.

DEFINITION OF GIRD AND TAMD

The working definition of GIRD has evolved since it was first described as an absolute value of < 25 degrees of IR in the involved shoulder.[30] To take into account individual variations and the effect of throwing exposure, a relative side-to-side IR difference of 25 degrees was adopted.[6] Recent studies have demonstrated that a GIRD of as little as 11 degrees[18] and 18 degrees[21] is associated with shoulder injury. In addition, a prospective study demonstrated that a GIRD of 18 degrees was related to a 1.9 times increased risk of injury.[21] At this time, the current recommendation for defining GIRD is a 20 degree side-to-side difference.

TAM was first described by Wilk et al.[12] It suggested that although there is a shift in the arc of motion on the dominant arm into more ER and less IR that total motion should remain equal. This adaptation has been shown to be caused by humeral retroversion.[7,10] At birth, humans have large amounts of humeral retroversion. However, throughout development, the humerus slowly rotates into anteversion. When adolescents begin throwing, it is suggested that the repetitive torsional forces along the axis of the humerus will alter and slow this process, leaving the humerus in more retroversion compared with the nondominant arm but maintaining the total motion arc.[31] When changes in TAM are present in mature athletes, it is thought to be caused by soft tissue tightness. Current research places the threshold for TAMD at a 5 degree side-to-side difference.[21]

WHAT CAUSES GIRD AND TAMD?

Alterations in all 3 anatomic components—bone, capsule, and muscle—contribute to changes in G-H rotation in throwers. There is most likely a spectrum and a sequence of causation for these tissue adaptations. Although scientific research is continually progressing, further research is required.

There have been several studies detailing the changes in humeral retroversion and the association with throwing exposure in youth athletes.[7,10,22,24,32,33] Research has shown that the average side-to-side difference for humeral retroversion is 10 to 15 degrees. This magnitude by itself is not considered to be pathologic or the cause of GIRD because it is less than the currently determined pathologic threshold, is less than the commonly observed 30 to 50 degrees seen in injured athletes, and will not change in athletes with closed epiphyses. However, it may create an additional amount of tightness that will require only small amounts of soft tissue alteration to create problems. In contrast, some authors consider the bony changes to be a positive adaptation, allowing a more effective position of ER in maximum cocking while maintaining anatomic arthokinematics.[6]

Concepts of soft tissue contributions to the pathophysiology of GIRD usually relate to scar tissue-induced changes in the posterior G-H capsule thought to be due to a chronic process of tensile loading producing microtears and reactive scarring.[1,6,14,21,34–36] This has been based on capsular plication studies,[1,14] arthroscopic evaluation, and clinical results from rehabilitation protocols.[21,34,36] This concept is consistent with studies demonstrating decreased IR rotation over time[24] and the frequent arthroscopic and magnetic resonance imaging appearance of the thickened posterior capsule. Diagnostic ultrasound studies demonstrated that baseball players have an increased thickness of the posterior capsule.[37] This was also shown to correlate with observed amounts of IR. This is further validation that the posterior capsule is involved with clinical GIRD. It also found that baseball players with an increased amount of humeral retroversion have a hypertrophied posterior capsule.[37] This demonstrates that players with large amounts of humeral retroversion are placing increased stress on the posterior capsule during the deceleration and follow through phase of the throw. Humeral retroversion, which may be a positive adaptation for producing greater ball velocity, may also be detrimental to the integrity of the shoulder capsule. The initiation of rehabilitation to minimize posterior capsule hypertrophy may be a necessary intervention to minimize the risk of shoulder injury.

Recent studies have shown that the magnitude of IR, GIRD, TAM, and TAMD can have relatively large changes in response to a single acute throwing exposure of high intensity and duration.[27,28] It has been shown to decrease as much as 15% immediately after a throwing exposure and 24 hours after throwing.[27] This decrease is too rapid to be caused by capsular changes. Therefore, muscle properties may account for these acute changes.

Studies have shown that the response to both acute and chronic muscle strain can directly affect joint range of motion. In an acute response to repetitive tensile strain in eccentric muscle contractions, actual "sarcomere popping," with detachment, has been demonstrated.[38,39] This muscle damage releases intramuscular calcium, which mediates fibril contraction, resulting in muscle shortening. This process peaks between 4 and 18 hours after throwing and is modifiable with gentle stretching at 12 hours. These muscular changes correlate well with the classic muscle soreness and stiffness described after pitching, the acute decrease in GIRD, and the improvement of GIRD after next day stretching.

The chronic muscle response to muscular strain is represented by thixotropy, which is an increased muscle stiffness that is mediated by exposure history of the muscle.[39,40] Repetitive exposure to strain results in increased stiffness within the muscles that can affect joint motion, and is not related to neurological changes. The thixotropic response is modifiable by stretching within certain ranges and velocities.[38] Also, changes in muscle stiffness, and consequent joint motion, have been associated with increased muscle tension in response to repetitive use in the face of eccentric muscle weakness.[41]

In summary, GIRD and TAMD have a multifactorial etiology. It can be postulated that changes in rotation occur in a sequential manner with considerable overlap among several factors. The earliest changes occur in the bone in

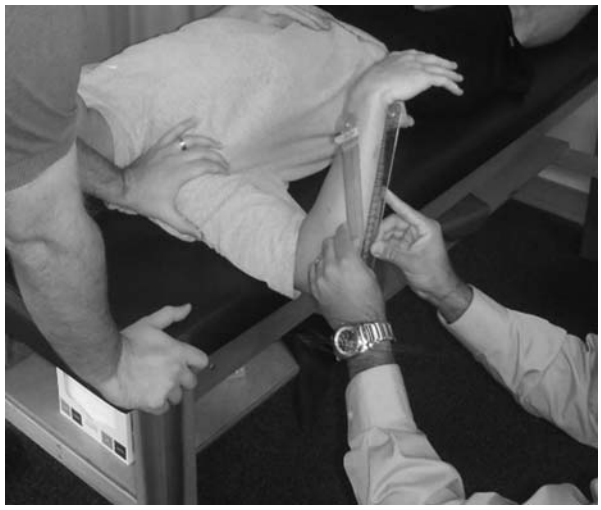

FIGURE 1. Goniometric measurement to determine glenohumeral internal rotation deficit.

response to torsional loading in the adolescent years. As the athletes become larger and exert more force in throwing, higher eccentric and tensile loads are applied to the posterior shoulder muscles, resulting in acute sarcomere changes and chronic thixotropic stiffening and increased muscle tension. These mechanisms are more frequently causative in the 16- to 30-year-old age group. With chronic rotational capsular strain, the capsular causation is seen more predominantly in the older throwers, from 25 to 40 years old.

HOW IS G-H ROTATION ACCURATELY MEASURED?

Measurement accuracy of G-H rotation is required for evaluating these adaptations, because repeated measurements are required, and changes of 5 to 10 degrees may be meaningful. Most clinical studies now rely on the same protocol with the arm at 90 degrees abduction in the plane of the scapula and using a bubble goniometer or a digital inclinometer.[28,42]

Each athlete should be placed in a supine position on a flat level surface. A second examiner should be positioned above the athlete to properly stabilize the scapula during testing by applying a posteriorly directed force to the coracoid to ensure that scapular movement does not occur. The second examiner can read and record the measurements obtained by the first examiner. The humerus is supported on the surface with the elbow placed at 90 degrees and the arm on a bolster in the plane of the scapula.[28] The following landmarks should be identified before placing the goniometer: the fulcrum set at the olecranon process of the elbow, the stationary arm perpendicular to the table as documented by the bubble on the goniometer, and the moving arm in line with the styloid process of the ulna (Fig. 1). The athlete's humerus is then passively moved into IR. Rotation is taken to "tightness," a point where no additional G-H motion occurs unless the scapula moves or the examiner applies extra rotational stress. For ER, similar methods are performed. TAM is then calculated as GIR plus glenohumeral external rotation. The procedures are repeated bilaterally to obtain measurements from both the throwing and the nonthrowing shoulder. This method has been found to be highly reproducible, with a test-retest reliability of 0.92.[28]

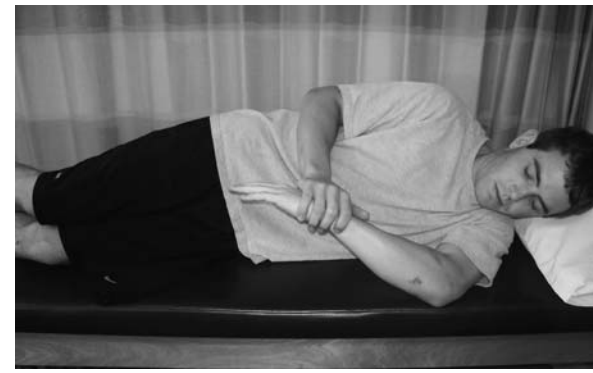

FIGURE 2. Sleeper stretch at 60 degrees abduction.

TREATMENT OF GIRD AND TAMD

Because the pathophysiology of GIRD and TAMD is thought to be primarily soft tissue related, the focus of the treatment should be the soft tissues. This includes interventions relating to muscle stiffness, inflexibility, muscle weakness, and capsular stiffness.

Multiple programs have been developed to correct the rotational alterations, most of which have demonstrated good success.[43–45] The sleeper stretch is the most commonly used exercise to increase IR. It is performed in a side-lying position to stabilize the scapula. To place strain on different parts of the posterior muscles and capsule, the arm is placed at 60, 90, and 120 degrees abduction and a rotational motion is applied (Figs. 2–4). The motion is then taken to and held at the point of tightness. Favorable results have been previously reported.[12,34,45,46] A program using a towel or a racquet to stretch IR behind the back also demonstrated improved motion.[47] A general stretching program has also been shown to increase both IR and ER and decrease posterior shoulder tightness.[43] IR stretching and posterior capsular mobilization decreased GIRD and posterior shoulder tightness.[48] Stretching by emphasizing horizontal adduction on a stabilized scapula resulted in the greatest amount of increase in shoulder IR when compared with other methods.[46] This method seems to specifically target the linearly oriented posterior shoulder muscles versus the circumferential capsule.

FIGURE 3. Sleeper stretch at 90 degrees abduction.

FIGURE 4. Sleeper stretch at 120 degrees abduction.

These results show that many types of stretches, mobilization techniques, and exercises place adequate stress on the tightened, inflexible, and weakened structures to improve the altered rotation. The specific choice of exercises depends on the resources, equipment, and training of the clinician. To be effective, the stretches, mobilization techniques, and exercises need to be performed on a daily basis throughout the playing season, to counteract the tendency to decrease GIR and TAM throughout the season.[25,47]

Data do not exist demonstrating the success of these stretching and mobilization programs in players with GIRD. Anecdotal evidence and clinical experience suggest that 90% or higher do improve. The "stretch nonresponders" are defined as players with GIRD who do not show an improvement after 4 to 6 weeks of appropriate therapy.[34] Nonstretch responders who have continued symptoms and are thought to have G-H joint internal derangement can be candidates for a surgical posterior capsular release as part of the comprehensive treatment of the shoulder pathology. In the relatively few athletes who meet these criteria, the posterior capsule is found to be thickened and scarred in the posterior inferior quadrant, right off the glenoid rim. Release of the thickened tissue using electrocautory and basket forceps will improve IR. This procedure is not indicated as an isolated procedure but should be carried out as part of the repair of the superior labral injury or treatment of the internal impingement injury (see the chapter on internal impingement).

CONCLUSIONS

GIRD and TAMD represent significant alterations in the normal arc of G-H rotation that disrupts optimal G-H arthrokinematics that have been associated with shoulder and elbow injury. There are bony, capsular, and muscular alterations contributing to motion deficits, although capsular and muscular alterations are most likely the major contributors to GIRD and TAMD. Many types of stretches and mobilization techniques have been shown to increase GIR and decrease GIRD and TAMD, and the large majority of athletes will respond to these programs. Because GIRD and TAMD have been linked to injury pathophysiology, have a tendency to progress during the playing season, and respond to corrective exercises, GIRD and TAMD should be screened for both in the preseason and during the season, and mobilization/stretching programs should be routinely used for players at all levels.

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