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Posterior glenohumeral joint capsule contracture

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Abstract

Glenohumeral joint posterior capsule contracture may cause shoulder pain by altering normal joint mechanics. Contracture is commonly noted in throwing athletes but can also be present in nonthrowers. The cause of contracture in throwing athletes is assumed to be a response to the high amount of repetitive tensile force placed on the tissue, whereas the mechanism of contracture in nonthrowers is unknown. It is likely that mechanical and cellular processes interact to increase the stiffness and decrease the compliance of the capsule, although the exact processes that cause a contracture have not been confirmed. Cadaver models have been used to study the effect of posterior capsule contracture on joint mechanics and demonstrate alterations in range of motion and in humeral head kinematics. Imaging has been used to assess posterior capsule contracture, although standard techniques and quantification methods are lacking. Clinically, contracture manifests as a reduction in glenohumeral internal rotation and/or cross body adduction range of motion. Stretching and manual techniques are used to improve range of motion and often decrease symptoms in painful shoulders.

Keywords

Shoulder; joint capsule; contracture; athlete; injury; repetitive strain

INTRODUCTION

The shoulder is a biomechanically complex joint system that is prone to injury, accounting for over 20% of the over 16 million self-reported musculoskeletal injuries annually in the USA [1]. In the UK, prevalence rates for musculoskeletal shoulder pain in the general population are estimated at approximately 10% [2]. One proposed mechanism for the development of shoulder pain is increased stiffness or contracture of the posterior glenohumeral joint (GHJ) capsule [3–6]. Posterior GHJ capsule contracture alters humeral head translations and/or the humeral axis of rotation during movement, creating conditions that lead to joint pathology [7]. Throwing athletes are especially prone to fibrotic contracture of the capsule [7–12] and posterior GHJ capsule stiffness in the general population is also reported [13].

The present review summarizes the currently available posterior GHJ capsular contracture literature, and considers ways of enhancing the science related to this soft tissue alteration. We first review the anatomy and tissue characteristics of the capsule, and follow with a

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Conflicts of Interest

None declared

summary of the theoretical connection between increased posterior GHJ capsule stiffness and shoulder pathology. Next, evidence in support of posterior GHJ capsule contracture as a mechanism of shoulder pathology is presented. Finally, we examine current measurement and treatment techniques for increased posterior GHJ capsule stiffness.

ANATOMY

The GHJ, the articulation between the glenoid of the scapula and the head of the humerus, is enveloped by a synovial capsule that can be divided into three main regions: anterior, posterior and the axillary pouch. The anterior region and axillary pouch are reinforced by the superior, middle and inferior glenohumeral ligaments (Fig. 1). The inferior glenohumeral ligament (IGHL) has anterior and posterior bands separated by the axillary pouch. The posterior capsule is defined as the region extending from the glenoid rim medially to the humeral head laterally, and from the biceps tendon superiorly to the posterior band IGHL inferiorly, with the posterior band IGHL reinforcing the posterior–inferior capsule. The rotator cuff tendons insert onto the outer surface of the capsule and are difficult to distinguish from the capsule on dissection [14]. The thickness of the posterior GHJ capsule is reported to range from 1.0 mm [15–17] to 2.5 mm [18]. Functionally, the posterior capsule limits posterior translation of the humeral head on the glenoid, and it tightens with GHJ internal rotation (IR) and GHJ horizontal adduction (HAD).

The GHJ capsule is primarily composed of synovial cells, fibroblasts and an extracellular matrix (ECM). The ECM is mainly type I collagen fibres [19], constituting 75% to 80% of the tissue dry weight, with elastin and ground substance representing the remaining tissue. Fibroblasts embedded in the ECM maintain homeostasis between collagen synthesis and degradation [20]. The collagen structure is primarily amino acid chains of glycine, proline and hydroxyproline, coiled together in a triple helix arrangement. These triple helices are arranged in a quaternary structure with oppositely-charged amino acids in alignment [21]. Capsule strength is influenced by collagen fibril diameter, cross-links between fibrils and the number of stable amino acid bonds [22].

Collagen fibre bundles in the capsule are oriented in both circular and radial systems [23]. Circular bundles, oriented in the superior–inferior direction, are primarily in the superficial layers of the tissue. Radial bundles, oriented medial–lateral, are present in the deeper tissue layers and are stronger than circular bundles. In the posterior GHJ capsule, a clear pattern of radial and circular fibres oriented at 90° to each other is present between the teres minor insertion and the posterior band IGHL. Moving superior or inferior from this region, the pattern is modified depending on the functional loads placed on the capsule. Radial fibre bundles are evident at the IGHL, suggesting that this region is exposed to and can resist high tensile loads [23].

Tissue and material properties

Although the posterior GHJ capsule is thinner on average than other capsule regions, ultimate strength and failure values are not different than other regions [15]. Additionally, because the posterior GHJ capsule is thinner but does not have lower failure thresholds, the strength and modulus of elasticity are significantly greater than the anterior, superior and inferior regions [15]. Posterior GHJ capsule material properties are not significantly different than the axillary pouch, nor are the material properties significantly different between males and females [16]. Material properties between the anterior band IGHL and three regions of the posterior GHJ capsule (superior, middle and inferior) do not differ appreciably, although failure strain is greater in the anterior band IGHL than in the middle and inferior regions of the posterior GHJ capsule [17]. From these analyses, it appears that the posterior GHJ capsule tissue is mechanically similar to other capsule regions.

Material properties within the posterior GHJ capsule are dependent on the direction in which the tissue is tested [24]. Comparing longitudinal (oriented medial–lateral) and transverse (oriented superior–inferior) tissue samples demonstrated that the tangent modulus and ultimate stress are greater in the longitudinal direction, whereas ultimate strain is not dependent on direction [24]. It is suggested that the posterior GHJ capsule tissue is highly responsive to the multi-axial loading conditions to which it is subjected during shoulder motion, contributing to GHJ stability.

PATHOPHYSIOLOGY

During throwing, average velocities of 7500°/second are generated by the combination of striding forward, hip and trunk rotation, and arm acceleration prior to ball release [25]. Immediately after ball release, shoulder HAD and IR are decelerated with eccentric activation of scapula retractors, horizontal abductors and external rotators. These posterior shoulder muscles control the distractive forces on the GHJ; however, their effectiveness may become limited by fatigue, leading to increased tensile loading on the posterior GHJ capsule. This repetitive tensile loading creates greater than normal mechanical input to the tissue, which responds by becoming stiffer. Over many games and seasons, this normal response results in connective tissue proliferation consistent with Wolff's law. Proliferation may be protective of the capsular tissue initially, although, eventually, it alters joint mechanics and leads to pathology. Longitudinal studies designed to examine this process for posterior GHJ stiffness are needed to confirm or refute this proposed pathomechanism. It is possible that the viscoelasticity of the capsule may limit adaptive changes in stiffness prior to a certain loading threshold. Animal studies or *ex vivo* experiments that examine the tissue's cellular and mechanical responses to repetitive tensile loads are warranted.

At the cellular level, fibroblasts sense changes in mechanical loading and alter their gene and protein expression. This normal biological response to mechanical stimulus is known as mechanotransduction [26]. Although the exact mechanism of mechanotransduction is unknown, a pathway involving cellular components such as the ECM, integrins, cytoskeleton, cell membrane proteins and stretch activated ion channels are likely to mediate the process [27]. Although no *in vivo* studies have demonstrated mechanotransduction at the posterior GHJ capsule, *in vitro* experiments show increased type I collagen synthesis in response to cyclical mechanical loading of ligaments [28, 29]. A similar mechanism in response to repetitive tensile loading of the posterior GHJ capsule may result in increased type I collagen content and a stiffer, less compliant tissue.

An accurate and acceptable term to describe posterior GHJ capsule change is needed. Stiffness is a structural property indicating the resistance of tissue to deformation. Because a change in capsule stiffness will directly impact joint motion, this term is accurate to describe posterior capsule change. Elasticity is a material property indicating the ability of a tissue to return to its original shape after stress is removed. However, a change in elasticity may not necessarily result in a change in range of motion (ROM) and so this term is less useful. Capsule contracture involves a change in tissue composition stemming from altered connective tissue fibroblast activity and, although this term is probably accurate, it describes the process, and not the result of the process. We prefer the term stiffness because it describes a structural property, is independent of material or mechanical property changes, and is related to the functional consequences of contracture or fibrosis.

Cadaver evidence

The first study to experimentally explore the influence of posterior GHJ capsule alterations on shoulder biomechanics was performed by Harryman et al. [30]. Their study examined the effect of posterior GHJ capsule tightening on humeral head translations during passive arm

motion. After suturing a 2-cm overlap into the posterior GHJ capsule, anterior and superior humeral head translations increased and occurred at lower angles of humeral flexion compared to the native capsule. Similar increases in humeral head translations were noted during a cross-body motion. A ‘capsular constraint mechanism’ was proposed where the tight capsule caused humeral head translations in the direction opposite to the tightened region. The results obtained confirmed the plausibility of posterior GHJ capsule tightness as a mechanism for pathology, with increased humeral head translations increasing the risk of rotator cuff tendon impingement.

Subsequent cadaver studies have examined the effects of altering the length of the posterior GHJ capsule. Because increased stiffness of the capsule results in a tissue that resists lengthening, reducing the length of the capsule is used to approximate increased stiffness. In these experiments, capsule length is altered either by suturing a segment of tissue, or by applying radiofrequency thermal energy to shrink the tissue. Both methods consistently result in decreased GHJ IR, mirroring what is seen clinically. Table 1 lists the studies that have altered the length of the capsule, the method used, and the ROM change.

Because increased posterior GHJ capsule stiffness is commonly seen in throwing athletes, cadaver models specific to throwers have also been developed. In these models, the anterior GHJ capsule is stretched and the posterior GHJ capsule is shortened. This combination results in increased GHJ external rotation (ER) and decreased GHJ IR, mimicking the ROM shift seen in throwing athletes. Table 1 includes these experiments. Many of these throwing shoulder models study humeral head translations during simulated throwing motions or positions. Fitzpatrick et al. report that, in the late cocking position, the humeral head was positioned more inferior and less posterior after the anterior capsule was stretched, but was further superior and more posterior after the posterior GHJ capsule was sutured [33]. Grossman et al. created posterior GHJ capsule stiffness by shifting the inferior leaflet of the capsule 10 mm superiorly with sutures [32]. Humeral head position was 1.2 mm more superior and 0.7 mm more posterior after altering the capsule. Huffman et al. quantified kinematic changes during the throwing motion in cadaver specimens after the anterior capsule was stretched and after a 1 cm by 1 cm region of the posterior inferior capsule was sutured [35]. The humeral head was shifted posteriorly by 2.3 mm in maximum ER (late cocking phase), whereas, in full IR (deceleration phase), the humeral head was 3.7 mm more anterior and 4.3 mm more inferior than in the native capsule condition. Clabbers et al. also studied the effects of posterior GHJ capsule tightness on humeral head position in the late cocking phase [36]. The length of the posterior GHJ capsule was decreased 20% and 40% using sutures, and, with 20% tightening, the humeral head translated anterior and superior, whereas 40% tightening resulted in posterior and superior translations.

Two studies have quantified subacromial pressure after altering posterior GHJ capsule length. Poitras et al. did not demonstrate a significant increase in peak subacromial pressure during scapular plane arm elevation after either a 1 cm or 2 cm plication of the posterior GHJ capsule [38]. However, Muraki et al. report increased peak subacromial pressure and increased contact area on the coracoacromial ligament in the follow-through position of throwing after plicating the posterior inferior GHJ capsule [39].

Although cadaver models are an efficient method to examine the effect of increased posterior GHJ capsule stiffness on motion and pathology, limitations exist. Across models, the type of change in capsule length is inconsistent. For example, Harryman et al. used a 2-cm superior to inferior plication [30]; Fitzpatrick et al. contracted the capsule by shifting the inferior leaflet of the capsule adjacent to the glenoid 10 mm superiorly [33]; and Huffman et al. sutured a 1-cm² region of the posterior capsule between the 5 o’clock position and 6 o’clock position on the glenoid [35]. With mean posterior GHJ capsular length from glenoid

to humeral head reported as 4.85 cm [37], a 2-cm change is approximately equivalent to a 40% change in length, whereas a 1-cm² change is closer to 20%. So, although these studies report similar changes in motion after capsule alterations, both precision and consistency in the contracture methods are lacking. It is also unknown how changes in capsule length equate to *in vivo* changes in tissue stiffness and mechanical properties. Another issue specific to the throwing shoulder models is that humeral retroversion is not accounted for in the models but is common in throwers and probably impacts the interpretation of ROM changes. Finally, the tissue changes that happen *in vivo* remain mostly speculative, and the specific region of the posterior GHJ capsule that undergoes change is not precisely identified or quantified. Imaging and/or arthroscopic studies should be the starting point for identifying and quantifying the region of change.

Imaging in support of posterior GHJ fibrosis

Few published studies have used imaging to analyse the posterior GHJ capsule. Tuite et al. used magnetic resonance (MR) arthrogram images of overhead throwing athletes with loss of IR to determine whether there was increased thickness of the posterior GHJ labrocapsular complex [40]. Twenty-six overhead athletes and 26 control subjects were evaluated using fat-suppressed T1-weighted images on a 1.5 T scanner. A blinded examiner quantified two capsulolabrum variables: labral length and thick-capsule labrum length. Labral length, the distance from the lateral glenoid rim subchondral bone to the lateral lip of the labrum, was significantly different between groups (Throwing: 6.4 ± 1.6 mm; Control: 4.9 ± 1.4 mm). Thick-capsule labrum length, the distance from the glenoid rim through the labrum to the point where it thinned to between 1 mm and 2 mm, was also significantly different between groups (Throwing: 8.8 ± 2.9 ; Control: 5.4 ± 2.1 mm). Both measurements were quantified from the oblique axial image slice that included the 8 o'clock position of the glenoid rim. Because these images were of the labrum, not the posterior GHJ capsule, they are an estimate of capsule changes in throwers.

Tehranzadeh et al. also used MR arthrograms from a 1.5 T scanner to examine a series of six professional baseball pitchers with glenohumeral internal rotation deficit (GIRD; loss of internal rotation compared to the contralateral arm) [41]. Images were examined by two board-certified radiologists, and all demonstrated a thickened appearance of the posterior GHJ capsule, specifically a thickened posterior band IGHL on transaxial and coronal oblique images, and a thickened capsule at the axillary recess on coronal images. No quantitative assessments or analyses were reported.

Thomas et al. used ultrasound (US) imaging to quantify posterior GHJ capsule thickness in both shoulders of throwing athletes and quantified thickness using Image J software (NIH, Bethesda MD, USA) [42]. They reported a statistically significant increase in capsule thickness on the dominant shoulder compared to the nondominant shoulder (2.03 ± 0.27 mm versus 1.65 ± 0.28 mm), as well as a 16.5° loss of IR ROM and a 6.3° gain of ER ROM on the dominant shoulder. This application of US to quantify the thickness of the posterior GHJ capsule is promising and would minimize costs associated with MR imaging.

To move forward in the study of the posterior GHJ capsule, it is recommended that standard imaging techniques for assessing the capsule are established. These standards could include the best joint position for imaging, the optimal anatomical landmarks to use for localizing the region of the capsule to be measured, and guidelines on quantifying capsule thickness. Normative values for capsule thickness and the threshold over which a capsule is considered to be thicker than normal should be described. Lastly, the precise location at which capsule thickening occurs could be determined using imaging techniques.

CURRENT CLINICAL PRACTICE

Measurement

Increased posterior GHJ capsule stiffness is often assessed by quantifying GHJ IR with the patient supine and the shoulder abducted to 90° (Fig. 2A). Another method, first described by Pappas et al., quantifies the amount of HAD ROM with the arm in neutral IR/ER [12]. Tyler et al. propose quantifying posterior shoulder stiffness by measuring the amount of HAD when lying on the side with the scapula manually stabilized [8]. Both intra-tester and inter-tester reliability of this measurement were reported as good [intra class correlation coefficient (ICC)= 0.92 and 0.80, respectively], and the correlation among HAD when lying on the side and IR ROM in a sample of collegiate baseball pitchers was $r = -0.61$. In a subsequent study, Tyler et al. reported reduced IR and HAD when lying on the side in patients with shoulder impingement [43].

Myers et al. and Laudner et al. both propose measuring posterior shoulder stiffness by quantifying HAD with neutral IR/ER in supine with the scapula stabilized (Fig. 2B) [10, 44]. The ICC for intra and inter-tester reliability were 0.93 and 0.91, respectively [44]. Concurrent validity of supine HAD was estimated by correlation With IR ROM after eliminating humeral retroversion as a confounder, with $r = 0.72$ [44]. Myers et al. compared HAD when lying on the side to supine HAD, with the reliability of the supine measurement (ICC = 0.91) being slightly better than when lying on the side (ICC = 0.83) [10].

One issue concerning the clinical measurement of posterior GHJ stiffness is that the current methods cannot differentiate between posterior GHJ capsule stiffness and posterior shoulder muscle stiffness. It is possible that posterior shoulder muscles, rather than the posterior GHJ capsule, limit the amount of IR or HAD. Poser and Casonato demonstrated in a case series that manual therapy applied to the posterior shoulder muscles, without stretching or changing the joint position, resulted in substantial increases in IR [45]. Similarly, a single muscle energy stretching technique for the posterior shoulder muscles improved IR and HAD ROM, further supporting muscle tightness as a contributor to ROM change [46]. Another important factor for clinical measurement is that changes in humeral retroversion will influence these ROM measurements. With increased humeral retroversion, a throwing shoulder positioned in neutral IR/ ER will actually be in relative IR. This relative IR may pre-tighten the posterior capsule and result in less joint rotation during a measurement as the capsule reaches maximum length sooner, which may be misinterpreted as posterior GHJ capsule stiffness.

To evaluate several potential posterior GHJ capsule measurements, Borstad and Dashottar quantified strain on the posterior shoulder muscles and posterior GHJ capsule before and after altering capsule length with radiofrequency thermal energy [37]. The largest strains on the capsule were noted in positions of sagittal plane GHJ flexion with maximum IR (Fig. 2C). These positions must be evaluated in human subject studies prior to clinical use.

Clinical measurement of the posterior GHJ capsule can be improved by clearly establishing that the method used is sensitive to stiffness changes of the capsule. Cadaver study is probably necessary to demonstrate that posterior capsule is either the only or primary tissue that impacts the measurement. Similarly, clinical measures that are most sensitive to posterior shoulder muscle alterations and that can quantify humeral retroversion must be developed. Having the ability to distinguish how the capsule, muscle and humerus each contribute independently to the ROM shift at the GHJ will greatly benefit measurement and treatment selection.

Treatment and prevention

Expert clinical researchers suggest posterior GHJ capsule stretching in patients with shoulder pathology [47–50]. The positions most often recommended are the sleeper position and HAD. The sleeper stretch involves positioning the patient lying on the side with the involved shoulder flexed to 90°, then adding GHJ IR. The HAD stretch involves positioning the involved shoulder across the chest and adding adduction overpressure. Both stretches can be administered by a clinician or performed independently by the patient.

Izumi et al. compared posterior GHJ capsule stretch positions by quantifying strain on cadaver capsules [51]. Significantly larger strain values were reported for the upper and middle capsule in 30° scapular plane elevation plus GHJ IR, and for the upper and lower capsule in 30° extension plus GHJ IR. The effectiveness of using these stretch positions for changing the stiffness of the capsule *in vivo* has not been reported.

Evidence for the effectiveness of stretching to decrease posterior GHJ capsule tightness is encouraging. McClure et al. compared the effects of the sleeper and HAD stretches in subjects with at least a 10% loss of dominant shoulder IR [52]. Subjects performed five repetitions of 30-second duration self-stretches every day for 4 weeks. The HAD stretch increased dominant shoulder IR compared to both the contralateral shoulder and a nonstretch control group. The sleeper stretch increased dominant shoulder IR compared to the contralateral shoulder but not to the control group. In another study, Laudner et al. reported small but statistically significant increases in IR immediately after three 30-second duration sleeper stretches [53].

Joint mobilization, the application of manual forces by a clinician, may also decrease stiffness of the posterior GHJ capsule. Cools et al. suggested a progression of patient positions and directions in which force is applied, starting in a neutral, adducted GHJ position with posterior forces applied near the humeral head, and progressing to flexed and internally rotated positions with axial forces applied through the distal humerus [54]. Tyler et al. report improvements in GIRD and posterior shoulder tightness after an intervention combining manual therapy and stretching [55]. Manual therapy included posteriorly directed glides of the humerus on the glenoid in the plane of the scapula, and with the shoulder in maximum internal rotation at 90° abduction. The stretching applied in the study included sleeper stretches and HAD with the scapula manually stabilized. Subjects also performed a home exercise programme of sleeper and HAD stretches.

Finally, arthroscopic release of the posterior GHJ capsule is a surgical option for increased posterior GHJ capsule stiffness, and was reported to completely relieve throwing pain in 14 of 16 throwing athletes. Capsule release also reduced GIRD from 28° to 7° [56].

The optimal treatment positions and doses for correcting posterior GHJ capsule stiffness remain unknown. It is also not known whether or not a home-based course of treatment is as effective as one that is administered by a clinician. Based on tissue/material properties, a key factor for effective treatment of posterior GHJ capsule stiffness is creep, which is the phenomenon where tissue will continue to deform under a constant load when that load is applied over time. To change posterior GHJ capsule stiffness, stretches and mobilizations are likely to be more effective if applied for longer durations, although this has not been examined in a controlled study. Ultimately, the stiff posterior GHJ capsule tissue must become more extensible and/or gain length, and these changes may require alterations in the structural composition of the tissue. Regardless of whether a change in stiffness from treatment is the result of altering the mechanical properties or the structural composition, the underlying cellular mechanisms must also be examined.

Prevention of posterior GHJ capsule contracture is a goal with overhead throwing athletes. Kibler and Chandler report that junior tennis players who performed a HAD stretch as part of a comprehensive stretching programme over 2 years had increased GHJ IR compared to a nonstretching control group, although no injury data were reported [48]. Additional prevention studies are needed and should determine the optimal stretch and dose for maintaining ROM and decreasing injury.

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Fig. 1. Anatomy of the glenohumeral joint. AB-IGHL, anterior band of inferior glenohumeral ligament; MGHL, middle glenohumeral ligament; PB-IGHL, posterior band of inferior glenohumeral ligament; SGHL, superior glenohumeral ligament.

Fig. 2. Measurements used to determine posterior glenohumeral joint capsule tightness. (A) Supine internal rotation; (B) horizontal adduction; (C) low flexion.

Table 1

Studies on the effect of altered the length of the capsule on the range of motion (ROM) change

Reference	Method of tightening capsule	GHJ internal rotation ROM		
		Initial	Final	Percent reduction
Harryman et al.[30]	Suture plication: 2 cm overlap	Not quantified	Not quantified	—
Gagey et al.[31]	Thermal shrinkage: amount not quantified. Aim was to reduce internal rotation ROM 10° to 15°	Not quantified	Not quantified	—
Grossman et al.[32] *	Suture plication: inferior leaflet shifted 10 mm superiorly	13.4°±12.2°	7.4°±11.2°	44%
Fitzpatrick et al. [33] *	Suture plication:			
	Model 1: inferior leaflet shifted 10 mm superior	Model 1:13.4°±38.6°	Model 1:8.8°±7.4° Model2:7.0°±6.8°	Model 1:34% Model 2:57%
	Model 2: capsule sutured medial to lateral by 10 mm or 15 mm	Model 2:16.6°±4.4°		
Gerber et al.[34]	Suture plication:			
	1 Posterior capsule shifted medial to lateral 10 mm	30.8°	9.8° 10.3	68% 66%
	2 Posterior inferior capsule shifted medial to lateral 10 mm			
Huffman et al. [35] *	Suture plication: 10 mm medial to lateral and 10 mm inferior to superior	11.3°	7.3°	35%
Clabbers et al. [36]	Suture plication; medial to lateral shifts:	Not quantified	Not quantified	—
	• 20% posterior inferior			
	• 20% total posterior			
	• 40% posterior inferior			
	• 40% total posterior			
Borstad&Dashottar[37]	Thermal shrinkage: mean decrease in horizontal length 18.9%	23.4°±14.2°	18°±7.6°	23%

* Indicates that model included stretching of anterior glenohumeral joint (GHJ) capsule in addition to posterior GHJ capsule tightening.