The biomechanics of the rotator cuff in health and disease - A narrative review

Abstract

The rotator cuff has an important role in the stability and function of the glenohumeral joint. It is a complex anatomic structure commonly affected by injury such as tendinopathy and cuff tears. The rotator cuff helps to provide a stabilising effect to the shoulder joint by compressing the humeral head against the glenoid cavity via the concavity compression mechanism. To appreciate the function of the cuff it is imperative to understand the normal biomechanics of the cuff as well as the mechanisms involved in the pathogenesis of cuff disease.

The shoulder joint offers a wide range of motion due to the variety of rotational moments the cuff muscles are able to provide. In order for the joint to remain stable, the cuff creates a force couple around the glenohumeral joint with coordinated activation of adjacent muscles, which work together to contain the otherwise intrinsically unstable glenohumeral joint and prevent proximal migration of the humerus. Once this muscular balance is lost, increased translations or subluxation of the humeral head may result, leading to changes in the magnitude and direction of the joint reaction forces at the glenohumeral joint. These mechanical changes may then result in a number of clinical presentations of shoulder dysfunction, disease and pain.

This narrative review aims to highlight the importance of functional rotator cuff biomechanics whilst assessing the kinetics and kinematics of the shoulder joint, as well as exploring the various factors involved in cuff disease.

Introduction

The rotator cuff comprises of four muscles and their respective tendons, namely, supraspinatus, infraspinatus, subscapularis and teres minor. These muscles function to stabilise the shoulder joint dynamically, helping the shoulder to be the most mobile large joint in the body. They also allow rotational motion of the humerus relative to the glenoid surface, aided by the contiguous insertion of the cuff tendons on the proximal humerus. Therefore, it is vital to understand the biomechanical properties of the rotator cuff and their role in the pathogenesis and effects of cuff tears. This narrative review will consider the anatomical structures and biomechanics of the rotator cuff and their role in the development of cuff dysfunction.

Anatomical structures

The supraspinatus muscle originates from the supraspinous fossa of the scapula with its tendon inserting onto the superior and middle facets of the greater tuberosity. Infraspinatus and teres minor originate from the infraspinous fossa with their tendons inserting onto the middle and inferior facets of the greater tuberosity. The subscapularis muscle originates from the subscapular fossa, with its tendon inserting onto the lesser tuberosity. The rotator cuff tendons interdigitate to form a continuous structure near their insertions onto the proximal humerus.[1] The subscapularis muscle has the largest tendon footprint of the four cuff muscles, inserting anteriorly along the medial aspect of the bicipital groove to provide internal rotation. The infraspinatus muscle has the second largest tendon, which inserts with its anterior border overlapping the posterior border of the supraspinatus insertion,[2] to provide external rotation. The supraspinatus muscle has the third largest tendon footprint, which inserts onto the superior facet of the greater tuberosity of the proximal humerus to abduct the shoulder. Finally, the teres minor muscle has the smallest tendon footprint, inserting directly inferior to infraspinatus, assisting the latter to rotate the humerus externally. The subscapularis and supraspinatus tendons combine to provide a sheath that surrounds the long head of biceps tendon, with a tendon slip from supraspinatus forming the roof of the sheath, and fibres from both tendons converging to form the floor. Furthermore, fibrous structures extending from the coracoid process to the interval between the subscapularis and supraspinatus muscles strengthen this region, known as the coracohumeral ligament.[3] These anatomical structures can be seen in figure 1.

Microscopically, a five-layer structure of the cuff and capsule complex near the tendon insertions of the supraspinatus and infraspinatus have been described in a cadaveric study.[4] The first, innermost layer contained superficial fibres of the coracohumeral ligament. The second layer, the main portion of the cuff tendons, has been shown to be composed of closely-packed parallel tendon fibres grouped in large bundles extending directly from the muscle bellies to the insertion on the humerus. The third layer was noted to be a thick tendinous structure but with smaller fascicles than in the second layer, with the fourth layer comprising of loose connective tissue with thick bands of collagen fibres which run perpendicular to the primary fibres of the cuff. This layer also contained the deep extension of the coracohumeral ligament. The fifth and outermost layer was the true capsular layer, in which the fibres were shown to be mostly randomly oriented.[4]

Biomechanics

Kinetics and kinematics

Shoulder movements represent carefully coordinated motion of all the rotator cuff components. For this to be achieved, the humerus rotates around the scapula at the glenohumeral joint (GHJ), the scapula rotates around the clavicle at the acromioclavicular (AC) joint, and the clavicle rotates around the sternum at the sternoclavicular joint.[5] In order to achieve 180 degrees of humeral elevation, movement of all of these components must occur. In normal motion, up to 120 degrees of glenohumeral elevation is permitted within the glenoid fossa. After this point, motion is blocked by impingement of the neck of the humerus on the acromion. For further humeral elevation to occur, the scapula must rotate in a superior direction. This rotation positions the glenoid fossa superiorly, allowing the humerus to elevate through an additional 60 degrees.[6] This combined movement of the scapula and humerus is termed scapulohumeral rhythm.[7] Inman *et al*[8] estimated the ratio between glenohumeral and scapulothoracic joint motion to be approximately 2:1. As the scapula upwardly rotates, it produces elevation of the acromial end of the clavicle, which can be up to 30 degrees.[8]

The scapula is positioned on the thorax approximately 30 degrees internally rotated in the horizontal plane, 3 degrees abducted in the frontal plane, and 20 degrees anteriorly tilted in the sagittal plane.[9] The scapula is known to upwardly rotate by 50 degrees, tilt posteriorly by 30 degrees, and externally rotate by 24 degrees during active scapular plane elevation.[10] Two further movements occur at this articulation in the coronal and sagittal planes. Protraction, defined as the forward movement of the scapula around the thoracic wall, combines linear translation away from the vertebral column, rotation of the scapula around the AC joint (anterior tilt), and internal rotation,[11] whereas retraction is the combination of the opposite of these movements.[12]

The humeral head and the glenoid articular surface show a high degree of conformity and may be considered as a ball-and-socket joint. During active and passive elevation of the arm, the humeral head can translate up to 0.35 mm in the superior-inferior direction in the healthy shoulder. Whereas, anterior-posterior translation has been shown to be significantly larger, with the head translating anteriorly by a mean of 3.8 mm during elevation, posteriorly by 4.9 mm during extension, and 4 mm during horizontal extension.[13,14] A smaller radius of curvature (32.2 vs 40.6 mm) is the primary reason for larger translations seen in the anterior-posterior direction.[15] These translations are thought to be induced by the tightening of the capsuloligamentous structures during motion.

Scapula kinematics may alter in patients with cuff dysfunction and several studies have been conducted to investigate scapular rotation during arm elevation. Lin *et al*[16] utilised 3D motion analysis and surface electromyography to analyse 3D movements of the shoulder complex during functional tasks and compared motion patterns between subjects with and without shoulder dysfunction. They discovered decreased scapular upward rotation in the shoulder dysfunction group. Similar results have been found in other studies.[17,18] Such findings suggest that increased scapular upward rotation may be a positive compensation in the presence of rotator cuff dysfunction. Some studies, however, have found no such differences in scapular kinematics in symptomatic subjects when compared to asymptomatic individuals.[19,20] Discrepancies in scapular upward rotation findings during arm elevation in various studies assessing shoulder impingement may relate to the limited clinical knowledge of the status or severity of cuff involvement, particularly with regard to full or partial thickness tears, or indeed the difficulties and variations in measuring scapular motion. The lack of significant differences, as well as observable clinically important differences, between groups to be detected for all variables consistently is perhaps not surprising as investigations are often undertaken with small sample sizes, which result in limited statistical power for some comparisons, particularly given the large variations seen in the movement patterns of healthy subjects.[10] A further explanation for the lack of significant differences to be identified is the presumed multifactorial aetiology of cuff disease, the limitations of clinical diagnosis, in addition to the variations in the measurements taken and models utilised.

Force couples & stability

The rotator cuff muscles have an essential role in the stability and function of the GHJ. Force couples occur when two opposing muscle groups create a moment around a fulcrum.[2] The rotator cuff creates a force couple around the GHJ with coordinated activation and inactivation of agonist and antagonist muscles, working synergistically to contain the otherwise intrinsically unstable GHJ and prevent proximal migration of the humerus. The deltoid and supraspinatus act as a force couple in the coronal plane, compressing the humeral head to the glenoid in abduction, whereas subscapularis and infraspinatus provide a compressive joint reaction force in the axial plane.[21] This can be seen diagrammatically in figure 2. This mechanism, where shoulder stability is provided by the glenoid concavity and the compressive force generated by the rotator cuff muscles, is known as concavity compression.[22]

The bony stability of the shoulder is insufficient, as the glenoid fossa is only a quarter the size of the articular surface of the humeral head. Therefore, the glenoid labrum, together with the joint capsule and glenohumeral ligaments, aids shoulder stability. Labral tissue increases the depth of the glenoid by 50% and, together with the compressive forces of the rotator cuff, imparts a concave compression on the humeral head into the glenoid. By increasing the effective depth of the glenoid, the labrum also helps maintain a negative intra-articular pressure within the joint, conferring stability.[23] Saha determined that dynamic stability is dependent on several factors.[24] These included the power of the horizontal steerers (rotator cuff), development and tilt of the glenoid, as well as retrotorsion (retroversion) of the head and neck of the humerus. Intramuscular electromyography has been used to investigate the activity of the cuff muscles which provide horizontal stability during movement in various planes. Through this technique, it was shown that in abduction, the subscapularis and infraspinatus muscles stabilised the joint from zero to 150 degrees whereas infraspinatus did so almost independently from 150 to 180 degrees, thus confirming the role of subscapularis and infraspinatus as stabilisers of the joint through this range.[24]

In their anatomical study, Turkel and colleagues[25] concluded from cadaveric and roentgenographic experiments that different soft tissue structures stabilise the shoulder joint at varying degrees of abduction. They determined that at zero degrees of abduction, subscapularis was the dominant stabilising structure, whereas at 45 degrees subscapularis and the middle and inferior glenohumeral ligaments provided a greater contribution to stability. As 90 degrees of abduction was approached, the inferior glenohumeral ligament provided the main stabilising effect to prevent dislocation from occurring during external rotation.[25] Mihata *et al*[26] also previously demonstrated that superior translation of the humerus is significantly increased after a tear of the supraspinatus tendon in their study on eight cadaveric models. Moreover, they showed that whilst patch grafting provided a reduction in the superior translation of the humerus, full restoration of GHJ stability could not be achieved. More recently, Ishihara *et al*[27] described that the superior shoulder capsule plays a vital role in passive stability of the GHJ in their study on seven cadaveric shoulders. The authors reported that a tear in the superior capsule at the cuff insertion on the greater tuberosity, as seen in some partial rotator cuff tears, significantly increased translations in the GHJ in both the anterior and inferior directions compared with those with an intact capsule. It was also discovered that a superior capsular defect, which can be observed in massive cuff tears, significantly increased glenohumeral translation in all directions.

Joint reaction forces

The glenohumeral joint reaction force (JRF) counteracts the combined muscle forces transmitted across the joint. The scale of the JRF depends on the torque generated from the activation of the muscles involved in moving the arm and resisting loads applied along its length.[21] Through dynamic shoulder tests at 90 degrees of abduction, the JRF has been estimated to be 337 ± 88 Newtons (N) when equal forces were applied to the cuff and deltoid muscles.[28] As the cuff and deltoid muscles are the primary abductors and rotators at the GHJ, the magnitude of the JRF during active motion provides an indication of the competence of the concavity compression mechanism (figure 3). It has been demonstrated in previous studies that disruption of the transverse force couple, which occurs in large and massive rotator cuff tears, not only leads to increased translations of the humeral head of up to 8 mm during the initiation of abduction,[29] but also to changes in the magnitude and direction of the JRF at the GHJ.[14,30] Consequently, the degree to which different rotator cuff tear configurations effect the mechanical integrity of the transverse force couple can be determined with respect to their effect on the magnitude and direction of the glenohumeral JRF during simulated active motion. In a study of nine cadaveric specimens where motion of the full upper extremity was simulated using a dynamic shoulder testing apparatus, Parsons *et al*[21] showed that extension of cuff tears beyond the supraspinatus tendon into the anterior and posterior aspect of the cuff led to a significant decrease in the magnitude of the JRF, from 337 N to 126 N. Such tears also resulted in a significant change in the direction of the JRF. These results emphasised the importance of the transverse force couple on GHJ motion, compression and stability.

Development of Cuff Dysfunction

The aetiology of rotator cuff tears is considered to be multifactorial, including extrinsic as well as intrinsic factors, which are summarised in table 1. The coracoacromial (CA) arch has a significant role in rotator cuff disease and comprises of the bony acromion, the CA ligament, and the coracoid process. The supraspinatus traverses through the supraspinatus outlet with the arch immediately above, and therefore, is at risk of compression between two bony surfaces; the CA arch above and the humeral head below. This abutment of the cuff against the CA arch, leading to impingement, tendonitis and cuff tear, was classically thought of as the primary driver of cuff disease.[31] This theory gained further momentum after Bigliani *et al*[32] proposed that a down-sloping acromion in the sagittal plane could impinge upon the anterior cuff, thereby causing cuff tears. Acromial morphology was divided into three types: type I (flat under surface), type II (curved), and type III (hooked). Several authors have published findings showing a correlation between a hooked acromion and the development of a cuff tear,[33,34] including a recent systematic review and meta-analysis by Morelli *et al*[35]. However, several other studies have found that shoulders with a Bigliani Type III acromion are no more likely to have a rotator cuff tear than shoulders with Type I or II acromions.[36,37]

Extrinsic compression can also be caused by factors including the presence of an os acromiale and the CA ligament itself, in addition to spurs arising from the acromion as well as the AC joint.[38] Nyffeler *et al*[39] proposed that the acromion index, a measurement of the lateral extension of the acromion, is associated with a higher incidence of rotator cuff disease. This was supported by Balke *et al*,[40] who concluded that the acromial index and low lateral acromial angle may be associated with a higher incidence of rotator cuff tears. However, intrinsic mechanisms of rotator cuff tendinopathy also exist, which impact on tendon morphology and performance. Neer[30] described cuff disease as progressing through three stages of pathology based on the age of the patient: less than 25 years (stage I), 25 - 40 years (stage II), and greater than 40 years of age (stage III). Advancing age has also been shown to have a negative impact on tendon properties.[41,42]

An inadequate vascular supply of rotator cuff tendons has been associated with cuff tendinopathy pathogenesis. This ‘critical zone’ of decreased vascularity, described by Codman,[7] resides approximately 1 cm from the cuff insertion on the greater tuberosity, and is the most common site for cuff tendon injury. The hypovascularity in this region decreases the healing capacity of tissues, predisposing patients to cuff tendinopathy[43] that tends to worsen with age.[44] However, there have been published studies refuting this notion, where a functional hypoperfusion area or ‘critical zone’ in the cuff was not demonstrated.[45]

Type I collagen fibres predominate in parallel bundles, with the thinner and weaker type III collagen occupying a much smaller proportion (approximately 5%). Non-uniform tissue with a low degree of fibre alignment has been shown to exist near the tendon insertion,[46] correlating with diminished mechanical properties. Histological studies have also shown greater disorganisation in the articular side than the more regularly arranged collagen in the bursal layers of the cuff tendons, which has been proposed to weaken the tendon and precede complete tendon tear.[47]

Cuff tears

Rotator cuff tears typically start at the deep surface of the anterior insertion of supraspinatus, adjacent to the long head of biceps (LHB) tendon, as this area is subject to greater loads even at rest. A popular mechanical narrative related to cuff progression describes rim-rent lesions resulting from degenerative cuff tissue that are found 7 mm[48] or between 13-17 mm[49] behind the biceps pulley. These lesions then induce reactive changes such as sclerosis and small cyst formation on the footprint of the cuff, which can be identified on plain radiographs. These lesions may heal, remain unchanged, or enlarge over time. If the latter occurs, over several months or years, a full-thickness defect will result, ultimately progressing into a small crescent-shaped tear.[48] As the cuff tear propagates and progresses from a small to moderate tear, the strong anterior leading edge of the supraspinatus tendon holds firm and withstands uprooting, whilst the flatter and thinner posterior tendon peels off and displaces easily, making the tear asymmetric or 'L'-shaped. The supraspinatus is thus weakened, allowing the humeral head to sublux superiorly, button-holing between the supraspinatus anteriorly and infraspinatus posteriorly.[48]

Cuff repair & healing

Arthroscopic rotator cuff repair continues to provide a high success rate of subjective and functional results. With modern techniques being utilised, healing of small to large tears (1-4 cm) appears to be improving, with healing rates ranging from 83% to 93%.[50,51] However, successfully repairing massive tears (>4cm) remains a challenge despite surgical advances, with reported failure rates ranging from 21% to 91%. Factors known to be associated with enlargement of tears include increasing symptoms, the involvement of 2 or more tendons, and a lesion of the rotator cable.[52-54]

The double-row repair technique has been shown to provide a more robust repair, resembling the native footprint compared to the classic single-row suture anchor repair. Although the former technique may be expected to decrease the re-tear rate, short to mid-term clinical results have not demonstrated a consistently clear clinical benefit over single-row repairs.[55] More recently, Pogorzelski *et al*[56] have published very encouraging results of transosseous-equivalent rotator cuff repairs using either knotted suture bridge or knotless tape bridge repair techniques. Significant improvements in patient-reported outcomes and excellent survivorship were observed with both techniques at a minimum of 5 years.[56]

Tendon healing following surgical repair generally progresses through three phases. These include an initial inflammatory phase, lasting around a week, followed by a proliferative phase, lasting a few weeks, before entering the final remodelling phase, which lasts many months.[57] During the inflammatory phase, vascular permeability increases and inflammatory cells enter the healing site, which produces several cytokines and growth factors that lead to recruitment and proliferation of macrophages and tendon fibroblasts. During the proliferative and remodelling phases of healing, fibroblasts proliferate and begin to produce, deposit, align and cross-link collagen fibres. In cuff repairs, abundant fibroblasts from the tendon and surrounding tissues produce a disorganised collagen scar tissue at the attachment site between the cuff and bone, composed primarily of type I and III collagen.

The optimal post-repair rehabilitation strategies for cuff tendons are mainly based on studies in the rat rotator cuff model, which have suggested a beneficial effect of immobilisation to prevent post-repair gapping and aid in healing. Protective immobilisation has demonstrated improved healing compared to other post-repair loading protocols such as exercise or complete tendon unloading.[58] The mechanisms behind the benefits of immobilisation are unclear, however, they are likely to include mechanical (prevention of gap formation) and biologic effects (reduced phagocytic macrophage accumulation).[59] However, further studies are needed to assess the most appropriate rehabilitation strategies following the different presentations and techniques used in rotator cuff repair.

Conclusions

The rotator cuff tendons have an essential role in the stability and function of the shoulder. In this article, we have provided the reader with current concepts concerning rotator cuff biomechanics, cuff disease mechanisms, the importance of maintaining balanced force couples, and the effect this may have if this mechanism is lost. It has also highlighted the critical function the superior cuff and capsule have in maintaining glenohumeral joint stability, all of which have implications to both the surgical techniques being considered and the subsequent rehabilitation protocols applied.

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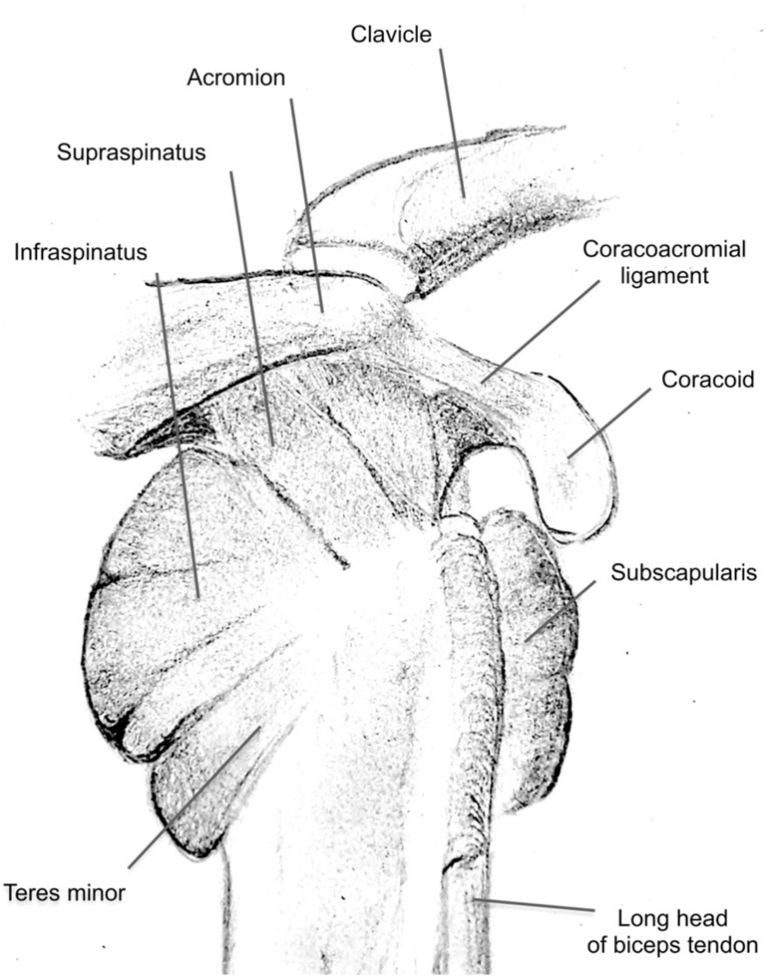

Figure 1 - Anatomical structures around the shoulder, in particular showing the insertions of the rotator cuff tendons (courtesy of shoulderpedia.co.uk)

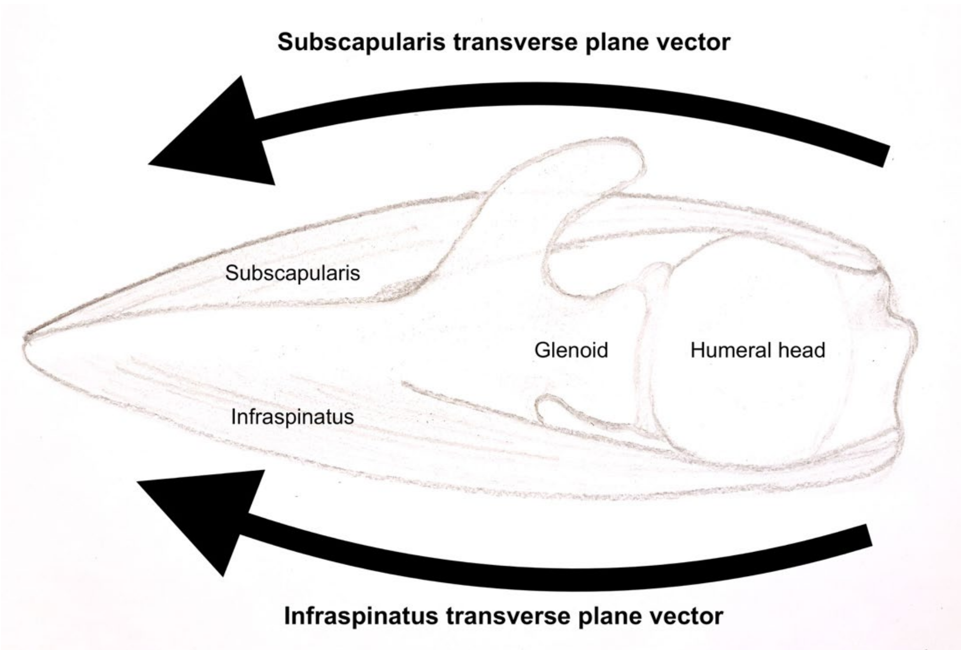

Figure 2 - Diagrammatic representation of the transverse plane force couple (courtesy of shoulderpedia.co.uk)

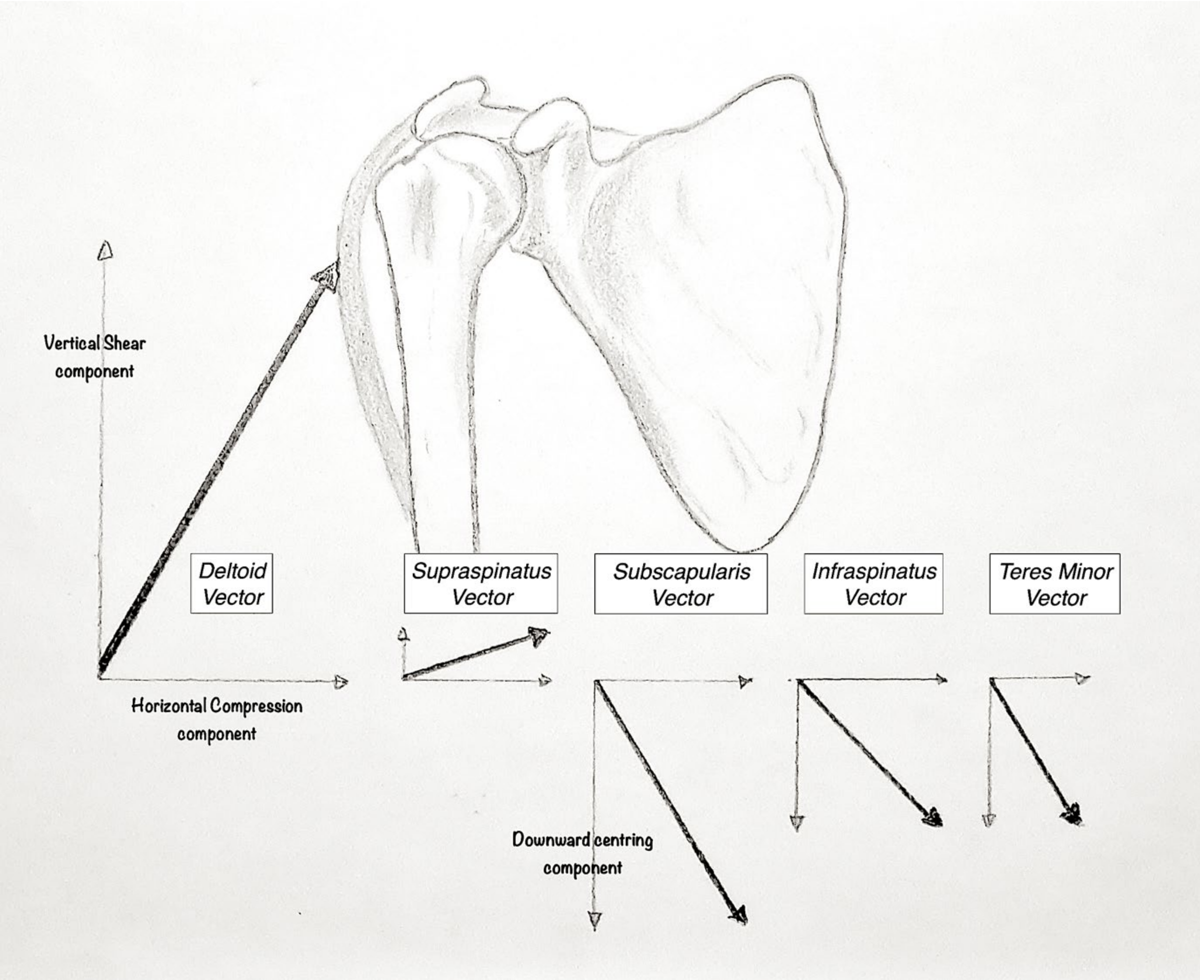

Figure 3 - Diagrammatic representation of the joint reaction forces acting across the shoulder joint (courtesy of shoulderpedia.co.uk)

Extrinsic factors	*Intrinsic factors*
Downsloping acromion	Age-related degeneration
*CA ligament / Os acromiale	Vascular insufficiency
AC joint spurs	Tendon properties
Lateral extension of acromion	

**CA = coracoacromial; AC = acromioclavicular*

Table 1 - Factors associated with the aetiology of cuff tears