



Review

Mechanisms of rotator cuff tendinopathy: Intrinsic, extrinsic, or both? ☆

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ABSTRACT

The etiology of rotator cuff tendinopathy is multi-factorial, and has been attributed to both extrinsic and intrinsic mechanisms. Extrinsic factors that encroach upon the subacromial space and contribute to bursal side compression of the rotator cuff tendons include anatomical variants of the acromion, alterations in scapular or humeral kinematics, postural abnormalities, rotator cuff and scapular muscle performance deficits, and decreased extensibility of pectoralis minor or posterior shoulder. A unique extrinsic mechanism, internal impingement, is attributed to compression of the posterior articular surface of the tendons between the humeral head and glenoid and is not related to subacromial space narrowing. Intrinsic factors that contribute to rotator cuff tendon degradation with tensile/shear overload include alterations in biology, mechanical properties, morphology, and vascularity. The varied nature of these mechanisms indicates that rotator cuff tendinopathy is not a homogenous entity, and thus may require different treatment interventions. Treatment aimed at addressing mechanistic factors appears to be beneficial for patients with rotator cuff tendinopathy, however, not for all patients. Classification of rotator cuff tendinopathy into subgroups based on underlying mechanism may improve treatment outcomes.

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Disorders of the rotator cuff (RC) or associated tissues are the most common problem of the shoulder (Chard et al., 1991; Van Der Windt et al.,

1995; Vecchio et al., 1995). The prevalence of RC disease, specifically partial and full thickness RC tendon tears, has been shown to increase as a function of age starting at 40 years (Iannotti et al., 1991; Milgrom et al., 1995; Sher et al., 1995; Tempelhof et al., 1999), and to exceed as much as 50% by the age of 60 years (Milgrom et al., 1995; Sher et al., 1995). Furthermore, RC disease contributes to pain and disability (Bartolozzi et al., 1994; Duckworth et al., 1999; Macdermid et al., 2004; Smith et al., 2000), and has an impact on health-related quality of life (Macdermid et

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al., 2004). Randomized trials suggest that the short and long-term outcomes of patients with RC tendinopathy treated with surgery are comparable to conservative treatment that includes exercise or exercise combined with a multimodal rehabilitation program (Brox et al., 1993, 1999; Haahr and Andersen, 2006; Haahr et al., 2005). However, regardless of treatment, more than a third of patients do not have a successful outcome with continued persistent pain and disability (Brox et al., 1993, 1999). Given the high prevalence and continued pain despite treatment in patients with RC disease, research to better understand the mechanisms to improve effectiveness of treatment, guide specific treatment choices, and better prognosticate treatment outcomes is necessary.

RC disease has been classically described as a progressive disorder of the RC tendons which begins with an acute tendinitis, progresses to tendinosis with degeneration and partial thickness tears, and results in full thickness rupture (Neer, 1983). The diagnostic terms of RC tendinitis and tendinosis represent tendon pathology subsets of RC tendinopathy. RC tendinitis is often used to define both acute and chronic pain associated with, by definition, inflammation. However, histological studies of patients with RC disease have found minimal to no inflammatory cells in the RC tendons (Fukuda et al., 1990) and subacromial bursa (Sarkar and Uthoff, 1983). Tendinosis is the diagnostic label for tendon pathology that is degenerative with or without inflammation. In contrast, RC tendinopathy is used to signify a combination of pain and impaired performance associated with RC tendons (Alfredson, 2003, 2005). Tendinopathy is a preferred term, to indicate a clinical diagnosis without knowing the specific underlying mechanism or tendon pathology (Almekinders, 1998; Fredberg and Stengaard-Pedersen, 2008). The focus of this review is RC tendinopathy which includes external or internal impingement, tendinitis, tendinosis with degeneration and partial thickness tendon tears. Full thickness tendon tears are unique and beyond the scope of this review.

Mechanisms of RC tendinopathy have been classically described as extrinsic, intrinsic or a combination of both. Extrinsic factors are defined as those causing compression of the RC tendons, while intrinsic mechanisms are those associated with degeneration of the RC tendon. Neer proposed an extrinsic mechanism to the etiology RC tendinopathy with compression of the RC tendons and associated tissues within the subacromial space under the anterior aspect of the acromion or surrounding structures (Neer, 1972) and coined this subacromial impingement syndrome (Neer, 1983). The diagnosis of “subacromial impingement” inherently implies an extrinsic compression mechanism due to narrowing of the subacromial space, which may not accurately represent all RC tendon pathology. A unique extrinsic mechanism, internal impingement, has been described particularly in overhead athletes (Burkhart et al., 2003; Jobe, 1995; Kibler, 1998; Kvitne and Jobe, 1993). Internal impingement occurs due to compression of the articular side rather than the bursal side of the RC tendons, between the posterior superior glenoid rim and humerus when the arm is in full external rotation, abduction, and extension (Davidson et al., 1995; Edelson and Teitz, 2000). Although internal impingement can be considered an extrinsic mechanism, narrowing of the subacromial space is not a hallmark finding. In contrast to extrinsic mechanisms of RC tendinopathy, Codman postulated an intrinsic mechanism due to degeneration within the tendon (Codman and Akerson, 1931), confounded by aging (Iannotti et al., 1991; Milgrom et al., 1995; Sher et al., 1995; Tempelhof et al., 1999). Fig. 1 illustrates the mechanisms and the relationships of the varied mechanisms of RC tendinopathy. Despite the debate over the pathogenesis, evidence indicates that the etiology of RC tendinopathy is multi-factorial and likely both intrinsic and extrinsic mechanisms play a role (Table 1).

1. Extrinsic mechanisms of rotator cuff tendinopathy

Extrinsic mechanisms of RC tendinopathy that result in bursal-sided RC tendon compression due to narrowing of the subacromial

space include anatomical factors, biomechanical factors, or a combination. The subacromial space is the interval between the coracoacromial arch, anterior acromion and the humeral head (Neer and Poppen, 1987). The acromiohumeral distance (AHD), a linear measure between the acromion and the humeral head used to quantify the subacromial space, has been studied in patients with RC disease using magnetic resonance imaging (MRI) (Graichen et al., 1999; Hebert et al., 2003; Saupé et al., 2006), ultrasonography (Azzoni and Cabitza, 2004; Azzoni et al., 2004; Cholewinski et al., 2007; Desmeules et al., 2004) and radiographs (Norwood et al., 1989; Nove-Josserand et al., 2005; Petersson and Redlund-Johnell, 1984; Saupé et al., 2006; Weiner and Macnab, 1970). AHD is normally between 7 and 14 mm in healthy shoulders, but is reduced in those with RC tendon tears (Azzoni and Cabitza, 2004; Azzoni et al., 2004; Ellman et al., 1986; Golding, 1962; Weiner and Macnab, 1970). Furthermore, AHD less than 7 mm with the arm at rest is a predictor indicator of less favorable surgical outcome (Ellman et al., 1986; Norwood et al., 1989; Walch et al., 1992; Weiner and Macnab, 1970). However, patients with RC tendinopathy do not consistently present with significant deficits in subacromial space narrowing with the arm at rest (Azzoni and Cabitza, 2004; Desmeules et al., 2004). Only measures of subacromial space taken with muscle activation are useful to detect deficits related to biomechanical factors that “functionally” narrow the subacromial space (Graichen et al., 1999). In a series of MRI studies, AHD during active arm elevation was smaller in subjects with RC tendinopathy compared to healthy shoulders (Allmann et al., 1997; Graichen et al., 1999; Hebert et al., 2003). Limited evidence suggests that changes in the subacromial space linear distance or extent of narrowing with arm elevation is a sensitive marker of RC tendinopathy (Cholewinski et al., 2007), and may predict the outcome of rehabilitation (Desmeules et al., 2004). Further research that examines changes in subacromial space with active arm elevation in patients with RC tendinopathy is advocated and may be useful to identify the presence of an extrinsic mechanism influencing the articular side of the RC tendons.

1.1. Anatomical factors

Anatomical factors that may excessively narrow the subacromial space and outlet to the RC tendons include variations in shape of the acromion (Bigliani et al., 1991; Epstein et al., 1993; Gill et al., 2002; Ogawa et al., 2005), orientation of the slope/angle of the acromion (Aoki et al., 1986; Edelson, 1995; Toivonen et al., 1995; Vaz et al., 2000) or prominent osseous changes to the inferior aspect of the acromio-clavicular (AC) joint or coracoacromial ligament (Farley et al., 1994; Nicholson et al., 1996; Ogawa et al., 2005). Bigliani et al. described the role of the shape of the acromion as an extrinsic mechanism of RC tendinopathy by describing the morphologic condition of the acromion as a Type I (flat), Type II (curved), or Type III (hooked) (Bigliani et al., 1986). An association between acromion shape and severity of RC pathology has been well documented (Bigliani et al., 1991; Epstein et al., 1993; Gill et al., 2002; Ogawa et al., 2005) with trends of a greater prevalence of Type III, or hooked acromion in patients with impingement (Epstein et al., 1993) and full thickness RC tears (Bigliani et al., 1991; Epstein et al., 1993; Gill et al., 2002; Toivonen et al., 1995). Success of conservative treatment for patients with RC tendinopathy has been related to shape/type of acromion; Morrison et al. found better outcomes in patients with type I acromions (Morrison et al., 1997). These findings were similar to those of Wang et al., who found 89% of patients with type I acromion had a successful response, 73% with type II, and 58.3% of type III (Wang et al., 2000). Whether acromial shape is congenital (Nicholson et al., 1996) or acquired with age (Bonsell et al., 2000; Edelson, 1995; Speer et al., 2001; Wang and Shapiro, 1997) remains controversial. Moreover, the acromial shape classification has been

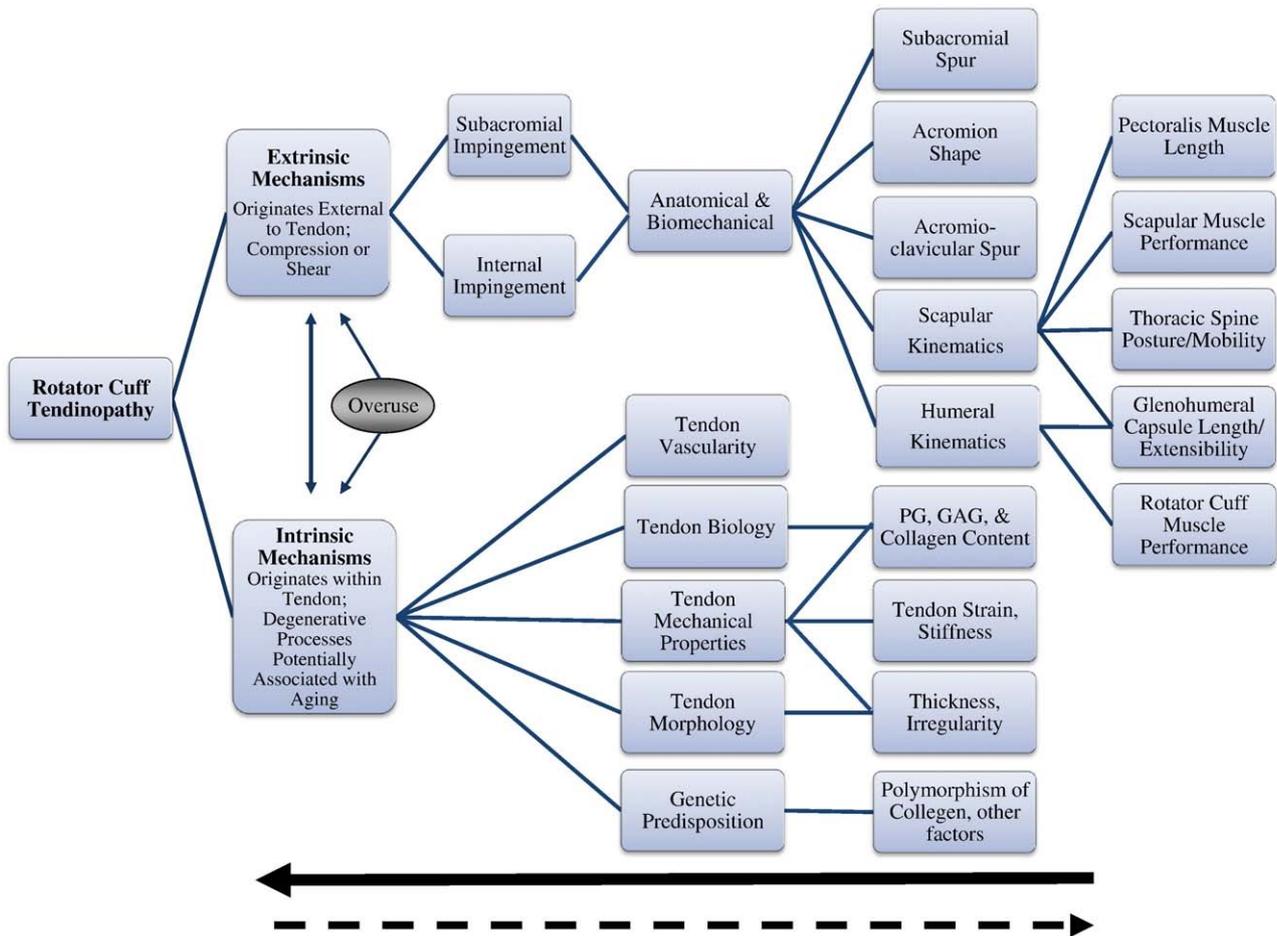


Fig. 1. Extrinsic and intrinsic mechanisms of rotator cuff tendinopathy. Lines indicate non-directional evidence of these relationships.

questioned because of poor interobserver reliability (Jacobson et al., 1995; Zuckerman et al., 1997).

Measurement of the slope or angle of the acromion is another method to capture the acromial shape, and both have been proposed to cause RC tendon compression (Aoki et al., 1986; Edelson, 1995; Toivonen et al., 1995; Vaz et al., 2000). A flatter slope or more horizontal position of the acromion is associated with subacromial impingement (Edelson, 1995), degenerative changes of the RC (Aoki et al., 1986; Toivonen et al., 1995), subacromial spur formation (Aoki et al., 1986; Toivonen et al., 1995), and a greater loss of function in patients with tendinopathy (Vaz et al., 2000). Similarly, other

anatomical factors like large subacromial spurs, thickening or ossification of the attachment of the coracoacromial ligament (CAL) are associated with RC pathology with bursal-sided partial thickness tears (Ogawa et al., 2005) and progression to full thickness RC tears (Farley et al., 1994; Nicholson et al., 1996; Ogawa et al., 2005); however, these same osseous changes in CAL have also been documented with age (Edelson, 1995).

Arthritic changes of the AC joint have also been theorized to contribute to external mechanical impingement of the RC tendons (Neer, 1972, 1983; Petersson and Gentz, 1983). The AC joint undergoes radiographic degeneration with age including narrowing of the joint space and development of osteophytes at the distal clavicle and acromion articulation (Cuomo et al., 1998; Nicholson et al., 1996; Petersson, 1983; Petersson and Redlund-Johnell, 1983). Inferior spurs off the distal clavicle associated with AC joint arthrosis correlate with the presence of RC pathology (Cuomo et al., 1998; Petersson and Gentz, 1983).

There is substantial evidence that anatomical variants such as subacromial spurs, AC joint spurs, and acromial shape may contribute biomechanically to an extrinsic mechanism of RC tendinopathy and progressive RC disease; however, the presence of these alone may be insufficient to result in RC tendinopathy. Soslowky et al. (2002) found that external mechanical compression of RC tendons in rats exposed to normal cage activity did not cause pathological changes, but when combined with overuse activity had a significant effect on tendon injury. Therefore, bony anatomy such as a hooked acromion may not necessarily cause, but predispose an individual to RC tendinopathy. Supporting this theory of a requisite overuse exposure, symptomatic RC disease is more often present in dominant than non-dominant shoulders (Yamaguchi et al., 2006).

Table 1
Rotator cuff pathological mechanisms.

Distinguishing features	Intrinsic	Extrinsic	Extrinsic-internal impingement
Conceptual mechanism	Degeneration of the tendon where tensile loading exceeds the tendon's intrinsic healing and adaptive responses	Compression of the tendon within the subacromial space from anatomical or biomechanical abnormalities	Compression of the tendon posteriorly between the humerus and glenoid rim
Pathology	Intratendinous and articular-sided tendon pathology without coracoacromial abnormalities	Bursal-sided tendon pathology with coracoacromial abnormalities more common	Articular-sided pathology without coracoacromial abnormalities. May be related to glenohumeral joint instability.

1.2. Biomechanical factors

Biomechanical factors that can lead to extrinsic mechanical RC tendon compression include abnormal scapular and humeral kinematics, postural abnormalities, rotator cuff and scapular muscle performance deficits, and decreased extensibility of pectoralis minor or posterior shoulder tissues. Scapular and humeral kinematic abnormalities can cause dynamic narrowing of the subacromial space leading to RC tendon compression secondary to superior translation of the humeral head (Deutsch et al., 1996; Hallstrom and Karrholm, 2006; Keener et al., 2009; Ludewig and Cook, 2002; Paletta et al., 1997; Royer et al., 2009) or aberrant scapular motion that causes the acromion to move inferiorly (Ludewig and Cook, 2000). Postural abnormalities, muscle deficits, and soft tissue tightness factors as external mechanisms can directly influence scapular and humeral kinematics.

1.2.1. Scapular kinematics and influence of posture, muscle deficit and soft tissue tightness

Scapular kinematic abnormalities have been identified in patients with RC tendinopathy compared to healthy individuals (Endo et al., 2001; Graichen et al., 2001; Hebert et al., 2002; Ludewig and Cook, 2000; Lukasiewicz et al., 1999; McClure et al., 2006; Warner et al., 1992). Subjects with subacromial impingement generally have decreased scapular posterior tilting (Endo et al., 2001; Ludewig and Cook, 2000; Lukasiewicz et al., 1999), decreased upward rotation (Endo et al., 2001; Ludewig and Cook, 2000; Su et al., 2004), and increased internal rotation (Endo et al., 2001; Hebert et al., 2002; Ludewig and Cook, 2000; Warner et al., 1992) compared to healthy subjects. As a result, the anterior aspect of the acromion may fail to move away from the humeral head during arm elevation and in theory contribute to a reduction of subacromial space and external RC compression (Ludewig and Cook, 2000). The anterior aspect of the acromion has been identified as the predominant site of RC compression or impingement (Brossmann et al., 1996; Flatow et al., 1994; Lee et al., 2001; Neer, 1972, 1983; Yamamoto et al., 2009). In contrast, increased scapular posterior tilting, upward rotation, and superior translation of the scapula have also been identified in patients with RC tendinopathy compared to asymptomatic subjects (Lukasiewicz et al., 1999; McClure et al., 2006). These aberrant patterns are theorized to be a favorable compensatory response to relieve compression of the RC tendons by increasing subacromial space (McClure et al., 2006). While variable patterns of abnormal scapular kinematics in patients with RC tendinopathy have emerged, the differences between groups are small in magnitude which casts doubt upon the significance of these findings related to changes in subacromial space and role of abnormal scapular kinematics as an extrinsic mechanism for all patients with RC tendinopathy.

Interestingly, Graichen et al. suggest that not all patients with RC tendinopathy have altered scapular kinematics, but a subset exists with significant alterations that are greater than 2 standard deviations from the mean of healthy individuals (Graichen et al., 2001). Moreover, patients with scapular alterations classified with obvious scapular dyskinesis (Tate et al., 2009) compared to less obvious, or subtle, alternations may have meaningful abnormal scapular kinematics that impacts the subacromial space and contribute to an extrinsic mechanism of RC tendinopathy. Silva et al. found a greater reduction in subacromial space in elite tennis players with scapular dyskinesis compared to players without dyskinesis (Silva et al., 2010); however, the clinical method used to identify scapular dyskinesis and associated reliability was not reported.

While there is evidence of abnormal scapular kinematics in a subset of patients with RC tendinopathy, the influence of these specific biomechanical alterations on subacromial space remains speculative. Alternatively, passive alterations in scapular position may influence subacromial space (Atalar et al., 2009; Solem-Bertoft et al.,

1993). In a study by Atalar et al., limiting scapular motion by externally binding the scapular down to the thorax while the arm is positioned at 90° compared to unrestricted scapula caused a reduction in subacromial space in healthy individuals (Atalar et al., 2009). In a study by Solem-Bertoft, positioning the scapula of 4 healthy individuals in protraction compared to retraction with sandbags reduced subacromial space (Solem-Bertoft et al., 1993). In contrast to these findings, cadaveric study by Karduna et al. found that inducing scapular upward rotation from a neutral position reduced subacromial clearance (Karduna et al., 2005). Further research is necessary to determine which scapular kinematic alterations are most related to changes in subacromial space and the magnitude of change in scapular kinematics needed to affect the subacromial space.

The mechanisms responsible for scapular alterations found in subjects with RC tendinopathy have not been clearly defined, but have been theorized to include adaptive shortening of the pectoralis minor muscle (Borstad, 2006; Hebert et al., 2002; Kendall et al., 1993; Ludewig and Cook, 2000), posterior shoulder tightness (Borich et al., 2006), aberrant scapular and rotator cuff muscle performance (Ludewig and Cook, 2000), and an increase in thoracic spine flexion or kyphosis (Kebaetse et al., 1999; Ludewig and Cook, 2000; Wang et al., 1999). Subjects with a relatively shorter pectoralis minor muscle length at rest demonstrate increased scapular internal rotation during arm elevation and decreased scapular posterior tilting at higher arm elevation angles (90° and 120°) when compared with subjects with a relatively longer pectoralis minor muscle length at rest (Borstad and Ludewig, 2005). Similarly, overhead athletes with a loss of glenohumeral internal rotation of 20% or more as compared to their opposite shoulder demonstrate increased scapular anterior tilt at end range glenohumeral internal rotation with the arm abducted or flexed to 90° (Borich et al., 2006). Scapular alterations associated with shortened pectoralis minor length and glenohumeral internal rotation deficit are consistent with previous studies of subjects with RC tendinopathy (Endo et al., 2001; Hebert et al., 2002; Ludewig and Cook, 2000; Warner et al., 1992). The relationship between pectoralis minor muscle length at rest has been indirectly linked to pain and functional limitations attributed to RC tendinopathy via alterations in scapular kinematics (Borstad and Ludewig, 2005). The extent of pectoralis minor shortening needed to decrease the subacromial space and contribute to an extrinsic mechanism has yet to be determined.

Aberrant scapular muscle activity has been identified in patients with RC tendinopathy (Cools et al., 2003, 2004, 2005, 2007; Diederichsen et al., 2008; Ludewig and Cook, 2000; Moraes et al., 2008; Ruwe et al., 1994; Wadsworth and Bullock-Saxton, 1997) and been directly linked to abnormal scapular kinematics in patients with RC tendinopathy (Ludewig and Cook, 2000). Of particular interest are the relative contributions of the upper and lower serratus anterior muscles and trapezius muscles, found to stabilize the scapula and induce scapular upward rotation, external rotation, and/or posterior tilt (Bagg and Forrest, 1988; Johnson and Pandyan, 2005; Kronberg et al., 1990) to potentially allow the humeral head to clear the acromion with elevation (Mcquade et al., 1998). Individuals with RC tendinopathy have decreased muscle performance of the serratus anterior in terms of force output (Cools et al., 2004), muscle balance/ratios (Cools et al., 2004), electromyographical (EMG) activity (Diederichsen et al., 2008; Ludewig and Cook, 2000), and latencies in activation (Moraes et al., 2008; Wadsworth and Bullock-Saxton, 1997). Similar deficits have been found in the lower trapezius muscle including increased latencies of muscle onset (Cools et al., 2003) and alterations in maximal EMG activity (Cools et al., 2004, 2007; Diederichsen et al., 2008; Ludewig and Cook, 2000). Relatively small changes in the muscle performance of the scapulothoracic muscles can alter the position of the scapula at a fixed angle of humeral elevation and, in theory, affect the length–tension relationship (point on the length–tension curve) of the RC muscles and the subacromial space.

Thoracic spine kyphosis posture has been directly linked to alterations in subacromial space (Gumina et al., 2008), alterations in scapular kinematics (Finley and Lee, 2003), and thus theorized to contribute to an extrinsic mechanism of RC tendinopathy. An increase in thoracic spine kyphosis/ flexion is associated with a decrease in subacromial space (Gumina et al., 2008) and a decrease in scapular posterior tilt (Finley and Lee, 2003; Kebaetse et al., 1999). These alterations in scapular kinematics are consistent with those found in patients with RC tendinopathy (Endo et al., 2001; Ludewig and Cook, 2000; Lukasiewicz et al., 1999).

1.2.2. Humeral kinematics and influence of posture muscle deficits, and soft tissue tightness

Excessive humeral head migration proximally on the glenoid is theorized to reduce subacromial space and contribute to RC tendon compression. Proximal, or superior, humeral migration and reduction of subacromial space have been used synonymously at times; however, the amount of superior displacement of the humeral head has not been correlated with linear measures or the 3D volume of the subacromial space and may not occur at a 1:1 ratio (refer to Fig. 2). This distinction may be futile in patients with a large RC tendon tears who present dramatic excessive proximal humeral migration with the arm at rest (Keener et al., 2009); however, in patients with RC tendinopathy the changes in subacromial space may only be apparent with active movement (Graichen et al., 2001). The extent of subacromial space narrowing that occurs with superior humeral head translation on the glenoid may be counteracted with scapular rotation that moves the acromion superiorly or posterior which may increase the subacromial space. Furthermore, a combination of aberrant humeral and scapular kinematics could cause a clinically meaningful reduction of the subacromial space. This relationship requires further study.

Proximal migration of the humerus on the glenoid while the arm is at rest is regarded as a sign of advanced RC disease (Bezer et al., 2005; Keener et al., 2009; Norwood et al., 1989; Yamaguchi et al., 2000), and attributed to chronically diminished RC performance to counteract

the superior pull of the deltoid (Deutsch et al., 1996). Similar to subacromial space, patients with RC tendinopathy do not exhibit proximal humeral migration on the glenoid with the arm at rest; but rather demonstrate excessive superior–anterior translations of the humeral head with active arm elevation (Deutsch et al., 1996; Hallstrom and Karrholm, 2006; Keener et al., 2009; Ludewig and Cook, 2002; Paletta et al., 1997; Royer et al., 2009). Patients with RC tendinopathy have presented with a 1.0–1.5 mm greater superior translation (Deutsch et al., 1996; Hallstrom and Karrholm, 2006; Yamaguchi et al., 2000) and 3 mm of greater anterior translation (Ludewig and Cook, 2002) with active arm elevation compared to asymptomatic subjects. Biomechanical mechanisms for excessive proximal humeral migration in patients with RC tendinopathy include shortening of the posterior–inferior glenohumeral joint capsule and decreased RC muscle performance.

Decreased posterior capsule length has been directly linked to excessive anterior–superior humeral translation in cadaveric study (Harryman et al., 1990). Glenohumeral internal rotation range of motion (IR ROM) and horizontal adduction at 90° of elevation are reliable clinical measures (Laudner et al., 2006b; Myers et al., 2007; Tyler et al., 1999; Warner et al., 1990) that potentially assess posterior capsule length. Content validity for IR ROM has been demonstrated in cadaveric study with a reduction of motion after the posterior–inferior capsule was artificially shortened (Gagey and Boisrenoult, 2004; Gerber et al., 2003). Construct validity has been demonstrated for the measure of horizontal adduction range of motion by its ability to identify deficits unique to overhead athletes (Myers et al., 2007). Clinical measures of glenohumeral internal rotation and horizontal adduction range of motion may also be influenced by potential adaptations of the infraspinatus, teres minor, and/or posterior deltoid musculature (Reinold et al., 2008), or osseous changes of humeral and/or glenoid retroversion (Crockett et al., 2002; Osbahr et al., 2002; Reagan et al., 2002; Schwab and Blanch, 2009).

A relationship between the two measures of posterior shoulder tightness, horizontal adduction and IR ROM, have been found in patients with RC tendinopathy (Tyler et al., 2000) and asymptomatic professional baseball pitchers (Laudner et al., 2006b). Posterior shoulder tightness has been demonstrated in patients with RC tendinopathy (Myers et al., 2006; Tyler et al., 2000; Warner et al., 1990). Furthermore, stretching to address impairments of posterior shoulder tightness has been identified as an important component to rehabilitation for patients with RC tendinopathy (Kuhn, 2009), and change in IR ROM was significantly correlated ($r=0.54$) with functional improvement in patients undergoing rehabilitation (McClure et al., 2004). While this mechanism for RC tendinopathy may be prevalent, this is likely not a contributing mechanism of all patients with RC tendinopathy.

Deficits in RC muscle performance contribute to RC tendinopathy, by leading to proximal migration and subsequent intrinsic breakdown or extrinsic impingement (Chen et al., 1999; Deutsch et al., 1996; Royer et al., 2009). In biomechanical studies, decreased RC muscles force, in particular the infraspinatus has resulted in increased superior humeral head translation and decreased abduction torque (Hurschler et al., 2000; Mura et al., 2003; Sharkey and Marder, 1995). Although more recently, the concept that decreased RC muscle performance alone can result in proximal humeral migration has been challenged with an in vivo study. Artificially induced paralysis of the supraspinatus and infraspinatus muscles in 10 healthy individuals resulted in no immediate effects on proximal humeral head translation (Werner et al., 2006). Results of this study suggest that time, or duration of the muscle impairment may also be a factor. Significant decreases in RC muscle peak isometric, concentric, and eccentric torque have been demonstrated in patients with RC tendinopathy compared to asymptomatic subjects (Leroux et al., 1994; Macdermid et al., 2004; Tyler et al., 2005; Warner et al., 1990). Reddy et al. found a decrease in electromyographic (EMG) activity of the infraspinatus, and



Fig. 2. Superior migration of the humerus on the glenoid (solid line) and diminished subacromial space (dotted line) in patient with a chronic large RC tear.

subscapularis from 30° to 60° of active elevation and in the infraspinatus muscle alone from 60° to 90° of active elevation in subjects with tendinopathy compared to healthy subjects (Reddy et al., 2000). Diederichsen et al. found decreased infraspinatus EMG muscle activity with resisted external rotation in patients with RC tendinopathy compared to healthy subjects (Diederichsen et al., 2008). However, alterations in muscle activity were also found in the asymptomatic side leading the authors to propose alterations in muscle activity are a factor in the pathogenesis not a result of RC tendinopathy. Lastly, Myers et al. found a decrease in co-activation ratios of the subscapularis–infraspinatus and supraspinatus–infraspinatus muscles with arm elevation from 0 to 30°, and an increase at elevation above 90° in patients with impingement compared to control participants (Myers et al., 2009). Decreased RC muscle co-activation levels may occur as a result of pain (Myers et al., 2009) or altered scapular or humeral head position or movement, changing the muscle length–tension relationship and therefore muscle force (Michener et al., 2003). Biomechanical consequences of altered RC muscle activity may be an extrinsic mechanism of RC tendinopathy as superior migration may narrow the subacromial space or result in altered stress and intrinsic tendon degradation. Diminished RC muscle performance correlates with patient-rated function and health-related quality of life in patients with RC tendinopathy (Macdermid et al., 2004).

No study has concurrently examined the influence of scapular position on RC muscle activity in patients with RC tendinopathy; however, there is evidence to suggest that a change in scapular position can alter muscle performance (Kebaetse et al., 1999; Kibler et al., 2006; Tate et al., 2008). Kebaetse et al. found a decrease in isometric abduction muscle force with the arm at 90° concurrent with an increase in scapular anterior tilt in healthy subjects actively assuming a slouched trunk posture compared to an erect posture (Kebaetse et al., 1999). Other research has found that passively altering scapular position, as with scapular retraction and scapular reposition tests, influences isometric arm elevation isometric force (Kibler et al., 2006; Smith et al., 2002; Tate et al., 2008), with an increase noted with reposition and conflicting results with scapular retraction. Changes in muscle force may be due to improved proximal stability or alterations in RC muscle length at the same humeral elevation angle.

2. Extrinsic mechanisms for the subgroup of internal impingement

A unique subset of RC tendinopathy with an extrinsic mechanism is internal impingement. Patients with internal impingement tend to present with pain located in the posterior and superior aspects of the shoulder typically while the arm is in abduction and external rotation of the late cocking phase of throwing (Jobe, 1995; Kvitne and Jobe, 1993). In this position, the articular aspect of the RC tendons becomes mechanically impinged between the posterior superior glenoid rim and the humeral head. This is accentuated with further hyperangulation of the humerus to the glenoid with anterior glenohumeral joint instability (Davidson et al., 1995) or in theory, with a reduction in scapular retraction (Burkhart et al., 2003; Kibler, 1998) and posterior tilt (Laudner et al., 2006a). Alterations in scapular kinematics were found in a cohort of baseball players with internal impingement, confirmed with arthroscopy, of greater scapular posterior tilt compared to age matched healthy baseball players (Laudner et al., 2006a). In contrast, a decrease in scapular posterior tilt has been frequently found in patients with RC tendinopathy (Endo et al., 2001; Ludewig and Cook, 2000; Lukasiewicz et al., 1999). Conflicting findings in scapular kinematics may not be due to causative or compensation patterns of RC tendinopathy as previously theorized (Ludewig and Cook, 2000; McClure et al., 2006), but may be a result of differences in underlying mechanism. Further study should examine

potential differences in mechanisms of this unique subgroup of RC tendinopathy.

3. Intrinsic mechanisms of rotator cuff tendinopathy

There is a growing body of evidence to support an intrinsic mechanism. Intrinsic mechanisms of RC tendinopathy influence tendon morphology and performance. Intrinsic factors of RC tendinopathy result in tendon degradation due to the natural process of aging (Iannotti et al., 1991; Milgrom et al., 1995; Sher et al., 1995; Tempelhof et al., 1999), poor vascularity (Biberthaler et al., 2003; Brooks et al., 1992; Fukuda et al., 1990; Goodmurphy et al., 2003; Rathbun and Macnab, 1970; Rudzki et al., 2008), altered biology (Kumagai et al., 1994; Riley et al., 1994a,b), and inferior mechanical properties resulting in damage with tensile or shear loads (Bey et al., 2002; Huang et al., 2005; Lake et al., 2009; Reilly et al., 2003a). A genetic component for the development of RC disease has also been identified (Harvie et al., 2004) and theorized to be related to polymorphism of collagen genes such as found with Achilles tendinopathy (Mokone et al., 2005); however, no specific genotype has yet to be identified as a risk factor for the development of RC disease (September et al., 2007). Furthermore, RC tendinopathy with an intrinsic mechanism may lead to a reduction in subacromial space creating an interaction of intrinsic and extrinsic mechanisms.

The morphology of the RC tendons has been studied in detail. The RC tendon near their insertions have been shown to interdigitate; specifically, the supraspinatus tendon consists of five axial plane layers from the bursal to articular side (Clark and Harryman, 1992) in the critical zone where pathology is most prevalent (Codman, 1934). RC tendinopathy can include symptomatic tendon pathology with degeneration and partial thickness tears that extend through several, but not all layers. It is commonly described as occurring in 3 regions: bursal sided, mid-substance, and articular sided. Furthermore, pathology that occurs within the mid-substance and articular-sided layers without bursal-side involvement is further support for the intrinsic mechanisms of RC tendinopathy (Fukuda et al., 1990; Hashimoto et al., 2003).

3.1. Age-related degenerative changes

Codman first proposed an underlying degenerative process within the tendon which precedes supraspinatus tendinopathy and tears (Codman and Akerson, 1931). Neer (1983) described RC disease as a continuum of pathology with 3 stages characterized by age: less than 25 years for stage I, between 25 and 40 years for stage II, and greater than 40 years of age for stage III respectively. Although Neer's theory is biased by an extrinsic mechanism, age was included as an important factor for RC disease. The prevalence of tendon degeneration including partial and full thickness tears increases as a function of age starting at 40 years (Iannotti et al., 1991; Milgrom et al., 1995; Sher et al., 1995; Tempelhof et al., 1999). Additionally, a prospective study has shown RC disease is progressive and leads to pain and disability in more than 50% of previously asymptomatic individuals in less than 4 years (Yamaguchi et al., 2001).

Age has been shown to have a negative impact on tendon properties. Evidence from biomechanical studies suggest that there is a reduced toe region of a stress–strain curve, decreased elasticity, and decreased overall tensile strength of tendons with age (Woo et al., 2000). Histological study of RC tendons have shown calcification and fibrovascular proliferation degenerative changes in elderly subjects without history of shoulder ailments that were not present in younger subjects, both without a history of shoulder ailments (Kumagai et al., 1994). Also, with age, there is a decrease in total glycosaminoglycan (GAGs) and proteoglycans (PGs) content in the supraspinatus tendon (Riley et al., 1994a). An overall reduction of collagen content and an increased proportion of weaker, more irregularly arranged type III

collagen has been found with aging (Kumagai et al., 1994); however, there is conflicting evidence that these changes in the supraspinatus are not age related but attributed to inferior healing response from microtrauma to the tendon (Bank et al., 1999; Riley et al., 1994a,b). There is no consensus whether changes in the tendon are primarily due to aging or a secondary consequence of reduced mechanical properties that make the tendon more susceptible to injury with repetitive motion. Regardless, age related changes to the tendon appear to be a significant factor in the intrinsic pathoetiology of RC tendinopathy.

3.2. Vascularity

A deficient vascular supply of the human RC tendons has been implicated in the pathogenesis and mechanism of RC tendinopathy. Codman first described the 'critical zone', an area within the supraspinatus tendon approximately 1 cm from the insertion on the greater tubercle with decreased vascularity and the most common site for RC tendon injury (Codman, 1934). Furthermore, this hypovascular zone and resultant diminished healing capacity predisposes one to RC tendinopathy (Biberthaler et al., 2003; Brooks et al., 1992; Fukuda et al., 1990; Goodmurphy et al., 2003; Rathbun and Macnab, 1970; Rudzki et al., 2008) and tends to worsen with age (Rudzki et al., 2008). However, this notion has been challenged with *in vivo* studies that found no apparent region of avascularity in the critical zone (Levy et al., 2008; Longo et al., 2008; Matthews et al., 2006) or evidence that hypovascularity is limited to the articular side and not the bursal side of the tendon (Lohr and Uthoff, 1990; Rudzki et al., 2008).

Research suggests an increased vascular response, or neovascularization, in regions of degenerative changes and smaller tendon tears such as with chronic RC tendinopathy (Fukuda et al., 1990; Goodmurphy et al., 2003; Hashimoto et al., 2003; Kumagai et al., 1994; Levy et al., 2008; Rathbun and Macnab, 1970), that is theorized to be a healing response to tissue microtrauma (Levy et al., 2008; Rathbun and Macnab, 1970). In contrast, tendinopathy that progresses to complete tendon tears have been shown to be avascular (Biberthaler et al., 2003; Fukuda et al., 1990; Matthews et al., 2006; Rathbun and Macnab, 1970). It is unclear whether this avascular condition is a cause of progressive tendinopathy or a consequence of a complete tear. In subjects with RC tendinopathy, imaging with laser or ultrasound color Doppler has been used to detect the presence of neovascularization *in vivo* (Alfredson et al., 2003; Levy et al., 2008). Levy et al. found subjects with acute RC tendinopathy (impingement without tear) had hypovascularity in the supraspinatus tendon compared to subjects without RC disease, while those with chronic RC tears had hypervascularity near the degenerative changes (Levy et al., 2008). The role of vascularity in the intrinsic mechanism of symptomatic RC tendinopathy has not been fully elucidated, however it does appear to be a factor that is influenced by and/or influences the extent and duration of tendon pathology.

3.3. Impact of alterations in tendon matrix on mechanical properties

The composition and organization of the tendon matrix dictate the morphology and mechanical properties of tendons. Tendons are composed of proteins, collagen, and cells referred to as tenocytes. Collagen fibers in tendons are composed predominately of type I molecules in tight and parallel fiber bundles and a small proportion (<5%) of type III collagen fibers that are thinner, weaker, and more irregularly arranged (Kumagai et al., 1994; Riley et al., 1994b). The collagens within the RC tendon matrix are stabilized by formations of cross-links, specifically hydroxylslypyridinoline and lysylpyridinoline (Bank et al., 1999). Within the RC tendons of elderly samples, the distribution of collagen types has been shown to vary with greater proportion of type II and III collagen near the insertional fibrocartilagenous region compared to more proximal tendon (Kumagai et al.,

1994). Since type III collagen fibrils are considered more extensible than type I fibers and tend to be more irregularly arranged, authors theorized that the insertional region of the supraspinatus may be subjected to greater non-linear stresses than other RC tendons. In agreement with this theory, Lake et al. quantified the degree of collagen fiber alignment in different longitudinal sections of the supraspinatus tendon and demonstrated a highly inhomogeneous tissue with a relatively low degree of fiber alignment in the region near the tendon to bone insertion (Lake et al., 2009). These changes correlated with diminished mechanical properties in this region. Furthermore, histological evidence of inferior tissue organization, greater disorganization in the mid-substance and/or the articular side compared to more regularly arranged collagen on the bursal-side layers of the RC tendons has been proposed to weaken the tendon and precede complete tendon tear (Fukuda et al., 1990; Hashimoto et al., 2003).

The intrinsic mechanism of RC tendinopathy assumes the demands placed on the tendon cells at some point exceeds the ability to effectively repair structural deficits (Riley, 2004) resulting in breakdown and eventually pain. Studies that have examined alterations in RC tendon matrix have found no differences in total GAG concentration and PG content (Riley et al., 1994a), but a reduction in total collagen content and an increased proportion of type III collagen fibers (Riley et al., 1994b) in patients with chronic RC tendinopathy compared to cadaveric samples of normal tendon. Additionally, greater tenocyte apoptosis (cell death) has been found in tendons of patients with chronic RC tendinopathy as compared to normal tendons (Tuoheti et al., 2005; Yuan et al., 2002). These matrix alterations are concurrent with morphology characterized by an irregular tendon contour and reduced tendon thickness (Selkowitz et al., 2007; Teefey et al., 2000, 2004; Wiener and Seitz, 1993). Cholewinski et al. found thinning of the RC tendons in patients with chronic unilateral (>6 months) subacromial impingement compared to an asymptomatic individuals without a history of shoulder injury (Cholewinski et al., 2007).

In contrast, an accumulation of GAGs and disorganization of the collagen fibers, which is theorized to cause tendon thickening in RC tendinopathy, has been demonstrated within 12 weeks of the onset of injury (Scott et al., 2007). The supraspinatus appears to have higher rates of collagen matrix turnover compared to other tendons and accelerates in the presence of RC pathology (Bank et al., 1999). In an animal model, tendon cells in the supraspinatus become more chondroid and increase proliferation in an acute injury (Scott et al., 2007). Joensen et al. (2009) found that increased RC tendon thickness of greater than or equal to 0.80 mm compared to the asymptomatic shoulder was associated with RC tendinopathy. The conflicting findings of tendon thickness in this study compared to those of tendon thinning by Cholewinski et al. (2007) may be attributed to the duration of symptoms. Inclusion criteria for Joensen et al. (2009) were greater than 1 month with 30% of the subjects having pain less than 3 months in duration compared to an inclusion criteria of pain greater than 6 months in duration in the study by Cholewinski et al. (mean duration was 7 months, range 6–48 months). Overall, tendon morphology has been suggested to vary based on the duration of tendon injury. An acute injury exhibits increased diffuse tendon thickness associated with matrix changes of a healing response (Malliaras et al., 2009) while a more chronic tendinopathy demonstrates focal defects and tendon thinning associated with degeneration.

3.4. Tensile tissue overload: inhomogeneous mechanical properties

Another proposed intrinsic mechanism of RC tendinopathy is related to the response of the tendons to tensile load, or mechanical properties of the supraspinatus tendon (Bey et al., 2002; Hashimoto et al., 2003; Huang et al., 2005; Reilly et al., 2003a). Lower ultimate strain values (Bey et al., 2002; Huang et al., 2005) and greater tissue stiffness

(Nakajima et al., 1994) to longitudinal loading have been found on the articular side of the supraspinatus tendon near the insertion as compared to the bursal side; although, this was in conflict with results of studies by other investigators who found no differences in mechanical properties between articular (deep) and bursal sided (superficial), but lack of homogeneity between the anterior and posterior supraspinatus to longitudinal loads (Itoi et al., 1995; Lake et al., 2009). Moreover, loading the tendon at various arm positions may result in strain differentials between the articular and bursal side of the supraspinatus. Greater strain has been shown on the supraspinatus tendon articular side with the arm positioned at the beginning of elevation (angles $<30^\circ$ abduction (Bey et al., 2002; Huang et al., 2005) and 62° abduction (Huang et al., 2005)) while Reilly et al. found a progressive increase in articular-sided strain with elevation (angles 0 to 120° abduction) (Reilly et al., 2003a). Greater bursal-sided strain was found when the glenohumeral joint is at mid-ranges (90°) (Huang et al., 2005). While there is inconsistency with the specific results and methods used among these studies, intratendinous degradation is theorized to result from shearing between various portions of RC, specifically the supraspinatus tendon (Fukuda et al., 1990; Lee et al., 2000) potentially due to the distinct mechanical characteristics and force differentials incurred with various loads (Bey et al., 2002; Huang et al., 2005; Lake et al., 2009; Nakajima et al., 1994; Reilly et al., 2003a). Intrastance degeneration in the supraspinatus initiates mid-substance tendon tears and propagates with continued loading to an articular side tendon tear before complete tendon failure (Reilly et al., 2003b). Biomechanical consequences of complex longitudinal and transverse inhomogeneous tendon properties would be exacerbated in combination with extrinsic factors such as repetitive tensile loading induced with daily activities such as lifting or pulling or the strain incurred with the follow through phase of overhead sports.

Other factors than collagen fiber alignment, such as tendon geometry can influence the mechanical properties. Alterations in tendon geometry including tendon irregularity and thinning have been demonstrated in patients with degenerative RC pathology (Cholewinski et al., 2007) which could influence its mechanical properties. Thickening of the tendon associated with an acute healing response to injury may create greater area to distribute forces; however, tendon thinning associated with degenerative or chronic tendinopathy would reduce the surface area for the same load conditions thus may perpetuate injury. A weak correlation has been shown between supraspinatus tendon thickness and in vivo mechanical properties (Bey et al., 2002). There also is a strong correlation between the extent of RC tendon degenerative changes and tensile strength; as tendon degeneration increases the tensile strength decreases (Sano et al., 1997). However, both of these studies examined the mechanical properties of cadaveric tissue samples. However in patients with RC tendinopathy, a decrease in tendon thickness has been shown to be associated with a decrease in muscle performance (Joensen et al., 2009).

4. Subgroups of patients with tendinopathy based on mechanism

Subgroups of RC tendinopathy may exist, based on intrinsic and extrinsic mechanism that may serve to facilitate treatment decision-making for patients with RC tendinopathy. In a cadaver study, bursal-sided tendon degeneration with partial thickness tears were always associated with attritional lesions on the coracoacromial ligament and anterior third of the acromion (Ozaki et al., 1988); however, this was not true of articular-sided RC pathology in which the undersurface of the acromion was almost always normal. Similarly in patients, milder pathological changes of the undersurface of the acromion and less severe RC degenerative changes were found in patients with articular-sided RC pathology compared to bursal sided (Ko et al., 2006). It appears there is a link between pathoanatomy and mechanism of RC

tendinopathy; articular-sided degenerative changes of the tendons are primarily associated with an intrinsic mechanism, and bursal-sided pathologies of the tendons are more associated with an extrinsic mechanism. As each of these distinct mechanisms progress, they may increasingly overlap. A patient with primary extrinsic compression mechanism of RC tendinopathy may progress with degenerative changes to the RC tendons over time. Alternatively, a patient with primary intrinsic degenerative mechanism of RC tendinopathy may progressively lose stabilizing function of the RC resulting in excessive superior humeral migration and extrinsic compression.

The literature suggests using the link between pathology and mechanism to drive treatment choices. For surgical treatment of RC tendinopathy attributable to an intrinsic mechanism, debridement of the RC without acromioplasty has been advocated (Budoff et al., 1998; Goldberg et al., 2001). In contrast, if the RC lesion is attributable to extrinsic mechanism, decompression in the form of an acromioplasty has been proposed as a key component of the surgical procedure (Neer, 1972, 1983; Rockwood and Lyons, 1993). Rehabilitation treatment decision-making for RC tendinopathy is not driven primarily by mechanism but also on impairments. Substantial evidence indicates exercise programs that include strengthening of the scapular stabilizers and RC muscles, flexibility exercises for the posterior shoulder, thoracic spine and pectoralis minor muscle, postural education and activity modification are beneficial in reducing pain and disability in patients with RC tendinopathy (Brox et al., 1993; Haahr et al., 2005; Kuhn, 2009; Lombardi et al., 2008). Manual therapy of the spine and shoulder in addition to exercise programs have been shown to be beneficial, and superior to exercise alone (Bang and Deyle, 2000; Conroy and Hayes, 1998). Exercise programs for patients with RC tendinopathy appear to be biased towards the treatment of outlet impingement, an extrinsic mechanism which is likely not present in all patients with RC tendinopathy. The varied nature of contributing mechanisms indicates RC tendinopathy is not a homogeneous entity, and thus may deserve different treatment interventions.

4.1. Factors for specific subgroups of RC tendinopathy

Mechanism of RC tendinopathy as an internal impingement, extrinsic, intrinsic, or a combination can be used to create patient subgroups. Outcomes of current rehabilitation approaches are not effective for all patients, with $>30\%$ of all patients treated with rehabilitation or surgery considered unsuccessful with persistent pain and disability (Brox et al., 1993, 1999). Improving specificity of treatment may improve treatment outcomes. Despite distinct differences in biomechanical mechanisms that appear to be associated with internal impingement and intrinsic versus extrinsic mechanisms, there is a paucity of research that indicates which clinical examination findings identify the mechanism, and are thus useful to develop specific rehabilitation components. Factors from the patient history and examination that potentially can be used to indicate the underlying mechanism are shown in Table 2 which includes the location of symptoms, pain response with special tests based on altering symptoms, and precipitating event or activity. These factors may provide evidence of a particular contributing biomechanical mechanism and useful for guiding treatment decision-making.

5. Conclusion

RC tendinopathy is a common disorder that poses challenges for effective treatment. Evidence suggests that extrinsic, intrinsic, and combinations of biomechanical mechanisms play a role. Intrinsic mechanisms, such as RC tendon mechanical properties, composition, and vascularity, and extrinsic mechanisms, such as alterations in scapular and glenohumeral kinematics that contribute to either internal and external impingement, appear to be particularly significant factors of RC tendinopathy. Research focused on prognosticating

Table 2
History and examination factors to subcategorize RC tendinopathy based on mechanism.

	Intrinsic	Extrinsic	Combination extrinsic and intrinsic	Extrinsic-internal impingement
History				
Precipitating event	Excessive tensile load (volume, intensity, or frequency) with overuse or traumatic onset	Overhead shoulder use	↔	Overhead abduction and external rotation with activity typically sport/recreation
Pain location	Anterior shoulder, C5 distribution	Anterior shoulder, C5 distribution	↔	Posterior shoulder, C5 distribution
Examination findings				
Anatomical		Bony abnormalities AC joint/acromion		
Thoracic posture and mobility	Mobile thoracic spine	Hypomobile flexed posture		
Scapulothoracic motion	If dyskinesia present – ↑posterior tilt, and/or upward rotation	If dyskinesia present – ↓ posterior tilt, excessive hiking/shrug pattern	↔	If dyskinesia present – ↓ posterior tilt, and/or upward rotation pattern
Symptom alteration tests	Negative (pain unchanged/↑) with scapular assistance/retraction tests	Positive (pain reduces ≥2/10 points) with scapular assistance/scapular retraction tests	↔	Posterior shoulder pain with apprehension test; reduced with relocation test
Posterior shoulder tightness/GH internal rotation deficit	No or mild if present	Yes present	↔	Yes can be present
Pectoralis minor muscle length		Decreased pectoralis minor index	↔	Decreased pectoralis minor index
RC muscle performance	Decreased	Decreased		Decreased
Scapulothoracic muscle performance		Impaired timing, weakness		Possible impaired timing/weakness

treatment outcome including the presence of a particular mechanism or combinations of mechanisms is needed. There are distinguishing characteristics from the history and physical exam that can be used to identify specific biomechanical factors to subcategorize RC tendinopathy as primarily an extrinsic mechanism, intrinsic mechanism, a combination, or internal impingement. Future research is needed to determine whether treatment distinct to these subgroups improves treatment outcomes.

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