

Graded rotator cuff loading

Shoulder pain is a common musculoskeletal complaint with one in three people suffering this at some stage in their life. While this is a burden for individuals of all ages and activity levels, its prevalence appears to increase with age (van der Heijden 1999, Taylor 2005). The actual source of pain in atraumatic shoulder injury is the subject of ongoing debate with current opinion favouring the rotator cuff, associated bursa, and perhaps the long head of biceps (LHB) tendon and its invested sheath (Lewis 2009). Rotator cuff tendinopathy is considered to be the central pathological process forming part of a continuum that culminates in partial and full thickness rotator cuff failure (Cook & Purdam 2009).

To-date there remains a lack of consensus around the aetiology of rotator cuff tendinopathy, although it is broadly agreed that it is **a** multifactorial combination of intrinsic (genetics, age, vascular change and internal loads), extrinsic (contact with surrounding structures) and lifestyle (obesity, metabolic syndrome, and smoking) **factors**. There is likely an interplay between all these factors; however, excessive tissue loading or maladaptive loading is suggested as the principal cause of tendinopathy (Cook et al 2016).

Paradoxically, structural change is not **commonly** observed in **the** tendon regions subjected to the greatest tensile load. In insertional tendinopathy, changes are often observed on the articular side or intrasubstance portions of the tendon (Lewis 2009, Payne et al 1997, Fukuda 2000). Recent papers have suggested that stress shielding or a combination of mixed loading (tension, compression, and shear) may be more important than absolute loading in the onset of tendinopathy at these sites (Soslowsky et al 2000). Further recent work investigating load and soft tissue injury suggests that rate of change of load may be more important than absolute load in the incidence of noncontact soft tissue injury (Gabbett et al 2016).

Tendinopathy models

The tendinopathy model proposed by Cook and Purdam in 2009 and subsequently revised in 2016 describes three stages (reactive, dysrepair and degenerative) of tendinopathy based on clinical, histological, and imaging findings, with the utility to guide management through each stage. The reactive phase describes an early cellular driven proliferative response and is regarded as reversible. Dysrepair and degenerative phases are characterised

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by gradually increasing matrix disorganisation, proliferation of abnormal tenocyte populations, neovascularisation and ultimately widespread collagen disruption and cellular apoptosis. In the rotator cuff it is thought that the degenerative stage ultimately leads to partial and full thickness rotator cuff tears and is considered irreversible (Hashimoto et al 2003, Cook et al 2016).

Current management **practices promote** load modiication in early-stage reactive tendinopathy, providing time for the tendon to return to a normal adaptive pathway. In later stages (dysrepair and degenerative), the focus of treatment is graduated tendon loading aimed at restoring functional load capacity (Cook & Purdam 2009, Cook et al 2016).

Previously described tendinopathy protocols have been largely based on lower limb tendon research. Stanish et al (1986) first proposed eccentric-based loading programs in the rehabilitation of tendinopathy. Alfredson (1998) formalised this concept with a 12-week eccentric loading program for Achilles tendinopathy. There have been several studies that have evaluated this protocol demonstrating satisfactory outcomes in greater than 80 per cent osubjects (Mafi et al 2001, Fahlstrom et al 2003). The results of this program have not been as good **when applied to** insertional Achilles tendinopathy, with one paper reporting satisfactory outcomes in only 32 per cent of cases (Fahlstrom et al 2003). This led to a modification o the original protocol with the aim of reducing compressive load during the exercise. Jonsson et

al (2008) trialled eccentric caldrops restricting ankle dorsiflexion to reduce compression of the tendon over the calcaneum. They achieved good outcomes in 67 per cent of the cohort. This application of loading while minimising compression has been adopted for hamstring origin and gluteal tendinopathies.

Although several studies have demonstrated that exercise is **efficac**ious in treatment of shoulder pain, there appears to be a lack of consensus on the type of exercise and **associated** loading parameters. Traditional protocols have been heterogeneous in nature, including strengthening (concentric and eccentric) and stretching (Ainsworth & Lewis 2007), and commonly **commenced** in the arm-by-side position. Little or no consideration seems to have been given to the exercise modifications adopted in lower limb insertional tendinopathies, namely reduction of mixed tendon loading (compression and shear).

The development of modified protocols for lower limb insertional tendinopathy has led us to question if there is a better application of exercise when commencing rehabilitation for rotator cuff tendinopathy. This paper sets out to describe a loading application for early-stage rotator cuff and LHB loading that we believe minimises mixed loading and urther, provides lexibility to progress load in small, objective increments.

What makes the rotator cuff and biceps different?

The rotator cuff's anatomical arrangement and morphology is far more complex than that found in lower limb tendons. It is made up of the subscapularis anteriorly, the supraspinatus, infraspinatus and teres minor posteriorly. They collectively form a continuous interwoven multilayered aponeurotic tendon that is bound to the glenohumeral capsule (Clark & Harryman 1992, Clark et al 1990) (see Figure/s 1 and 2). The interwoven structure sees the supraspinatus and subscapularis fuse together forming portions of the biceps pulley. The supraspinatus and infraspinatus and the teres minor and infraspinatus also fuse as they approach their respective insertions.



Figure 1.



Figure 2.

These figures highlight the interwoven aponeurotic architecture of the rotator cuff tendon and its footprint. Images courtesy of Springer Nature & Wolters Kluwer Health, Inc.

Further the supraspinatus tendon has five distinct layers described by Clark and Harryman (1992) and is made up of between six and nine individual parallel fascicles separated by seams of loose connective tissue (endotendon) that are rich in glycosaminoglycans (GAG) and large proteoglycans (Fallon et al 2002). GAGs and larger proteoglycans have been suggested to have a role in protecting tendons from compression, shear and frictional stresses. Berenson (1996) describes the rotator cuff morphology as highly suggestive that articular side compression, intratendinous shear, and tensile loading occur commonly during shoulder movements and with the shoulder affording large ranges of movement portions of the cuff may be stretched while others are in compression (see Figure 3).

Bey et al (2002) further supports this hypothesis with the observation of differential strain between the superior and inferior layers of the supraspinatus tendon layers during abduction, being greatest between 30–60 degrees. Significant cuff tendon compression was observed in the adducted shoulder position, being greatest on the inferior layers. Internal rotation whilst in the arm-by-side position was associated with a threefold increase in subacromial pressure when compared with the neutral position, while external rotation accompanied a relative reduction in subacromial pressure. Cook and Purdam (2012) also suggest that loading the shoulder in the adducted position is likely to increase compression of the supraspinatus tendon as it approaches its insertion.



Figure 3. the differential tension and compression across the rotator cuff tendons in different shoulder positions. Images courtesy of John Wiley & Sons.

The proximal LHB has also been found to be rich in large proteoglycans such as aggrecan, along with significantly greater GAG content compared to the distal biceps (Berenson 1996). These findings support the view that the anatomical relationship of the proximal LHB tendon, as it passes over the humeral head, results in significantly greater tendon compression at this site. Cook and Purdam (2012) also note that increasing degrees of shoulder extension are likely to result in increased compression in the proximal LHB tendon.

This data supports for our view that the position and range through which shoulder exercise is performed signicantly impacts both the **mix and magnitude** ofload, to which the, rotator and LHB tendons are subjected. With thesefindings in mind, it is our view that exercise which considers the local **cuff** tendon architecture, shoulder position and range of movement may better manage compression and shear loads, leading to more effective management of tendinopathy at these sites.

How can we apply this in the shoulder?

If not arm by side, then where? Despite shoulder pain being commonly reported during elevation, we in fact suggest commencing shoulder loading in supine at 90 degrees shoulder flexion or side lying in 90 degree abduction. We hypothesise these positions afford several advantages for the rotator cuff (see Figure/s 4 and 5, which demonstrate the low load starting position).

Although 90 degree elevation in standing is a long lever task, requiring signicant cuand deltoid activity (torque) to maintain the shoulder position, in supine it is a nil lever position requiring very little activity. As well, we suggest the depth of movement (like the modiied Alfredson concept) allows a degree of regulation over compression and differential strain. This starting position is low load and allows a high degree of control over the magnitude and type of load being borne by the rotator cuff and LHB **and further, activates** the cuff in synergy with the deltoid rather than in the isolated fashion that occurs in traditional armby-side external rotation.



Figure 4.





Are we loading the rotator cuff?

Wattanaprakornkul et al (2011) examined activation of the rotator cuff in push/pull versus flexion/extension movements of the shoulder. They found that the supraspinatus and infraspinatus were significantly activated in a bench press movement and that there was no significant difference in the level of activation of either muscle when performing the pressing movement, compared with a long lever shoulder flexion movement performed in prone lying (see Figure 6).



Figure 6. Note no signdifference in rotator cuff activity (shaded area)in the bench press movement compared to shoulderperformed insupine. Image courtesy of Elsevier.

What about load progression?

Long lever loading can be introduced with the addition of resistance tubing between the foot and hand (see Figure 7). This addition demands a small increase in torque production (activity) from the deltoid and rotator cuff to maintain **shoulder flexion**, essentially mimicking the effect of gravity (if the patient were standing upright). Load can be objectively progressed over

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time increasing the long lever demand on the cuff and deltoid. In this exercise the cuff is again working together with the deltoid in a functional synergy rather than in isolation that occurs in traditional arm-by-side external rotation.

In summary, load can be manipulated in two ways:

- 1. Increasing dumbbell load through a short lever movement, provides graduated exposure to tensile, compressive and differential strain/shear loads.
- 2. Increasing tubing resistance gradually increases the long lever load (simulated gravity).



Figure 7. This image demonstrate the application of a light resistance tube that acts to mimic gravity on the arm while the patient is in the supine position. Modified depth manages compression across the rotator cuff and LHB tendons.

How much load, how often and how many repetitions?

There are several bodies of evidence that we have lent on to form our exercise prescription parameters. Magnusson et al (2010) raised the concept of a ceiling effect in collagen synthesis following loading. They suggest that incremental increases in collagen synthesis are not achieved when performing greater than 100 repetitions and in fact suggest that degradation may occur with cumulative increases in loading. Collagen synthesis and degradation following loading also results in a net deficit in collagen in the first 24–36 hours, not returning to pre-loading levels for up to 72 hours (see Figures 8 and 9).

The red line in Figure 8 highlights the ceiling effect (approximately 100 repetitions) at which further loading cycles do not result in incremental increases in collagen synthesis. Adapted from data in Magnusson (2010). Figure 9 illustrates the net collagen deficit in the first 24 hours following loading (Magnusson et al 2010).



Figure 8.

Shoulders: simplifying the complex

This course will provide you with an anatomical and biomechanical understanding of shoulder mechanics and pathomechanics. It provides a biomechanical overview of the role of the scapula in shoulder function, a logical strategy for the assessment of the glenohumeral joint and scapula as well as a framework around which to diagnose specific muscle dysfunction and select.

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Figure 9. Images courtesy of Springer Nature.

McCreesh et al (2017) found that the thickness of the supraspinatus tendon increases in subjects with tendinopathy, compared to normal subjects, at one and six hours following an acute bout of loading that returns to baseline levels after 24 hours. Further Rio²⁸ suggests that externally paced HSR programs can both improve physiological adaptation of muscle/ tendon units and modulate tendon pain and corticospinal control of muscle.

With this research in mind we have adopted several considerations when prescribing exercise for the rotator cuff and LHB. We commence with low load, externally paced, slow cycle (time under tension), exercise aimed at producing fatigue. Initial load selection is based on dynamometry, measuring force to the onset of pain in several shoulder positions. The application of "Time under tension' exercise (three seconds concentric/three seconds eccentric) provides an additional parameter, which clinically, we have found to be an important loading variable in tendon rehabilitation rather than weight and repetitions alone.

Initially we commence with a low volume of repetitions to ensure tolerance (2–3 sets of 8–15 repetitions, 20–40 repetitions total for a single exercise), increasing to a maximum of 80–100 total repetitions across 2-3 exercises. Several sets of 15 repetitions, initially performed daily, is generally well tolerated. Essentially, load progression is implemented through volume before increases in weight; however, like Konsgaard's (2009) HSR protocol, repetitions per set will decrease with increasing load (for heavier loads 3–4 sets of 5–8 repetitions per exercise, again 20–40 repetitions initially increasing to a total maximum of 80 repetitions). Ultimately the cuff needs to meaningfully loaded, within tolerance, to achieve desired improvements in capacity.

Given the short-term tendon changes described by McCreesh (2017) and net deficits in collagen following bouts of exercise (Magnusson 2010), with higher relative loads, we suggest second daily exercise to allow adequate tendon recovery (maximum 3–4 times per week). Dynamometry scores from the patient's uninjured shoulder can be used as a guide to the level of load you may ultimately prescribe.

Lower limb tendon loading programs have suggested that some pain ($\leq 4/10$ on VAS) is acceptable (Silbernagel et al 2007). Clinically we are comfortable with patients working with this level of pain during exercise (with appropriate patient education around response to load) if it resolves within 12–24 hours, and there's no marked exacerbation during daily activity.

Several papers examining exercise rehabilitation of shoulder pain have demonstrated good results over 12 weeks or more, as such we encourage graduated loading over at least this time frame to achieve desired tendon adaptation (Kukkoken et al 2014, Haahr et al 2005).

Above all, it is important for patients and practitioners to understand the rehabilitation time frames and the loading requirements to foster realistic expectations of the work required and the likely outcomes. Appropriate time dedication to thorough patient education is key to achieving superior patient outcomes.

This loading approach requires further scientific examination but given our current understanding of tendon response to loading this seems a reasonable clinical guide at this time.

We acknowledge that complete rotator cuff rehabilitation will ultimately take in other standing, arm by side and against gravity positions with mixed loading forces. Our principal goal was to promote further thinking around how we apply load in the context of the more complex rotator cuff and LHB tendons. We believe an approach that considers the local architecture of the rotator cuff and LHB in relation to shoulder position, range of movement and irritability may be more efficacious than traditional exercise programs.

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