ACTIVE STIFFNESS AND STRENGTH IN INDIVIDUALS WITH UNILATERAL ANTERIOR SHOULDER INSTABILITY: A BILATERAL COMPARISON

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ATTESTATION OF AUTHORSHIP

I hereby declare that this submission is my own work and that, to the best of my knowledge and belief, it contains no material previously published or written by another person, nor material which to a substantial extent has been submitted for the award of any other degree or diploma of a university or other institution of higher learning.

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This thesis, that for so long has been ‘nearly finished’, is now done. Thank you.

ABSTRACT

Objective: The objective of this study was to investigate active shoulder stiffness and strength in recurrent shoulder instability. Additionally, this study sought to investigate the relationship between active stiffness, and quality of life, functional outcome, and perceived instability. The relationship between strength and quality of life, functional outcome and perceived instability was also investigated.

Study Design: A cross sectional study of a cohort of subjects with unilateral recurrent anterior shoulder instability was undertaken.

Background: Recurrent shoulder instability affects up to 94% of young athletes following a dislocation (Rowe & Skallerides, 1961; Rowe & Zairns, 1956). Active stiffness is possibly an important factor in protecting the joint from episodes of instability (Myers, 2001; Riemann & Lephart, 2002). While studies have examined passive stiffness at the shoulder, there is little that has examined active stiffness.

Method: Maximal Voluntary Strength (MVS) of the muscles involved in horizontal flexion and their active stiffness at 30%, 50% and 70% MVS was tested in 16 male subjects, with unilateral traumatic anterior shoulder instability. Additionally, quality of life, function and perceived instability were measured using the Western Ontario Stability Index (WOSI), American Shoulder and Elbow Surgeons Questionnaire (ASES) and the Single Alpha Numeric Evaluation score (SANE) respectively.

Results: There was a significant decrease in horizontal flexion strength in the recurrently unstable shoulder. Stiffness was also decreased significantly at 30% and 50% MVS. No statistical difference was demonstrated in stiffness values between limbs at 70% MVS. No significant correlation was shown between active stiffness controlling for strength, and quality of life, function or perceived instability. Additionally, no significant association was shown between strength and quality of life, function or perceived instability.

Conclusion: The observed reduction in stiffness in the unstable shoulder warrants the inclusion of exercises in the rehabilitation program to enhance this parameter in an effort to protect the joint from perturbations that might lead to dislocation. Deficits in strength in horizontal flexion indicate that strengthening exercises may also be valuable to enhance performance in activities that incorporate horizontal flexion. The lack of an association between active stiffness and quality of life and overall function may indicate that stiffness investigated in one plane of motion does not adequately reflect tissue stiffness during functional activities. Further examination into stiffness in the unstable shoulder is necessary, utilising methodology that examines stiffness in all three dimensions simultaneously.

CHAPTER 1: INTRODUCTION

1.1 The Problem

Stability of the glenohumeral joint has been defined as the proper alignment of the humeral head in the glenoid fossa through equalisation of forces around the joint (Lephart & Fu, 2000). At any one time only 25-30% of the humeral head is in contact with the glenoid fossa (Bost & Inman, 1942). While this bony alignment allows for the large range of movement required at the glenohumeral joint, it also contributes to its inherent instability.

Shoulder instability is excessive translation of the humeral head in the glenoid fossa that results in symptoms such as pain (Myers, 2001). Recurrent shoulder instability is manifested most obviously in those under 20 years of age, where rate of re-dislocation have been reported to be between 66% and 94% (Rowe & Skallerides, 1961; Rowe & Zairns, 1956; Simonet, Melton, Cofield, & Ilstrup, 1984; Vermeiren, Handelburg, Casteleyn, & Opdecam, 1983). While the incidence of shoulder dislocations is relatively low amongst the general population (8.2/100,000 person/years (Simonet et al., 1984)), the effect on quality of life can be devastating. Monetary costs from surgical and therapeutic intervention following a dislocation are also significant, reaching approximately $NZ 7000 per dislocation (Accident Compensation Corporation [ACC], 2007). With additional time off work due to injury, the final sum can be considerable. Conservative treatment remains the initial intervention of choice for most patients following shoulder dislocation, as not all patients want or can have operative treatment. A number of impairments have also been noted following recurrent shoulder dislocation, including loss of strength, and fear of re-injury

(Jakobsen, Johannsen, Suder, & Sojbjerg, 2007). In respect to recurrent instability, poor outcomes are often reported following conservative treatment. Several studies (Handoll & Almaiyah, 2003; Handoll, Hanchard, Goodchild, & Feary, 2006; te Slaa, 2004), have shown patients treated conservatively, following primary anterior shoulder dislocation, to have higher rates of recurrent instability, leading to decreased function and quality of life, compared to those treated operatively.

Some researchers have suggested that stiffness of the muscle and ligamentous tissue around the shoulder may be a factor in preventing injury (Myers, 2001; Riemann & Lephart, 2002). Stiffness has been defined as the rate of change in force to the rate of change in angular rotation (Gottlieb, 1978), or length, and is the reciprocal of compliance (McNair, Wood, & Marshall, 1992). Muscle stiffness is affected by muscle size, muscle and tendon architecture and fibre type, with less deformation of stiffer materials in response to an applied external load (Magnusson, 1998). Stiffer muscles are thus more resistant to force and the capsuloligamentous tissue is less likely to be injured upon application of an external force (Borsa, Sauers, & Herling, 2000; McNair et al., 1992).

Studies of shoulder stiffness have been undertaken in vitro. Research utilizing cadavers has investigated the ability of specific anatomical structures to resist external load (Makhsous, 2003), and in vivo studies have examined stiffness of the shoulder moving passively without muscle activation. Passive stiffness however, does not reflect the level of muscle activity during functional activity. Thus investigations into active shoulder stiffness are warranted.

Previous studies have shown that muscle activity increases stiffness at the joint and this mechanism may protect the joint from re-injury and episodes of instability. At the knee joint McNair et al (1992), showed a positive association between active stiffness and function in the unstable knee. However, only one study (Myers, 2001) has been undertaken investigating active stiffness in the unstable shoulder. Myers (2001), perturbated shoulders into external rotation at 20 and 50% of MVS. No difference in stiffness was found between the unstable group and the healthy control group.

One reason that Myers (2001) did not find a difference in stiffness values may be that shoulder dislocation commonly occurs due to a combination of external rotation and horizontal extension (Kirkley, Griffin, Richards, Miniaci, & Mohtadi, 1999). Horizontal extension may be a key plane associated with shoulder instability, and therefore active stiffness in this plane needs to be investigated.

1.2 Purpose of the study

The primary purpose of the current study was to compare bilateral active stiffness measured at 30, 50 and 70% of maximal activation of the shoulder horizontal flexor muscles, in men less than 40 years of age with a history of recurrent unilateral instability. It was hypothesized that shoulders with a history of recurrent instability would have lower active stiffness, than shoulders with no history of shoulder pathology.

A secondary aim was to investigate whether the above mentioned levels of stiffness were associated with levels of function and quality of life. It was

hypothesized that those lindividuals with lower levels of stiffness in the shoulder would suffer from decreased levels of function and quality of life.

As part of the stiffness evaluation, horizontal flexion strength was examined bilaterally. It was hypothesized that subjects with unilateral recurrent shoulder instability would have decreased strength in the unstable limb.

1.3 Significance of the problem

Active stiffness of the shoulder has received little attention from researchers. The findings of the present study could have significance for surgeons, physicians, physiotherapists, rehabilitation specialists, athletes and coaches by providing additional information pertaining to the prevention of recurrent shoulder instability. If stiffness is shown to be decreased and/or be associated with function and quality of life, treatment programmes that incorporate exercises focused upon increasing stiffness might be useful for patients with shoulder instability.

CHAPTER 2: REVIEW OF LITERATURE

2. 1 Introduction

This chapter is divided into five major sections. The first section outlines the search strategy used, and the papers located. The second section provides a review of shoulder instability; examining the classification of shoulder instability, mechanisms of injury, as well as anatomical and pathological sequelae following an episode of shoulder instability. This section also includes alterations in reflex latency that present following episodes of shoulder instability, and the relationship between shoulder instability and strength. The third section begins with a review of stiffness, outlining methods and findings from pertinent studies. The fourth section discusses passive and active shoulder stiffness. The chapter concludes with a summary.

2.2 Literature search

2.2.1 Introduction

Literature pertaining to shoulder stiffness, strength, instability, reflex latency and function was considered for this review.

2.2.2 Search Strategy

2.2.2.1 Inclusion Criteria.

The following criteria were used to determine which studies would be examined in the literature review.

- Studies pertaining to shoulder instability with specific regard to pathoanatomy, alterations in reflex latency, ligamentous stiffness, strength, and treatment.

- In vivo and in vitro investigations of active and passive stiffness in peripheral joints

- Studies of other shoulder pathology and shoulder functional outcome scores.

- Studies of physiology, histology, structure and ultrastructure of muscle and tendon as they related to stiffness.

2.2.2.2 Exclusion Criteria

Studies were excluded from the review if they related to atraumatic shoulder instability or laxity. Additionally, studies were excluded if they were published in the popular press, such as magazines or newspapers. Studies written in languages other than English were also excluded. Studies were not excluded based upon study design or date of publication.

2.2.2.3 Databases and resources searched

Studies were located electronically using the following databases or resources.

- Allied and Complementary Medicine (AMED, 1985+)
- Cochrane Database of Systematic Reviews
- Cumulative Index to Nursing and Allied Health Literature (Cinahl, 1982+)

- EBSCO Health Databases
- Evidence Based Medicine Reviews
- Medline (1950+)
- PEDro (Physiotherapy Evidence Database)
- Proquest
- Sports Discus
- Scopus
- E-Journals

Reference lists of all included studies and texts were manually searched for further studies that may have been overlooked using the electronic search criteria.

2.2.2.4 Search terms used

Keywords used in the search are present in Table 2.1. Index keywords and combinations of keywords were utilized.

Table 2.1: Search terms: Keywords used in the search

Keywords		
shoulder	rotator cuff	titin
glenohumeral	deltoid	actin
dislocat*	latissimus dorsi	myosin
sublux*	infraspinatus	functional outcome
instabil*	supraspinatus	quality of life
stabil*	subscapularis	hill sach*
stable	pectoralis major	bankart
unstable	bicep brachii	morpholog*
laxity	architecture	labrum
anterior*	sensorimotor	ligament
trauma*	propriocept*	inferior
strength	kinaesthe*	superior
peak torque	reflex	middle

isokinetic	articular pressure	treatment
isometric	stiffness	conservative
force	complian*	electromyogra*
muscle	active	EMG
slow twitch	passive	collagen
fast twitch	intrinsic	gender
type	extrinsic	operat*

2.2.2.5 Search returns

The search strategy returned 266 papers that met the inclusion criteria.

2.3 Shoulder instability

2.3.1 Classification

Shoulder instability refers to the inability to maintain the humeral head centrally on the glenoid fossa and is conceptually different from shoulder laxity, where shoulder laxity is the asymptomatic translation of the humeral head on the glenoid fossa. While shoulder laxity may predispose the shoulder to instability, the magnitude of laxity cannot be used to determine clinically unstable shoulders from stable shoulders (Dewing et al., 2008; Sperber & Wredmark, 1994; Urayama, Itoi, Sashi, Minagawa, & Sato, 2003). Lewis et al (2004) stated that shoulder instability is laxity which is abnormal and results in the presentation of symptoms.

Given that a range of different symptoms and conditions exists around the phenomenon of shoulder instability, several classifications have arisen to define and delineate this condition. Thus shoulder instability is commonly classified according to degree, direction, frequency and aetiology. This will now be discussed in more detail.

Degree of instability:

Shoulder instability exists on a continuum from no instability to symptomatic increased joint movement which may result in rotator cuff impingement, through to subluxation, and thereafter a dislocation of the shoulder. A dislocation has been defined as a complete separation of the glenohumeral surfaces (Warner & Caborn, 1992). Subluxation has been defined as excessive glenohumeral translation with accompanying symptoms and a spontaneous realignment. This is thought to be without complete separation of articular surfaces (Lewis et al, 2004; Rockwood & Matsen, 1998; Warner & Caborn, 1992).

Direction of instability:

Direction of instability refers to the direction with which the humeral head has translated relative to the glenoid. It is necessary to distinguish unidirectional instability from multi-directional instability, as they require different treatment approaches, both conservatively and operatively (Lewis et al, 2004). Unidirectional instability is often the result of trauma, with ninety-five percent of traumatic dislocations occurring in the anterior direction (VandenBerghe et al, 2005).

Frequency of instability:

Primary instability refers to shoulders that have suffered only one episode of shoulder instability (Rockwood & Matsen, 1998). Recurrent instability refers to a glenohumeral joint that has been unstable on multiple occasions. This may be repeated dislocation, subluxation, or a combination of both (Rockwood & Matsen, 1998).

Aetiology of instability:

Aetiology of shoulder instability is important to enable differentiation between traumatic and atraumatic causes. Traumatic instability refers to instability episodes that occur as a result of excessive external force applied to the shoulder and may be independent of the degree of tissue laxity in the system.

One study (Rodeo, Suzuki, Yamauchi, Bhargava, & Warren, 1998), investigated the collagenous make-up of patients with traumatic anterior and atraumatic multi-directional instability and found shoulder capsules of patients with a history of shoulder instability to have more stable and reducible collagen cross-links, more cysteine and a higher density of elastin staining, when compared with a control group with no history of shoulder pathology. No difference was seen between the collagen make-up of multi-directional instability and those with traumatic anterior instability, indicating that the difference between traumatic and atraumatic shoulder instability may not be due to structural capsular differences.

2.3.2 Mechanism of injury

The most common cause of anterior shoulder dislocation is trauma; usually a fall onto an outstretched arm in the abducted, externally rotated position; or tackling during contact sport such as rugby or football, with the arm abducted. This shoulder position places the anterior capsule and labrum under some tension, and contributes to the potential failure of these structures (McMahon, 2002; VandenBerghe et al, 2005).

2.3.3 Anatomy and pathoanatomy of the unstable glenohumeral joint

Stability of the glenohumeral joint is provided by both static and dynamic structures about the joint. The static structures include bony morphology, labrum, capsule and ligamentous tissue (inferior, middle and superior glenohumeral ligaments) and negative intra-articular pressure. Dynamic stability is largely provided by the four rotator cuff muscles (supraspinatus, infraspinatus, teres minor and subscapularis). Other muscles such as biceps brachii and pectoralis major also provide stability at a more global level. These features will now be discussed in more detail.

2.3.3.1 Bony morphology

It is well known that bony morphology is responsible for only a limited amount of static stability at the glenohumeral joint. At any one time 25-30% of the humeral head is in contact with the glenoid fossa (Bost & Inman, 1942). Although this bony alignment allows for the large range of movement required at the glenohumeral joint, it contributes to the joint's inherent instability.

The presence of an abnormal glenoid face following injury may result in an increase in episodes of recurrent instability (Itoi, Lee, Berglund, Berge, & An, 2000; Rowe & Zairns, 1981). Itoi et al (2000), assessed the relationship between an abnormal glenoid face due to an osseous Bankart lesions and glenohumeral instability, and found Bankart lesions did not affect stability in the abduction/external rotation position, but did affect stability in abduction/internal rotation. Additionally, stability of the joint was negatively affected when the width of the Bankart lesion was greater than 21% of the glenoid length.

Greis et al (2002) studied eight cadavers and mean contact pressure was recorded with a force transducer. The glenoid was divided into four quadrants and progressive bone loss from 10% to 30% was induced in the humeral head. Bone loss of 30% of the anteroinferior quadrant (as seen in osseous Bankart lesions), increased mean contact pressure in this quadrant by 300%-400% compared with the intact specimens.

More recently Rhee & Lim (2007), investigated the effect of glenoid defect size on function and recurrent instability, following an open or arthroscopic Bankart repair. As the size of glenoid defect increased, the Rowe score decreased, indicating decreased function, stability and motion.

However, not all studies support the association between decreased bony contact and recurrent shoulder instability. Sugaya (2003) studied computer tomography images of the glenoid both obliquely and 'en face', and showed no difference between shoulders with recurrent instability and normal shoulders. Fifty percent of shoulders with recurrent instability had an osseous Bankart lesion, and forty percent had an anomalous configuration of the glenoid. The discrepancies illustrated by Sugaya (2003), indicate that further evidence is required to confirm the association between bony contact area and recurrent instability.

Another bony lesion which can be present with shoulder instability, is a Hill Sachs lesion and refers to the indentation of the posterolateral aspect of the humeral head, as it abuts on the glenoid rim (Chen, Hunt, Hawkins, &

Zuckerman, 2005). Pavlov et al (1985), observed Hill Sachs lesions in up to 80% of acute traumatic dislocations. Furthermore, Taylor & Aciero (1997) examined Hill Sachs lesions of the humeral head and found lesions that comprise less than 20% of the articular surface were not clinically significant, whereas lesions that involved more than 40% of the articular surface, contributed to recurrent instability. Similarly, Kralinger et al (2002), also demonstrated that the size of a Hill Sachs lesion was positively correlated with recurrent instability.

2.3.3.2 Labrum

The congruence of the glenohumeral joint is increased by the presence of a glenoid labrum, a fibrocartilaginous rim that encircles the periphery of the glenoid fossa (Curl & Warren, 1996). The labrum acts as a 'chock block' to prevent translation of the humeral head (Wilk, Arrigo, & Andrews, 1997), and can limit translation of the humerus by up to 20% (Lippit, 1992). It also acts as an attachment point for the biceps tendon and the inferior glenohumeral ligament, the latter being a principal stabilizer of the shoulder joint (Warner & Caborn, 1992). The labrum has also been shown to play an important role in the centering of the humeral head. Fehringer et al (2003) showed that resection of the labrum affects the humeral head position, even at very low loads.

Tears in the anteroinferior labrum following dislocation, are also classified as a type of Bankart lesion and have been reported to occur in as many as 97% of shoulder dislocations (Sugaya, 2003). Another labral lesion that may occur is a SLAP lesion. SLAP lesions are tears in the superior labrum from anterior to posterior and can occur following traction, compression or dislocation of the

glenohumeral joint. Complete superior labral lesions may destabilise the biceps attachment due to increased anterior–posterior translation (Pagnani, Deng, Warren, Torzilli, & Altchek, 1995).

2.3.3.3 Ligamentous Tissue

Ligamentous tissue has been shown to be a critical factor in restraining the humeral head from dislocation. The capsuloligamentous restraints can be divided into the superior glenohumeral ligament (SGHL), middle glenohumeral ligament (MGHL) and inferior glenohumeral ligament (IGHL) and appear as thickenings in the capsular complex.

At 90 degrees abduction, the principal stabilizer is the IGHL (Turkel, Panio, Marshall, & Girgis, 1981) which comprises an anterior, inferior and posterior portion (Burkart & Debski, 2002). The anterior band of the IGHL runs across the mid-portion of the glenohumeral joint and is the principle ligament for restraining anterior and inferior translation of the humerus (Burkart & Debski, 2002). Investigations into the tensile strain of the IGHL have shown high tensile strain in the anterior band of the IGHL (Ticker et al., 1992; Ticker et al., 2006), and higher strain rates at the glenoid side compared with the humeral side (Malicky et al., 2002). This discrepancy in strain rate may account for the localisation of pathology at the capsule-labral interface.

Several authors (Ahmad, Freehill, Blaine, Levine, & Bigliani, 2003; Bankart, 1938; Urayama et al., 2003) have documented increased anterior-medial capsular redundancy in patients with recurrent anterior shoulder instability. While some authors (Uhthoff & Piscopo, 1985) postulate that this is embryonic

in nature, Ahmad et al (2003), point to the significant relationship between the presence of a redundancy with a greater number of dislocations and a greater duration of symptoms prior to surgery. Other studies (Urayama et al., 2003) have also demonstrated increased elongation of the anterior medial capsule of the unstable shoulder, when compared to the contralateral stable shoulder. However, a recent study (Dewing et al., 2008) measuring capsular area failed to find any significant difference between stable and unstable shoulders. It is thought that the difference in these results is largely due to the discrepancies present when recording linear ligamentous elongation vs. capsular volume.

2.3.3.4 Atmospheric Pressure

Capsular volume may also affect intra-articular pressure (IAP). Kumar and Balasubramaiam (1985) first examined the role of IAP in the glenohumeral joint. Twenty-four cadaveric shoulders were fixed on a stand and radiographs were taken before and after venting the capsule with a needle. Puncturing the capsule resulted in a hissing sound as air entered the joint and the shoulder subluxed inferiorly. Before venting the capsule, the pressure measured in the cadavers was approximately –42 cm H_2O and the authors suggested that this negative pressure played a role in stability of the glenohumeral joint. Since this initial finding, there have been several studies (Inokuchi, Olsen, Sojbjerg, & Sneppen, 1997; Itoi et al., 1993; Yamamoto et al., 2006) confirming the presence of IAP in the glenohumeral joint. As these studies have measured pressure and not force, it is difficult to appreciate the contribution of IAP towards restraining the dependent limb, or the limb under load.

2.3.3.5 Shoulder Musculature

Stability of the shoulder is enhanced by the muscles of the shoulder joint. This may be through one of several mechanisms: muscle bulk which provides passive tension, compression of articular surfaces from muscle contraction, tightened passive ligaments as a result of joint motion, restraint from contracted muscle, or co-ordination of muscular forces to centre the humeral head (Abboud & Soslowsky, 2002; Warner & Caborn, 1992).

The rotator cuff co-contracts about the joint to centre the humeral head, thus acting as force couples to increase stability. This force couple is thought to increase stability in one of two ways. One mechanism is co-activation of agonist and antagonist muscles about the joint. This creates a low net torque to increase control and stability about the joint. A second mechanism involves coordination of the agonist and inhibition of the antagonist. This force couple then allows controlled rotation of the joint as the force is transferred through the joint (Abboud & Soslowsky, 2002).

Force couples in the rotator cuff may exist either in the transverse or coronal plane. The transverse force couple refers to the relationship between subscapularis and infraspinatus, while the coronal force couple refers to the relationship between supraspinatus and deltoid. It is thought that the transverse force couple is responsible for centering of the humeral head and affects translations in an anterior–posterior direction (Funk, 2005). This force couple therefore provides resistance to anterior dislocation. Additionally, Abboud and Soslowsky (2002) have demonstrated that in the absence of an intact supraspinatus tendon, the remaining rotator cuff (i.e. the transverse force

couple) are sufficient to provide compression of the glenohumeral joint during abduction, and normal kinematics are preserved.

However, other work by Lee et al (2000), has shown supraspinatus and subscapularis to be most active in controlling glenohumeral joint stability, while Kronberg et al (1990), provided evidence that infraspinatus, subscapularis and latissimus dorsi act as stabilisers in flexion, while subscapularis acts as a stabiliser in external rotation and supraspinatus during extension. Thus, some controversy exists over the stabilising action of subscapularis and infraspinatus, while little evidence exists advocating the role of supraspinatus in the role of resisting anterior translation. The role that subscapularis, infraspinatus, biceps brachii and pectoralis major play in providing stiffness in the stable and unstable joint is of primary importance. Pathological features of these muscles will now be discussed in more detail.

Subscapularis:

Specific attention has focussed upon the role of subscapularis in stabilising anterior translation of the humeral head. Subscapularis is thought to stabilise the glenohumeral joint anteriorly when the arm is abducted and in neutral rotation (Klapper et al, 1992). The dense collagenous structure and tendinous insertion is thought to provide passive stability especially in the hanging arm position, and during low degrees of abduction (Turkel et al., 1981). DePalma et al (1967), first investigated the role of subscapularis in recurrent shoulder instability and noted increased laxity and loss of tension of subscapularis in all 38 cases of recurrent shoulder instability. De Palma et al (1967) stated that subscapularis was the most important buttress against dislocation of the

glenohumeral joint. Initial joint disruption resulted in subscapularis laxity, as well as loss of muscle ‘tone’, power and volume. The muscle was subsequently unable to resist minimal forces which may have dislocated the joint. Halder et al (2000b) also noted the structural properties of subscapularis and observed regional differences between upper and lower portions of the muscle. The superior and mid-superior portions were found to have significantly higher stiffness than the inferior region. The authors surmised that this increased stiffness may help to explain the infrequency of tears in the upper portions of subscapularis. The lower levels of stiffness in the inferior and mid-inferior portions could facilitate shoulder dislocation, as it is this portion that stabilises the joint in the abducted position. This finding concurs with other cadaveric studies (Saha, Das, & Dutta, 1983; Turkel et al., 1981) which have confirmed the role of subscapularis in restraining anterior translation.

Gamulin et al (2002), observed interstitial fibrosis and scarring of subscapularis in 20% of patients with recurrent anterior instability. Biopsies of subscapularis and deltoid were taken during surgery from patients with recurrent anterior instability, and an increased number of type I fibres relative to type II was detected. This change in tissue type may have a significant effect upon the stiffness of the muscle, as will be discussed later. More recently, Tuoheti et al (2005) has observed an alteration in subscapularis morphology in patients with recurrent instability. Utilizing magnetic resonance imaging (MRI), the subscapularis tendon of the unstable shoulder was decreased in thickness of 18.7% and decreased in cross-sectional area by 29.1% compared with the contralateral stable shoulder. This decrease in muscle area may also have implications upon the stiffness of unstable shoulders system, as passive

stiffness is a function of muscle size (Magnusson, Simonsen, Aagaard, Johannsen, & Kjaer, 1997).

The role of subscapularis in providing stability to the unstable joint remains under the spotlight. Itoi et al (1994), found loading subscapularis in a position of 90 degrees glenohumeral external rotation to result in the greatest amount of anterior translation in the intact shoulder. However, more recent architectural studies (Ward et al., 2006), have confirmed that subscapularis is responsible for stability in a position of apprehension, a position in which dislocations may occur. Furthermore, Werner et al (2007) hypothesized that subscapularis may facilitate anterior-inferior dislocation of the shoulder in some positions, depending on 'yet to be indentified' anatomical or biomechanical factors. It may be that restriction from the capsule influences the direction humeral head movement during loading of the subscapularis muscle. Thus, the role of subscapularis in attenuating shoulder instability appears to be a complex one. Further research is needed in this area to quantify the effect of subscapularis disruption following shoulder dislocation and subluxation.

Infraspinatus and Teres Minor:

Infraspinatus has been shown to be a stabiliser of the glenohumeral joint in flexion and abduction (Kronberg et al., 1990). In abduction, it pulls the humeral head posteriorly to press it into the glenoid cavity (Kronberg & Brostrom, 1995), thus resisting anterior translation as seen in the unstable shoulder. However a study in cadavers (Halder, Zobitz, Schultz, & An, 2000a), studied the mechanical properties of infraspinatus tendons and concluded that the high levels of stiffness within this tendon may explain the low incidence of posterior

shoulder dislocations. No other studies have detailed the role of infraspinatus in preventing shoulder dislocation by restraining anterior humeral translation. Thus role of infraspinatus in providing stability to the unstable shoulder remains inconclusive

Biceps Brachii

The role of biceps brachii in stabilising the shoulder has created some controversy over the last few years. Itoi et al (1994), demonstrated a minimal displacement of the humeral head when the biceps brachii was loaded, providing some evidence to support its role in dynamic stability of the glenohumeral joint. Other studies in cadavers (Itoi, Motzkin, Morrey, & An, 1999; Kumar, Satku, & Balasubramaniam, 1989; Rodosky, Harner, & Fu, 1994) also concur with this finding. However, Levy et al (2001) found no activity of the long head of biceps brachii during shoulder movement and postulated that the biceps brachii provides only passive stability to the glenohumeral joint via tension of the tendon.

Furthermore, other authors (Myers, Ju, Hwang, McMahon, & Lephart, 2004; Tibone, Fechter, & Kao, 1997) examined biceps latency in the stable and unstable shoulder and found a variety of results. Tibone et al (1997) stimulated the biceps tendon (among other structures) of stable and unstable shoulders of patients under anaesthesia, measured somatosensory cortical evoked potentials and found no difference between stable and unstable shoulders. The latency times reported (between 2.5 to 3.3 msec) were extremely short when compared to other studies of biceps reflex latency (Latimer, Tibone, Pink, Mohr, & Perry, 1998; Myers et al., 2004). While no other studies have been

undertaken measuring somatosensory cortical evoked potentials, such short latencies raise questions regarding possible methodological errors that may have been present in the study by Tibone et al (1997).

Myers et al (2004) tested shoulder muscle reflex latencies in subjects with stable and unstable shoulders. Subjects were positioned in sitting with the shoulder in abduction/external rotation, while the limb was perturbed into external rotation. Instability subjects demonstrated suppressed biceps brachii mean activation, and increased biceps brachii reflex latency (69 msec) when compared to those with stable shoulders (58 msec). The authors hypothesized that decreased level of muscle activation may contribute to the recurrent episodes of instability seen in this pathological population. Evidence of the increased latency in the biceps brachii muscle of the unstable shoulder provides support for decreased levels of active stiffness which may be present in the unstable shoulder, especially in the few milliseconds following an external perturbating force.

Pectoralis Major

Electromyography studies have also been undertaken to measure pectoralis major activity in the unstable shoulder. Wallace et al (1997), applied perturbations into external rotation in stable and unstable shoulders while measuring latency in pectoralis major. No significant difference was demonstrated with respect to onset times or peak voluntary or involuntary reflex muscle contraction, indicating no alterations in pectoralis major following instability episodes.

Myers et al (2004) also measured reflex latency of pectoralis major during external rotation perturbations in the apprehension position (as described above), and similarly found no difference in onset latency times in patients with instability, when compared with a control group with healthy, stable shoulders. However, suppressed pectoralis major activity was found in patients with unstable shoulders, when compared with the control group. The authors postulated that the suppressed activation of pectoralis major may contribute to episodes of recurrent instability. Based upon this finding, it may be postulated that decreased levels of activity of pectoralis major would result in decreased stiffness about the joint.

Contrasting results however, have been reported by Labriola et al (2005), who examined the effect of pectoralis major activity in the unstable shoulder in a position of apprehension, both via modelling and in cadavers. Increased pectoralis loading in cadavers was shown to increase compressive forces by 12% but increase anteriorly directed forces by 1180%. A similar increase in anterior directed forces was also shown during modelling. These authors concluded that increased active and passive pectoralis major forces result in decreased stability in the unstable shoulder at end of range abduction/external rotation. Thus some controversy continues regarding the influence of pectoralis major activity in the unstable shoulder. Given the high level of pectoralis major activity seen during horizontal flexion tasks, and that excessive forces into horizontal extension is a mechanism of shoulder dislocation, stiffness and strength of pectoralis major warrants further exploration.

Rotator Cuff Architecture:

Skeletal muscle architecture has been defined as the arrangement of muscle fibres relative to the axis of force production and has a role in providing stability to the joint (Lieber & Friden, 2000). The architectural arrangement of the rotator cuff muscles is consistent with the hypothesis that the rotator cuff muscles are responsible for stabilising the humeral head in the glenoid fossa (Ward et al., 2006). This architectural study is not necessarily in agreement with electromyography studies as will be discussed in the next section which is focused upon reflex activity of shoulder muscles.

2.3.4 Reflex latency in shoulder instability

Instability of the glenohumeral joint has been shown to result in alterations in reflex latencies in the surrounding musculature (Myers, 2001; Myers et al., 2004). It is postulated that these deficits arise because of decreased articular mechanoreceptor stimulation either from increased tissue length or deafferentation (Lephart & Henry, 1996; Tibone et al., 1997).

Myers et al (2004), tested reflexive muscle activity during external rotation perturbation in eleven subjects with anterior instability and gender/age matched controls. Subjects with instability displayed suppressed biceps brachii and pectoralis major muscle activation. Subscapularis, supraspinatus and infraspinatus muscles had increased peak activation in those with instability compared with controls. The instability subjects also showed significant suppression of the supraspinatus–subscapularis coactivation. These findings add to the notion that subjects with shoulder instability present with motor

programme alterations, particularly in those muscles that might resist motion into horizontal extension and external rotation.

This concept is also supported by Hundza & Zehr (2007), who studied EMG patterns in shoulder instability to investigate modulation of the cutaneous reflex and muscle activation in the unstable shoulder. Background EMG activity was found to be of a larger amplitude in the unstable shoulders compared with controls. Significant differences were also noted in the cutaneous reflexes between the unstable group when compared with the controls. The authors hypothesized that differing neural regulation in individual motor tasks may explain the variation between unstable and stable shoulders in each motor paradigm, and hence may require a more complex treatment plan.

2.3.5 Measures of function in the unstable shoulder

Patient reported questionnaires are a valuable method of gaining information regarding shoulder pain, function and quality of life. Numerous questionnaires are available (Constant, 1987; DASH, ; Kirkley, 1998; Richards, 1994; Rowe & Zairns, 1981; SST, ; Williams, Gangel, Arciero, Uhorchak, & Taylor, 1999), although relatively few of these have undergone extensive psychometric testing (Constant, 1987; Rowe & Zairns, 1981). In order for questionnaires to measure pain, function and quality of life accurately, they must be valid, reliable, have high levels of internal consistency and be responsive to clinical change (Kirkley & Griffin, 2003). Three questionnaires that have received notable attention are the Western Ontario Shoulder Instability Index (WOSI), American Shoulder and Elbow Surgeons Questionnaire (ASES) and the Single Alpha Numeric Evaluation (SANE)

The Western Ontario Shoulder Instability Index (WOSI) was constructed in the late 1990's by Kirkley et al (1998). The questionnaire of 21 questions comprised four equally weighted domains; ten questions of patient symptoms, four questions of function, four questions on the effect of instability on lifestyle and three questions examining the emotional effect of shoulder instability. These questions are all scored on a visual analogue scale (VAS) scale anchored at either end by the extremes of the item measured. The highest score possible is 2100 with a zero score indicating no shoulder related impairment upon quality of life. Validity was examined by comparison with other shoulder questionnaires, the Disabilities of the Arm, Shoulder and Hand (DASH), the Constant Score, ASES, the UCLA Shoulder Rating scale, the Rowe, and SF12. The highest Pearson product-moment correlation was with the DASH (0.7), and the lowest with the SF12 mental score (0.2). Reliability was examined over a two and three-month period and found to have an ICC of 0.949 and 0.911 respectively. The WOSI was found to be highly responsive to instability (0.931), when compared with other scales (Rowe= 0.791, DASH = 0.707, Constant=0.591 and ASES=0.535).

Another questionnaire that has received considerable interest is the self-report section of the American Shoulder and Elbow Surgeons Questionnaire (Richards et al, 1994). This comprises equally weighted sections of pain and function. The pain question examines the degree of pain experienced on a scale of 0 to 10. Function is examined by 10 questions pertaining to daily activities. Kocher et al (2005) examined this questionnaire and found it to be highly reliable (ICC=0.94), to have an acceptable level of internal consistency for instability (Cronbach alpha=0.61), and to have an acceptable floor and ceiling effect (0%

and 1.3% respectively). Acceptable criterion validity, construct validity and responsiveness to change in instability patients (standardized response mean=0.93) were also demonstrated. Michener et al (2002) also examined the ASES and found good test-retest reliability (ICC=0.84, CI lower limit 0.75) and internal consistency (Cronbach alpha=0.86). The standard error of the measure was 6.7 (90%CI, 11.0), construct and discriminant validity were demonstrated as was responsiveness (standardised response mean=1.5 and an effect size=1.4).

The Single Alpha Numeric Evaluation (SANE) has also been used previously as a measure of function following shoulder surgery. It has been shown to correlate well with ASES and the ROWE score (Williams et al., 1999). Subjects were asked to rate their shoulder as a percentage of normal (0%-100% scale with 100% being normal). In the present study, the SANE was used to assess levels of perceived instability. A 10cm scale was anchored at zero by 'very stable' and at ten by 'very unstable' to give a global measure of perceived instability.

2.3.6 Strength and Shoulder Instability

The use of a maximum voluntary contraction (MVC) to measure strength provides a similar reference point for both shoulders in studies of unilateral shoulder instability. However, the use of MVC remains controversial. In order to produce forces at MVC level, subjects need to be motivated, familiar with exercise and free from any pain-avoidance behaviour (Crombez, Vlaeyen, Heuts, & Lysens, 1999). Additionally the MVC can vary due to the level of verbal encouragement that the patient receives (McNair, Depledge, Brettkelly, & Stanley, 1996). It is also possible, that subjects do not fully engage in the task

because of ‘fear avoidance’ due to either pain or instability, despite consistent verbal encouragement for all subjects. The possibility of increased error due to apprehension or fear avoidance remains a consideration when testing pathological populations.

Decreased strength has been demonstrated in other peripheral joints with instability (Kaminski & Hartsell, 2002; Van der Esch, Steultjens, Knol, Dinant, & Dekker, 2006). Findings from studies of shoulder muscles however, are less conclusive. Warner et al (1990), studied isokinetic strength in subjects with impingement and subjects with instability, and compared the results with healthy subjects. A significant difference was found in the peak torque of the dominant arm in subjects with instability and impingement when compared with those with asymptomatic shoulders. There was also a trend towards greater strength in the dominant shoulder, although this result was not significant.

Tsai et al (1991), examined isokinetic strength in shoulders with anterior instability. Twenty-six patients were tested an average of seven years following initial dislocation. The unstable shoulder demonstrated significantly decreased peak torque during abduction and internal rotation. The severity of the impairment (Rowe score, decreased range of motion and peak torque) was not related to either the number of dislocations sustained, or the duration of the instability.

Brostrom et al (1992), measured strength and outcomes in 33 shoulders with recurrent anterior and multi-directional instability. Muscle strength was measured with an isokinetic pulley device. Initial readings showed strength

deficits in both external and internal rotators. Subjects performed a training programme three times per week for eight weeks as well as home theraband exercises. After treatment, seven of the 33 patients 'became stable', 21 shoulders improved and five did not improve. No definition was provided by the authors on 'becoming stable'. There was a mean increase in external rotation (mean=11.8N (SD=10.1)), and internal rotation (mean=13.7N (SD= 13.2)) strength. Subjects who had abnormal skeletal anatomy (i.e. humeral head retroversion) or multi-directional instability did not show as large an improvement as those with anterior instability and normal skeletal anatomy.

Rupp et al (1995) tested isokinetic profiles of swimmers, 50% of whom tested positive for anterior instability. When compared with a control group matched for age, sex and dominance, a significantly lower external rotation/internal rotation ratio of peak torque and total work was seen in the swimmers at both 60 º/sec and 180 º/sec. Bak et al (1997), also studied isokinetic strength in a sample of swimmers with symptomatic instability and impingement. In contrast to Rupp et al (1995), a decrease in internal rotation torque was noted when compared to the asymptomatic side and a control group. The functional strength ratio (eccentric external rotation : concentric internal rotation) was also significantly increased in the painful shoulder, indicating an alteration in the levels of co-contraction of the rotator cuff musculature. The presence of pain in these subjects may have been a confounding factor in this study.

Pain has been shown to decrease muscle strength in the shoulder (Ben-Ishay, Zuckerman, Gallagher, & Cuomo, 1994; Forthomme et al., 2002). Ben-Ishay et al (1994), found significant increases in abduction power (mean 82%), peak

torque (mean 48%) and work (mean 90%) ($p<0.05$) following the injection of Lidocaine and Marcaine. In a more recent study, Forthomme et al (2002), also noted an increase in peak torque following the administration of anaesthesia, although this effect was not shown to be statistically significant. These authors suggested that the small sample size in this study may have prevented results reaching a statistically significant level.

More recently Dauty et al (2007), examined isokinetic strength profiles in 25 subjects (23 +/- 6 yrs) with recurrent unidirectional anterior instability, one month before, and three months after a Latarjet stabilisation. Prior to surgery, rotator peak torques were similar between the stable and unstable sides. The one exception however was the concentric external rotation/internal rotation ratio which was higher on the unaffected side (50% +/- 9 vs. 44% +/- 8 at 60 º/sec, and 48+/- 8 vs. 43% +/-10 at 120 º/sec, $p<0.05$) as the external rotators were slightly weaker on the unstable side.

Some studies of strength in shoulder instability have been less conclusive. Falla et al (2003), examined internal rotator strength in 18 baseball players with instability, confirmed by a positive anterior drawer test. Subjects were positioned in prone on a plinth with the arm abducted to 90 degrees and resisted internal rotation measured by a strain gauge. No significant difference was found between subjects with instability and control subjects. The positioning of subjects may have contributed to this result, as the scapula was not fixated in this prone position.

No assessments of horizontal flexion strength in unstable shoulders were found, despite the importance of horizontal flexors in providing resistance to external forces which may dislocate the shoulder. Some researchers (Flocks, 1995; Silva et al., 2006) have studied horizontal flexion in the stable shoulder. A study of elite tennis players with no shoulder pathology has reported increased horizontal flexion strength in the dominant arm (Silva et al., 2006), while male normative horizontal flexion values have been reported to be 42.97 (aged 18-21) and 50.92 (aged 22-35 years) in the dominant arm (Flocks, 1995).

2.3.6.1 *Strength and Function.*

Few studies have been undertaken investigating the relationship between strength and function in the unstable shoulder. Tsai et al (1991), measured both function and strength but no mention was made of a correlation between function and strength. Furthermore, function was not influenced by the number of dislocations or the duration of the instability.

Other authors (Cools, Witvrouw, Declercq, Vanderstraeten, & Cambier, 2004; Gladstone, Bishop, Lo, & Flatow, 2007; MacDermid, Ramos, Drosdowech, Faber, & Patterson, 2004) have found a correlation between strength and function following rotator cuff tears. However, no mention was made regarding the level of instability in these subjects.

2.3.7. Summary

Shoulder instability results in several pathological changes in the shoulder joint, as well as the surrounding musculature. These changes include bony glenoid defects, labral tears, decreased cross sectional area of subscapularis,

increased latency in biceps brachii, and suppressed pectoralis major activity. These pathological changes may result in decreased protection to external perturbating forces, thus predisposing the joint to further injury. The passive and active tissue resistance to external forces is known as stiffness and will be further discussed in the following section.

2.4 Stiffness

2.4.1 Introduction

Some authors (Myers & Oyama, 2008; Riemann & Lephart, 2002) have proposed that intrinsic stiffness may be reduced in those with unstable shoulders, thus predisposing them to further episodes of instability. Stiffness has been defined as the rate of change in torque to the rate of change in angular rotation (Gottlieb & Agarwal, 1978), or the rate of change in force to the rate of change in length, and is the reciprocal of compliance (McNair et al., 1992). Stiffer joints are more resistant to external force and the capsuloligamentous tissues are less likely to be injured when exposed to external forces (Borsa, Dover, Wilk, & Reinold, 2006; Borsa, Sauers, & Herling, 2002; McNair et al., 1992). Intrinsic stiffness has been defined as the level of stiffness prior to reflex activity, and is the first line of defence when the joint is exposed to an external perturbating force, providing an immediate response before the reflex activity is initiated (Myers, 2001).

2.4.2 Physiological factors influencing stiffness

2.4.2.1 Studies in isolated muscle

Early work (Hill, 1938; Wilkie, 1956) examining muscle stiffness, focused upon isolated muscle. Wilkie (1956) described a technique to examine muscle

stiffness called the ‘quick release’ technique. It was based upon a model of muscle advanced by Hill (1938), that described series elastic and contractile elements of muscle. Experimentally, muscle is held at a fixed position, stimulated by an electric current, and then the end point is suddenly released. Force/time and stress/strain profiles of muscle illustrated the stiffness of both contractile and series elastic tissue. This methodology has since been adopted for use in vivo, although its use is not without a number of assumptions related to muscle shortening, and the inertia of limbs in the period immediately after release. For these reasons, this technique has not been performed widely in in vivo research.

Hill (1968), examined resting muscle to further examine elements of muscle that may influence stiffness. A small elastic effect was noted at the beginning of stretch of a resting muscle. This was referred to as the ‘short range elastic component’ (SREC). It was hypothesized that the cross bridges on the myosin filaments were cross linked with actin filaments, providing ‘flexural rigidity’. The elastic behaviour of the resting muscle was thought to be ‘short range’ as the cross bridges could only stretch a small distance before the attachments ‘broke’. It was further proposed that the frictional resistance between the sliding filaments of the muscle was independent of velocity and due to the SREC.

Thereafter, Joyce and co-workers (1969), described a technique to measure the increase in contractile component muscle stiffness immediately following a short stretch. Joyce et al (1969) noted that the tension developed during lengthening or shortening was modified by changes in length that had preceded those situations. Rack and Westbury (1972), further proposed that short range

stiffness reflected the combined stiffness of linkages between thick and thin myofibrils. It was noted that these linkages could not be stretched indefinitely, and after a short displacement, separated and then reformed. The stiffness of the first part of the motion was independent of velocity, provided that the movement was not too slow, indicating the presence of an elastic phenomenon.

Further work by Rack and Westbury (1974) indicated that constant velocity movement of small amplitude resulted in a steep rise in tension during lengthening. Longer movements resulted in decreased resistance in the latter stages as the tension change became more gradual. The explanation for the observed increase in stiffness was based upon the sliding filament theory of Hansen and Huxley (1955), and Huxley (1957), together with Hill's (1938) concept, that the stiffness of the muscle fibres was proportional to the number of cross bridges formed between actin and myosin, and the stiffness of the individual cross bridges. The movement of the muscle to the yield point was thought to be three to four percent of the physiological range (Rack & Westbury, 1974).

Flitney and Hirst (1974; 1978), suggested that the yield point was related to the backward rotation of the myosin head. A continued stretch of muscle resulted in yielding or breakdown of the cross bridges and a consequent decrease in force. Recovery of force occurred as cross bridges were reformed, and a new equilibrium force level was established which was appropriate to the new muscle length. The degree of extension required to induce yielding of areflexic muscle was shown to represent the maximum range of sliding movement that a

cross bridge between the actin and myosin could accommodate, before it was forcibly detached (Flitney & Hirst, 1978).

Externally applied stretch is distributed between muscle and tendon fibres according to their respective stiffness, as these two components lie in series. One method of delineating stiffness of the tendon from that of the contractile component was developed by Morgan (1977) and involved the measurement of the short range stiffness at a number of different muscle tension levels, and the construction of an alpha diagram. Alpha was the ratio of the isometric tension level and the musculotendinous stiffness and was plotted against isometric tension levels. A straight line characterised this plot, with a positive slope over the various tension levels. The intercept on the ordinate, alpha (zero) was described as “the amount of shortening required to reduce the tension to zero if the short-range stiffness continued to act” and represented the stiffness of the tendon fibres. Muscle compliance was represented as the slope of the straight line and was assumed to be constant across isometric tension levels. The results of Morgan’s work indicated that cat soleus muscle fibre stiffness was a linear function of load, and was independent of muscle length and stimulus rate. The stiffness of the muscle was therefore proportional to the number of active cross bridges, a finding also supporting Hill’s (1938) model.

Work by Morgan (1977) and Walmsley and Proske (1981) suggested that tendon stiffness was relatively constant. However Rack and Westbury (1984) provided contrasting evidence concerning this supposition. These researchers stimulated the motor nerve to generate an isometric contraction while subjecting the muscle and tendon to sinusoidal stretching. This enabled a new method of

measuring entire tendinous components of muscle, called the ‘null point’ method (Rack & Westbury, 1984). The method was based upon the knowledge that muscle spindles were very sensitive indicators of changes in muscle fibre length (Matthews & Stein, 1969), and hence could be used to detect movement in muscle fibres when the musculotendinous unit was sinusoidally stretched at different isometric tension levels. If no signals were observed from the muscle spindles during the sinusoidal stretches, thus signifying the null point, then movement associated with the stretch was assumed to be occurring in tendinous structures of the muscle. Using this method, tendon stiffness was shown to be greater than contractile tissue stiffness at low levels of muscle activation. However, as muscle activation increased, stiffness of the contractile elements approached that of the tendon.

In regard to the null point method, Rack and Westbury (1984) and Proske and Morgan (1987) stressed the possibility that fusimotor stimulation from beta motor neurons may unload any passive tension in the muscle spindle, and thereby provide erroneous evidence that the muscle fibres were at a null point. Proske and Morgan (1987) argued that this would cause stiffness values, above about 25-30 percent of maximum isometric tension, to be overly high. It therefore seemed likely, that above these tension levels, tendon compliance would be invariant.

2.4.3 Relative stiffness in tendon compared with muscle

Tendinous structures have been shown to have greater stiffness than contractile components at low levels of muscle activity (Rack & Westbury, 1984). As muscle activation levels increase, it has been shown that the stiffness of the

contractile component increases and muscle at maximal levels of contraction becomes at least as stiff as tendon. The implications of this finding are that at low levels of muscle activation the tendon is stiffer relative to the muscle. At higher levels of muscular contraction, the muscle has increased stiffness relative to the tendon. Additionally, as muscle fibres have been shown to have a viscous, as well as an elastic component, the movement of the muscle fibre will lag behind the external movement (Rack & Westbury, 1984).

Maganaris and Paul (1999) utilized ultrasound to examine the mechanical properties of muscle and tendinous tissue. Based upon previously developed methodology (Fukashiro, Itoh, Ichinose, Kawakami, & Fukunaga, 1995; Fukunaga et al., 1996), Maganaris and Paul (1999) determined the intersection between muscle and tendinous tissue and observed movement of this intersection during an active muscle contraction. It was proposed that muscle performance during maximal isometric and dynamic contraction was influenced by the stiffness of the tendon. These authors concluded that in agreement with isolated tendon studies, tendon force and stress increase curvilinearly as a function of displacement and strain.

It was further proposed by Maganaris (2002) that the primary role of the tendon is to transmit force to the skeleton, in order to generate joint movement. The gastrocnemius tendons of six males were examined using ultrasound during tendon loading and unloading from muscle contraction and relaxation. The tendon insertion returned to its original point in the unloaded condition with greater displacements than in the loading condition, indicating the presence of hysteresis properties in the tendon. Further examination of tendon force-

elongation data indicated that passive recoil of the tendon contributed to the overall mechanical work of the muscle-tendon complex. The elastic work of the gastrocnemius tendon during walking was approximately six percent of the total external mechanical work produced. It was further proposed that with more active exercises, such as running, the relative contributions of passive tendon recoil would increase.

Bojsen-Moller et al (2005) also studied the vastus lateralis tendons of sixteen trained men to further examine the relationship between the mechanical properties of the tendon and contractile muscle output during high force levels. Rate of torque development (RTD) was found to be positively related to stiffness of tendon, with the stiffness of tendinous structures accounting for up to 30% of RTD. The authors surmised that stiffer tendinous tissue results in more effective force transmission from the contractile elements to the bone.

Muraoka et al (2005) utilized ultrasound to study the elastic properties of the Achilles tendon and found it to be related to muscle strength of gastrocnemius and soleus. Subjects with greater muscle strength were found to have stiffer tendons and therefore were able to deliver greater force from the muscle more efficiently.

2.4.4 In vivo methodology in assessment of stiffness

Diagnostic ultrasound has been used to measure tissue stiffness. Fukashiro et al (1995) developed the method of measuring tendon-aponeurosis stiffness using B-mode ultrasonography. This technique allows non-invasive measurements of the tendon in vivo and can be used in superficially located

muscle-tendon units. The insertion point of the muscle fascicle into the aponeurosis is clearly visible under ultrasonography and movement of this point (change in length) relative to isometric contraction (change in force), allows for calculations in stiffness.

Maganaris and Paul (1999) further utilized this technique and noted that true resting length of the tendon was difficult to measure in vivo and was therefore defined as the situation in which the net torque about the joint is zero. A further limitation was that the stiffness measurement is dependent not only upon the tensile force added, but also the length of the in-series contractile structures. An increase in the number of sarcomeres in series increases the absolute shortening of the entire muscle and the lengthening of the tendon during an isometric contraction. Incorporating the ratio of tendon length : muscle fascicle length into stiffness calculations is one way of accounting for this limitation. Real-time ultrasonography has been previously used to measure superficial lower limb tendons such as the Achilles tendon (Muraoka et al., 2005; Urlando & Hawkins, 2007) and the vastus lateralis tendon–aponeurosis complex (Bojsen-Moller et al., 2005). Assessment of shoulder tendon stiffness however, would be technically more difficult due to the complex anatomical arrangement of rotator cuff tendons about the shoulder. Additionally, as rotator cuff tendons co-contract to provide joint stability, examination of a single tendon may not reflect total joint stiffness, and therefore may not be functionally important.

Another technique in measuring stiffness is the loaded movement technique developed by Goubel et al (1971) and is similar to the quick release method used for isolated muscle in vitro. This technique relies upon the silent period in

EMG activity of agonist and antagonist muscles. The silent period at the end of movement was suggested to be the period of time that only the series elastic component was involved in the force/time record, and hence compliance could be calculated. As in the quick release method, this method relies upon knowledge of muscle length and the moment of inertia of the limb. The researchers made no comment whether the possible residual tension associated without any electromechanical delay from the time of EMG cessation was accounted for.

Several authors (Cnockaert, Pertuzon, Goubel, & Lestienne, 1978; Pousson, Van Hoecke, & Goubel, 1990) have made other adaptations to the original controlled and quick release methods of Hill (1938) and Wilkie (1956) to test muscle elasticity in vivo. Subjects perform an isometric muscle action against resistance, positioned at a known distance from the axis of rotation of the joint. When the muscle action is at a specified load, the resistance is removed causing the limb to accelerate and the muscle shortens. There is an initial increase in angular acceleration before the limb then decelerates. The series elastic component is calculated from the beginning of the deceleration to the onset of EMG activity in the antagonistic muscle group. This method requires knowledge of the moment of inertia of the limb, against which the limb is acting during the release. Pousson et al (1990) used this quick release method to examine muscle compliance in the upper limb. The relationship between muscle compliance and force of the biceps brachii was best described as a power function of force ($Y=aX^b$).

Total stiffness of a limb has also been examined. Cavagna (1970) determined the stiffness of the lower leg when landing from a jump with knees held in extension. These analyses were based upon oscillation theory, which states that a single degree of freedom mass-spring system will oscillate at its resonant (natural) frequency when perturbed from its equilibrium position by a transient force. This frequency is a function of the stiffness of the spring and the magnitude of the attached mass. If a damping component is added to this system, the resulting oscillations will decay at an exponential rate, which is governed by the amount of damping present. The lower limb could thus be modelled as such a system, in that the muscle has viscoelastic properties. The stiffness of the lower limb could then be calculated from knowledge of the damped frequency of oscillation and the coefficient of damping. The equation is usually written as:

$$K=4\pi^2 mf^2 + c^2/4m,$$

where k is the stiffness (N/m), m is the mass, f is the damped frequency of oscillation and c is the coefficient of damping.

The theoretical basis of this technique has also been used to measure total stiffness in the upper limb. Wilson and co-workers (1991) used a damped oscillation technique while subjects performed a bench press exercise. In this method, the bar was perturbated as the subjects maintained an isometric position at a prescribed upper limb angle. One problem with the technique is that a notable percentage of subjects do not oscillate the limb when perturbed, invoking a voluntary action to stop movement (McNair et al, 1992).

While assessment of total limb stiffness provides some indication of regulatory patterns of the CNS, it remains a generalised measure of joint stiffness. Other methodology has been developed to measure the stiffness of individual muscle groups. McNair et al (1992) utilized the damped oscillation technique at the knee joint to measure stiffness characteristics of the hamstring muscles and reported a non-linear relationship between stiffness and muscle load, best represented by a second order polynomial. This technique enables the measurement of stiffness in a single joint. However, it would be technically very difficult to apply to the shoulder. Weights would need to be fixed to the upper arm, and their mass and inertia, in addition to that of the limb, would need to be calculated. Furthermore, the technique requires that muscle activation is relatively constant throughout the time of perturbation. Given the number of muscles that may influence shoulder motion, it would be difficult to monitor their activation levels, and hence be sure of attaining a constant activation level.

Another technique which has been utilized to measure stiffness is applied vibration. Hunter and Kearney (1982), measured stiffness of ankle plantar and dorsi flexors, and reported a linear increase in stiffness with increasing isometric contraction of these muscles. Weiss et al (1988) examined the same muscle group using this technique and also reported a linear relationship between muscle stiffness and load. Adapting this technique to the shoulder however, would be complicated as it would be difficult to isolate one muscle in the shoulder to be vibrated.

While some studies (Hunter & Kearney, 1982; Weiss et al., 1988) have reported a linear relationship between a single muscle and single joint stiffness, there

has been relatively little work examining the effect of multiple muscle activation upon multiple-joint stiffness. Osu et al (1999), studied the regulation of multiple-joint stiffness by measuring human arm stiffness and electromyography (EMG) signals. It was assumed that the EMG reflected corresponding muscle stiffness, while joint stiffness was predicted from the EMG using a two-link six-muscle arm model and a constrained least squares regression method. A strong correlation was seen between effective muscle stiffness and joint stiffness when the muscles were acting as agonists (i.e. joint torque is positive for flexor muscles and negative for extensor muscles). During co-contraction, joint stiffness increased in response to a linear increase in muscle stiffness.

Another technique for measuring stiffness utilises a single perturbation of a joint, while changes in torque and angle are recorded. Ma and Zhalek (1985), applied very rapid, small amplitude, perturbations to the forearm to flex the elbow, while subjects held an isometric contraction of the elbow extensors. After accounting for inertial and viscoelastic effects, the time course of muscle moment produced by the perturbations was examined, together with EMG data. It was concluded that the initial phase of the muscular response (less than 50 msec) was due to intrinsic stiffness. All responses after 50 msec were thought to be due to reflex mediated mechanisms.

Olmstead and others (1986), also used a single perturbation to examine the relationship between stiffness and stability during valgus and varus perturbations to the knee joint. Subjects were positioned with the knee in slight flexion and EMG recordings of hamstrings and quadriceps taken during extension and flexion contractions while a valgus and varus perturbation was

applied to the lower leg. Stiffness was seen to increase with increased levels of resistive torque. It was concluded that the knee extensors were better at decreasing the varus moment created when a force is applied, while the knee flexors act as knee stabilisers.

Sinkjaer et al (1988) applied single perturbations at the ankle joint and observed an increase in stiffness at different levels of resistive torque as a function of the level of contraction. This effect has also been demonstrated in the wrist. Sinkjaer and Hayashi (1989) noted an decrease in wrist displacement with increased wrist joint stiffness. As seen in other studies (Hoffer & Andreassen, 1981; Nichols & Houk, 1973), the stretch reflex was found to increase joint stiffness, and once the reflex component was eliminated the stiffness of the joint was compromised.

Active stiffness has also been calculated by measuring change in torque and angle, during high-velocity, small amplitude perturbations on an isokinetic dynamometer (McHugh & Hogan, 2004). This method is based upon the work of Morgan (1977) and enables differentiation between stiffness in tendinous and muscular structures. Stiffness values at incremental levels of MVC can be plotted and a regression line drawn between data points. The slope of the regression line is said to represent the contractile tissue element while the y-intercept represents the contribution of tendon stiffness (McHugh & Hogan, 2004).

The current study builds upon previous studies (McHugh & Hogan, 2004; Sinkjaer et al., 1988) and uses perturbation to examine stiffness in the horizontal plane in the stable and unstable shoulders of males' with unilateral

shoulder dislocation. One of the benefits of this methodology is control of the shoulder in one plane of motion. Many of the previously discussed methodologies have been undertaken in peripheral joints such as the ankle or knee which predominantly operate in a single plane of motion. Perturbation in a single plane at the shoulder allows analysis of stiffness in this plane without confounding movements in a three dimensional manner. This high velocity perturbation technique also allows examination of intrinsic and extrinsic stiffness as EMG recordings demonstrate the onset of reflex activity. Additional advantages of this methodology were the high levels of reliability, and ease of use in safely applying to a pathological population.

2.4.5 Anatomical structures affecting stiffness

A variety of methodologies have been utilized to show that tissue stiffness is dependent upon muscle fibre type, as well as the degree of collagen or titin within the muscle. Initial studies investigating the influence of muscle fibre type of tissue stiffness examined the short-range stiffness in the semi-tendinosus of the Australian blue-tongued lizard (Proske & Rack, 1976). Skeletal muscles of reptiles consist of easily distinguishable slow and fast twitch fibre types. Slow twitch fibres were found to resist extension with greater stiffness than the fast twitch fibres over a variety of differing velocities. Kovanen et al (1980), proposed that such differences in stiffness in fibre type are due to different percentages of collagen. These researchers studied slow and fast twitch fibres in the rat. The muscle fibres of endurance and untrained rats were stained and histochemical analysis undertaken. These showed significantly more collagen in slow twitch muscle than in the fast twitch muscle. It is thought that the cross linkages between the collagen fibrils increase the tensile strength and stiffness

of the system, and that the cross linking between collagen molecules occurs during maturation (Danielsen & Gottrup, 1981). The stiffness of the collagen may also depend upon the state of the collagen, the type and orientation of the collagen fibres, the level of elastin in the collagen, and the bonds with the extra-cellular matrix (Ducomps et al., 2003).

Later work by Kovanen et al (1984), investigated the effect of exercise upon collagen concentration and stiffness. Rats were trained on a treadmill five days a week, for four weeks. An increase in collagen was found in the perimysium and endomysium of the slow twitch muscle compared to fast twitch muscle. A significant relationship was observed between the level of collagen and stiffness in the slow twitch fibres when compared with the fast twitch fibres. Additional lathryogen treatment to one group of rats was applied to induce fragility in the collagenous tissue. The lathryogen group demonstrated decreased ultimate tensile strength and provided evidence that the amount of collagen was of great importance in influencing the stiffness of the tissue.

More recently, DuComps and others (2003) undertook an experiment where rabbits jumped over a barrier for food and water. The bar was raised incrementally during the experiment. After 150 days, there was an increase in collagen concentration in fast twitch pennate and bipennate muscles compared with the sedentary controls. Stiffness and stress were also seen to increase and both parameters were significantly correlated with the presence of collagen concentration (Ducomps et al., 2003).

Other studies in humans (MacDougall, Sale, Alway, & Sutton, 1984; Pousson et al., 1990) show an increase in collagen concentration and tensile strength as a result of eccentric training. MacDougall et al (1984), examined the biceps brachii of elite and intermediate bodybuilders and found increased collagen and other connective tissue when compared to a control group who had no history of resistance training. Pousson et al (1990), demonstrated that eccentric exercise altered the elastic characteristics of human muscle. In this study, ten sedentary males either completed an eccentric exercise protocol or were sedentary controls. Compliance of the elbow flexors was measured before and after the six-week training period, using a quick release methodology. The training group demonstrated decreased compliance (and thus increased stiffness), compared to the sedentary group.

Strength training has also been shown to affect the viscoelastic properties in the elderly. Reeves et al (2003) examined the tensile stiffness of the patellar tendon in the elderly population. Fourteen elderly patients exercised isotonically three times per week to load the patellar tendon. Subjects were tested at baseline and again after 14 weeks of training. The stiffness of the patellar tendon was found to be significantly increased by 64% and hysteresis (an indication of tissue viscosity) decreased by 28%. The authors noted an increased rate of force development occurred as a result of this increased stiffness, leading to increased efficiency of force production. It was surmised that this may have been due to increased packing density and diameter of the collagen fibrils, as well as alteration in their crimp formation (Reeves et al., 2003).

Some studies (Kiiskinen, 1977; Woo et al., 1981) however, have found no change in tensile strength or stiffness after training. Kiiskinen (1977), studied immature mice and noted no change in tensile strength, despite increased dry weight of Achilles tendon following 5-7 weeks of physical training on a treadmill. Woo et al (1981), also noted no change in collagen concentration after training pigs aerobically on a treadmill for 12 months. Training was seen to increase the strength of the tendon insertional site, but have minimal effect upon tendon substance. It has been hypothesized that training in the immature animal results in increased collagen turnover and fewer cross linkages in immature collagen cells. It may also be that increased stiffness seen in previous studies is due not only to collagen concentration, but also to increased proteoglycan concentration (Magnusson, Hansen, & Kjaer, 2003).

Earlier work from Purslow and Trotter (1994) indicated that collagen was responsible for creating stiffness in the outer range of motion. In this lengthened position, collagen fibrils were seen to align longitudinally, and thus transmit force through the collagen fibres. In the shortened position, the collagen fibrils are aligned with a slight circumferential bias. Therefore it has been proposed that other structures such as titin, may be responsible for providing passive stiffness in the shortened position.

Titin is a large polypeptide that spans the distance between the Z-disc and the M-line, attaching the myosin filament to the Z-line (Granzier & Labeit, 2006). The three major roles of titin in skeletal muscle are; (1) keeping myosin filaments centred in the sarcomere for activation, (2) functioning as a molecular spring responsible for the development of a retractive force upon stretch of a

non-activated muscle and (3), providing a structural framework for other sarcomere proteins (Minajeva, Neagoe, Kulke, & Linke, 2002). Several authors (Granzier & Labeit, 2006; Herzog, Schachar, & Leonard, 2003; Hoang, Herbert, & Gandevia, 2007; Labeit, Kolmerer, & Linke, 1997; Minajeva et al., 2002; Neagoe, Opitz, Makarenko, & Linke, 2003) have investigated the contribution of titin to passive stiffness. Furthermore, different isoforms of titin are known to exist, due to the variation in length of the Ig band in the titin molecule. These isoforms vary according to fibre type (Kellermayer & Granzier, 1996).

Slow twitch muscles have been shown to have uniform titin isoforms which are greater in length, while the titin isoforms in fast twitch muscle fibres are of variable length (Agarkova, Ehler, Lange, Schoenauer, & Perriard, 2003; Prado et al., 2005). Thus the sarcomere in fast muscle fibres exhibits a perfect alignment of actin and myosin, with thin Z-discs, and either a short or long titin isoform. In contrast, the sarcomere in slow twitch muscle fibres exhibit less ordered arrangement of contractile structures; the length of actin filaments is more variable, the M-bridges more elastic and the extensible portion of titin more compliant. These fibre type changes in titin allow increased structural stability during continuously contracting slow twitch muscle, while the arrangement of titin in fast twitch fibres optimizes powerful contractions (Agarkova et al., 2003). Several authors have proposed that the increased passive tension seen in slow twitch muscle is due to other intra-sarcomeric proteins such as desmin (Chopard, Pons, & Marini, 2001), dystrophin (Ho-Kim & Rogers, 1992) and other components of the dystrophin-glycoprotein complex (Chopard, Pons, Charpiot, & Marini, 2000).

A recent study by Prado et al (2005) utilized rabbits to illustrate the relative contribution of titin and collagen in slow and fast twitch muscle. An increase in the percentage of type I fibres corresponded with an increase in total passive tension and an increase in the contribution of extramyofibrillar structures. The contribution of titin to passive stiffness was shown to decrease as the percentage of type I fibres increases. Accordingly, an increase in type II fibres corresponds with an increase in the reliance upon titin, and a decrease in the contribution of extramyofibrillar structures, in providing passive tension within the muscle. The relative contribution of collagen and titin, as well as other intra-sarcomeric proteins such as desmin, dystrophin and obscurin, to total passive tension of the muscle is worthy of further exploration.

2.4.6 Stiffness and muscle architecture

Skeletal muscle architecture is one of the most important parameters for predicting muscle functional properties and has been defined as the arrangement of muscle fibres relative to axis of force production (Lieber & Friden, 2000). Loren et al (1996) examined the relationship between muscle architecture and tendon compliance and concluded that strain during muscle contraction is dependent upon joint angle, muscle and joint range of motion, force variation and tendon strain of the muscle-tendon unit, and that each parameter contributes uniquely to its design.

Sarcomere length is also known to influence the biomechanical properties of muscle. Walmsley & Proske (1981), investigated the effect of the number of sarcomeres on muscle stiffness, and found an increase in compliance with an increase in number of sarcomeres. It was stated that the number of sarcomeres

in length, accounts for the active component of short-range stiffness entirely (Walmsley & Proske, 1981). Ljung et al (1999), also examined the effect of sarcomere length upon stiffness, and studied several samples of muscle tissue along the flexor carpi ulnaris (FCU) and pronator teres (PT) in cadavers. The FCU was chosen because of its relatively simple architectural structure where the fibres run parallel along the muscle length. Pronator teres was chosen because of its complex architecture which is thought to result from multiple axes of motion in elbow flexion and forearm pronation. No difference in sarcomere length was found along FCU. A significant difference was found however in sarcomere length both between PT and FCU, and within PT. The authors surmised that muscle has an ability to regulate sarcomere number in response to various length changes, to establish a certain sarcomere length.

Friden and Liber (2003) further examined the relationship between stiffness and sarcomere length. Fibres were taken from subjects undergoing flexion contracture release secondary to cerebral palsy, and were compared with fibres sampled from normal subjects. Fibres taken from patients with cerebral palsy were shown to develop passive tension at significantly shorter sarcomere length (1.84+/- 0.05μm) when compared with normal subjects (2.20 +/- 0.04μm). The elastic modulus of the stress-strain relationship in patients with cerebral palsy (55.00 +/- 6.61kPa) was almost double that of normal patients (28.25+/- 3.31kPa). The authors hypothesized that structural changes to muscle components such as titin and collagen may be responsible for the alterations in sarcomere length and elastic modulus.

2.4.7 Stiffness and co-contraction

Joint stiffness can also be altered by the degree of muscle activation about the joint. Activation of the agonist muscle results in increased torque through the joint, as the joint moves through its range of motion. Co-contraction of the agonist and antagonist muscle results in no net torque (as the joint remains stationary), but does result in an increase in joint stiffness as the muscles on both sides of the joint work to fixate the joint. Akazawa et al (1983) examined stiffness with co-contraction at a constant force across the first metacarpo-phalangeal joint. Ten male thumbs were fixed so that the distal joint could be flexed, thus isolating flexor pollicis longus. Reflex responsiveness and stretch evoked stiffness were shown to increase linearly with increasing co-contraction (Akazawa et al., 1983). Stiffness can also be augmented by the activity of synergists (e.g. scapular stabilisers), and is dependent upon the background torque (Hoffer & Andreassen, 1981).

Louie and Mote (1987) measured the ability of quadriceps–hamstring co-contraction to reduce knee laxity. Subjects voluntarily contracted specific muscle combinations while the foot was placed in different amounts of internal and external rotation. A strain gauge measured the applied vertical force and torsion on the foot, while a potentiometer measured knee joint rotation, to enable a measure of stiffness. Increased activation levels of the musculature resulted in increased joint stiffness and decreased knee joint laxity. Nielsen et al (1994), demonstrated the effect of co-contraction at the ankle joint. Stiffness in seven male subjects was assessed using the increment in torque following the stretch of the plantar flexors divided by the stretch amplitude. All seven subjects showed increased stiffness and joint stability with co-contraction of plantar

flexors and tibialis anterior, when compared to contraction of plantar flexors alone.

2.4.8 Extrinsic mechanisms that mediate stiffness

Sinkjaer and Hayashi (1989), examined the effect of stretch reflex activation upon wrist displacement following a perturbation. Subjects' hands were perturbated into extension before and after ischaemic compression to minimise the effect of the stretch-induce reflex response. Stiffness decreased and the limb deflected further when the stretch reflex response was minimal, indicating that the reflex response played a role in increasing joint stiffness and thus joint stability. Some researchers however, have postulated that injury occurs faster than this reflex response (Latimer et al., 1998; McNair et al., 1992; Pope, Johnson, Brown, & Tighe, 1979; Sinkjaer & Hayashi, 1989; Sinkjaer et al., 1988).

Johansson et al (1991), provided evidence to support the role of joint receptors in mediating muscle stiffness at a joint via the gamma-muscle-spindle system. It was hypothesized that since the gamma muscle spindle system participates in the regular contribution of muscle stiffness, this system may also be involved in the preparatory setting of stiffness characteristics about the joint, and therefore influence joint control and functional joint stability (Johannson et al., 1991). Intrinsic muscle stiffness is largely due to the existing actin-myosin bonds or the degree of muscle contraction at any given point in time. Thus intrinsic muscle stiffness is partly the result of preceding reflex mediated stiffness. Therefore if joint afferents affect the regulation of reflex-mediated stiffness, they will also contribute to the levels of intrinsic stiffness, which is responsible to maintenance

of joint stability against perturbation. Because reflex responses are thought to be too slow to protect the joint from fast perturbations, it is hypothesized that the primary function of the joint afferents is continuous preparatory adjustment of intrinsic stiffness, regulated through reflex-mediated stiffness (Johannson et al., 1991). The use of the joint receptors in such a manner, relies upon previous experience (Huxel, 2005). It is thought that preparatory muscle activation incorporates motor programmes stored in the cerebral cortex to produce muscle activity and joint movement in response to external load (Rose, 1997).

Smith (1996) proposed that stiffness was mediated by a central control. He postulated that; (1) viscoelastic properties of joint can be regulated through movement and thus affect joint stability, (2) optimal strategies including modulation of agonist–antagonist ratios and co-contraction could be learnt as a response to proprioceptive stimuli, and (3), that feed-forward stimuli for particular muscle activation patterns are consistent with the role of the cerebellum.

Biryukova et al (1999), provided experimentally derived evidence to support the proposal of Smith (1996). Elbow flexor stiffness was measured by releasing an applied load to the forearm. Subjects were instructed to hold their arm in the initial horizontal position. The load was either released by the experimenter, or by the subject using their other hand. An overall increase in stiffness was seen when the subjects were in control of the load release. This suggests that subjects learnt to compensate for the effects of unloading using central commands to preset joint stiffness and elbow angle, resulting in increased levels of joint stiffness upon release of the load.

Pre-activation of the muscle, in preparation for the application of external load may be due to the presence of a feed-forward loop (Johannson et al., 1991). Swanik et al (2004), investigated the stiffness, flexibility and EMG activity during a landing task in females with a deficient anterior cruciate ligament (ACL). The ACL–injured group had a significant increase in preparatory activity in the lateral hamstring before landing, and less hamstring stiffness compared with the controls. This study supports others (Branch, Hunter, & Donath, 1989; Dietz, Noth, & Schmidtbleicher, 1981; Greenwood & Hopkins, 1976; Kalund, Sinkjaer, Arendt-Nielsen, & Simonsen) suggesting the presence of pre-programmed muscle activation strategies in order to compensate for injury in the limb.

2.4.9 Extrinsic mechanisms mediating stiffness and functional outcomes

There are also numerous studies (McNair & Marshall, 1994; McNair et al., 1992; Rudolph, 2001) illustrating the importance of an interaction effect between stiffness, function, muscle activation timing and activation by central processing. McNair et al (1992) investigated hamstring stiffness in subjects with an ACL deficient knee, drew comparisons with the contralateral limb, and correlations with function. While no difference was shown between limbs, a moderate correlation was shown between increased hamstring stiffness and increased function. However, in a similar study, Jennings & Seedholm (1998) examined chronic anterior cruciate ligament (ACL) deficient knees and reported a significant difference between angular stiffness values between affected and unaffected limbs. No measure of functional ability was taken by Jennings and Seedholm (1998).

More recently Bryant et al (2008), investigated lower limb stiffness in ACL reconstructed patients and reported a moderate correlation between knee function and lower limb musculotendinous stiffness normalised to body weight. These authors postulated that ACL reconstructed subjects with higher levels of involved limb stiffness were more functional and able to participate in more demanding physical activity with fewer symptoms due to a protective mechanism mediated by neuromuscular control apparatus (Bryant et al., 2008).

The relationship between stiffness and performance in eccentric, isometric and concentric activities in the upper limb was examined by Wilson et al (1994). Thirteen subjects performed a series of maximal bench press efforts, either isometrically, concentrically, or eccentrically. Stiffness was found to be related to isometric and concentric, but not eccentric activities. The authors surmised that stiffer musculotendinous structures facilitate performance by improving the length and rate of shortening as well as enhancing the energy transmission.

Thus a great deal of evidence exists to support the proposal that increased stiffness enhances performance and stability in the peripheral joints. While there is a large body of research to support this phenomenon in the wrist, elbow, knee and ankle, there is a scarcity of such evidence in the shoulder. The shoulder joint varies from other peripheral joints in that it has a heavy reliance upon muscle activity for stability, requires motion through three degrees of freedom, and requires a large amount of movement in order to achieve functional tasks. Thus, while it can be hypothesized that active stiffness is positively related to

performance, quality of life and function, there are presently no studies examining the presence of such relationships in the shoulder.

2.5 Shoulder stiffness

2.5.1 Passive Shoulder Stiffness

A limited number of studies have investigated passive shoulder stiffness in vivo. Borsa et al (2000) studied the patterns of shoulder stiffness in males and females using a custom-designed arthrometer. Subjects sat with the arm fixed at twenty degrees of abduction and in neutral rotation, while a force transducer was applied to pull the humerus first anteriorly, and then posteriorly. Measurements of linear displacement were taken using linear displacement transducers fixed to the skin. Women were found to have increased anterior laxity and decreased anterior stiffness compared with men, and significantly less anterior stiffness compared with posterior stiffness. Although this instrument has been shown to be reliable as a measure of anterior-posterior (AP) laxity in the shoulder (ICC= 0.94 (0.90-0.97)), these results should be interpreted with some caution. Bony translation was measured with sensors on the skin surface, thus making it difficult to be certain of the contribution from bone, muscle, sub-cutaneous, and cutaneous tissue to the degree of movement. The position of testing was at 20 degrees of scapular elevation, whereas most patients report instability at 90 degrees abduction and external rotation. The subjects also had asymptomatic stable shoulders, making it difficult to generalise the results to a pathological population. Further studies (Borsa et al., 2002), were undertaken using the above mentioned arthrometer in males and females in a posterior, anterior and inferior direction. The methodology was as previously described, with the hand supported and arm

pulled in an inferior direction. Neither gender nor direction of force translation, was found to be significantly associated with joint stiffness.

A computerised stress arthrometer (Ligmaster, Sports Tech, Charlottesville, VA) has been used to test passive glenohumeral stiffness in baseball pitchers (Borsa et al., 2006). The Ligmaster has been reported to have moderate within (ICC=0.55) and between (ICC=0.66) session reliability, and excellent within (SEM=1.4 mm) and between (SEM 1.0 mm) session precision for glenohumeral joint laxity measurements (Sauers, Crawford, & McCleod, 2005). With the shoulder positioned in 90 degrees abduction, and 60 degrees external rotation, a load cell measured force applied to the shoulder, while displacement was measured using a displacement transducer. Anterior stiffness was found to be significantly greater than posterior stiffness. No significant difference was found across sides. Some caution must also be taken when interpreting these results. Intraclass coefficient values ranged from 0.2-0.89, dependent upon the limb tested, and the direction measured.

Crawford and Sauers (2006) have also examined the passive shoulder stiffness in high school pitchers using the same computerised stress arthrometry (Ligmaster, Sports Tech, Charlottesville, VA) to assess capsuloligamentous adaptations secondary to repetitive throwing. Anterior glenohumeral laxity in the 90-degree external rotation position was significantly decreased, and stiffness increased, compared with the anterior and posterior in the neutral position. This was thought to be due to the increased tension of the inferior glenohumeral ligament, subscapularis and other soft tissue structures in this position.

While studies into the passive stiffness of shoulders provide some background to the amount of laxity present in the joint, passive stiffness does not replicate activities of everyday life. Additionally, some authors have questioned the relevance of passive stiffness measures (Prado et al., 2005). Thus while investigations into passive stiffness in the shoulder provide some information regarding the background passive tension in the shoulder, studies of active stiffness are required to adequately assess the role of active muscle in providing joint stiffness during functional activities.

2.5.2 Active Shoulder Stiffness

Limited study has been undertaken into active shoulder stiffness. Zhang et al (2000) measured active shoulder stiffness in stable shoulders in the abduction plane. Seven men were positioned with the shoulder in 45 degrees abduction, elbow flexed at 90 degrees with the forearm taped to a fibreglass cast. Isometric MVC was recorded at the beginning of the experiment. Small amplitude perturbations were applied to the arm in the scapular plane by a servomotor across different levels of torque. Glenohumeral stiffness was calculated from the joint abduction angle and abduction torque, and was shown to increase with increasing muscle contraction. However, a number of limitations should be noted. The centre of the humeral head was calculated on two subjects and then used to correlate the humeral position to anatomical landmarks. The humeral head position on the remaining subjects was calculated with palpation of anatomical landmarks. Extrapolating measurements across the group from only two subjects may have increased the error present in this study.

More recently Huxel (2005), investigated the influence of gender, joint position and level of muscle contraction on shoulder stiffness in healthy subjects. Stiffness was measured in a device consisting of a servomotor, gear box and attachment arm. Subjects were positioned in side lying, fixated with a full body vacuum splint, while the arm was perturbated into external rotation. A significant difference was noted between gender during the passive and active conditions, with males exhibiting 39% and 53% more shoulder stiffness that females in the respective passive and active conditions. Generalised joint laxity and strength were shown to predict passive joint stiffness. Strength was the only statistically significant variable in predicting active stiffness. Joint laxity was observed more frequently in the female group but not significantly correlated with shoulder stiffness.

Only one study has investigated active stiffness in the unstable shoulder. Myers (2001), has investigated active stiffness in the unstable shoulder and compared shoulders with history of three or more episodes of instability, with stable shoulders. Nine patients (seven males and two females) were used to study intrinsic and extrinsic stiffness and were age, height and weight matched with a control group. Resistive shoulder torque was calculated using data from a load cell, the inertia of the Biodex arm, the angular acceleration of the Biodex arm, the weight of the human arm, and the distance from the elbow to the centre of mass of the lower arm. Shoulder moment vs. position data was fitted with a linear regression equation. The slope of the data prior to reflex activity was utilized to measure intrinsic stiffness, while the slope of the data following reflex activity provided measures of extrinsic stiffness. Stiffness was found to increase significantly with increased percentage MVC in both stable and unstable

shoulders, but no difference was observed between stable and unstable subjects.

As mentioned previously, shoulder dislocation occurs due to a combination of forced horizontal extension and external rotation (Kirkley et al., 1999; VandenBerghe, 2005). There are no previous studies investigating active shoulder stiffness in unstable shoulders during perturbations in horizontal extension. Given that previous studies (Myers et al., 2004) have demonstrated suppressed activity of pectoralis major in the unstable limb, and that this muscle is responsible for resisting movements into horizontal extension, further examination of active stiffness in this plane of motion is warranted.

2.6 Chapter Summary and Conclusion

Recurrent shoulder instability results in several pathological abnormalities such as osseous Bankart lesions (Itoi et al., 2000), subscapularis atrophy (DePalma et al., 1967; Gamulin et al., 2002), increased biceps latency and suppression of pectoralis major activity (Myers et al., 2004), all of which may predispose the shoulder to episodes of recurrent instability. Given that the current non-operative treatment approach for those with recurrent shoulder instability results in low levels of satisfaction and high levels of recurrent instability (Jakobsen et al., 2007), some authors (Myers & Oyama, 2008; Riemann & Lephart, 2002) have postulated that tissue stiffness may be an important factor in preventing episode of recurrent instability. Active stiffness refers to the resistance from the contractile tissue, primarily as the limb is exposed to external forces which may sublux or dislocate the shoulder. In vivo studies of muscle and tendon stiffness have been undertaken using several different methodologies (Goubel et al.,

1971; Maganaris, 2002; McHugh & Hogan, 2004; Rack & Westbury, 1974). The technique that was thought to be most appropriate for the current study involved perturbation of the shoulder while collecting data associated with force and angular displacement. *A* previous study by Myers (2001) used a similar technique to examine the stiffness of internal rotators of the shoulder, and found no significant difference between stable and unstable shoulders. However traumatic anterior shoulder dislocation commonly occurs during excessive external force into a combination of external rotation and horizontal extension. No previous studies have investigated the stiffness of tissues which resist movement into horizontal extension, and therefore investigation of this parameter seems warranted. Furthermore, the relationships between stiffness in the unstable shoulder and function and quality of life also merit investigation.

CHAPTER 3: METHODS

3.1 Introduction

This chapter has been divided into three sections. The first section provides details of the design and participants. The second section describes the procedure. The final section presents the data and statistical analyses.

3.2 Study design and participants

This is a cross sectional study that utilized subjects with unilateral recurrent shoulder instability and compared the unstable shoulder with the contralateral healthy stable shoulder.

3.2.1 Power & effect size

A pilot study was undertaken to examine the reliability of the procedures and establish the sample size required for the main study. Based on a small to medium effect size (0.4), a power of 0.8, and with alpha set at 0.05, the dependent variable (active shoulder stiffness) was assessed. Using these criteria, a sample size of 15 subjects was required.

3.2.2 Participants

In accordance with the requirements of the Auckland University of Technology (AUT) Ethics Committee (Appendix A), subjects were invited to participate by means of posted advertisements (Appendix B) and by word of mouth. Written and verbal explanations of all experimental procedures were provided (Appendix C). All subjects signed a document of informed consent (Appendix D)

and subjects aged less than 20 years were required to gain parental consent (Appendix E).

3.2.3 Inclusion criteria

Participants were males, aged 16-40 years, with a history of two or more instability episodes, and a positive apprehension and subluxation/relocation sign. An instability episode was defined either as a dislocation requiring assistance to relocate the arm, or a subluxation where the patient perceived the shoulder to move away from the glenoid fossa, with or without associated neural symptoms, which prevented movement of the arm for some period after the incident (Lewis, 2004).

3.2.4 Exclusion criteria

Exclusion criteria included previous shoulder/cervical surgery, bilateral instability, atraumatic instability, current pain in the shoulder or cervical region, or other conditions that may alter sensory or motor function e.g. diabetes, rheumatological disorders or peripheral nerve disorders.

3.3 Procedure

Each participant was tested in a single session in a temperature-controlled laboratory at the Health and Rehabilitation Research Centre at the university.

3.3.1 Shoulder Questionnaires

The Western Ontario Shoulder Instability Index (WOSI) (Appendix F) has been shown to be a valid and reliable measure of quality of life in individuals with shoulder instability (Kirkley et al, 1998). High WOSI scores indicate decreased

quality of life. Valid and reliable measures of shoulder function have also been reported using the patient report section of the American Shoulder Elbow Surgeon Score (ASES) (Appendix G) (Michener, 2002). Low ASES scores indicate decreased levels of function. A global measure of perceived instability was also recorded using the SANE (Single Alpha Numeric Evaluation) (Appendix G). For this question, a high score of 10 indicates a 'very unstable' shoulder. To provide a measure of the participants activity levels, the Brophy Upper Limb Activity (Appendix H) (Brophy, Beauvais, Jones, Cordasco, & Marx, 2002) score was utilized, where a high level of activity is indicated by a maximum score of 20. This activity score has been shown to have excellent reliability and construct validity (Brophy et al., 2002).

3.3.2 Maximum Isometric Voluntary Strength

Peak torque generated during horizontal flexion was determined using a dynamometer (Biodex System 3 Research Toolkit, Biodex Medical System, Inc, NY, USA). Following a five minute warm up on a rowing ergometer, the participant was positioned in supine, and his arm was strapped to the dynamometer arm at 90 degrees flexion and 0 degrees external rotation. Straps were placed over the anterior aspect of the clavicle and attached under the axilla in order to stabilise the scapula and limit movement at the scapulothoracic joint. Additional straps were also placed across the trunk/pelvis area to stabilise the pelvis and prevent movement in the trunk (see Figure 3.1). Subjects were instructed to exert force and attempt to move the elbow to the opposite shoulder. Subjects were taught to limit internal rotation during this movement. Subjects then performed two isometric contractions at approximately 80% of maximal effort. Thereafter, three isometric contractions at 100% MVS were

recorded with verbal encouragement from the investigator. The highest of the three contractions provided the MVS for later calculations. Strength measurements were corrected for limb weight.

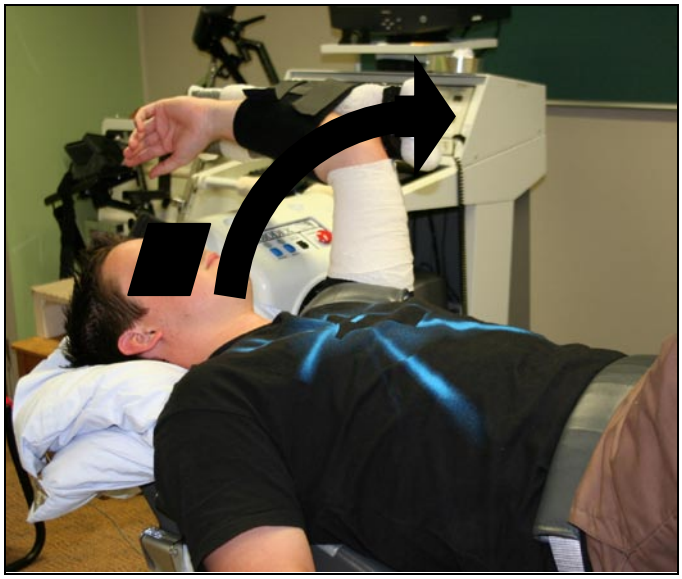


Figure 3.1 Photograph of a subject situated in the testing device during testing. The arrow indicates movement of the limb into horizontal extension.

3.3.3 Active Stiffness

Subjects were positioned in supine with their arm strapped to the Biodex arm as described above for the strength testing. The unstable limb was tested first, followed by the stable limb. The Biodex safety stop was set to allow 24 degrees of horizontal extension movement. Utilizing the Biodex Research Toolkit, the dynamometer was programmed to accelerate the limb to a constant angular velocity of 250 deg/sec in 60 msec. Torque, angle and velocity data were recorded simultaneously from the Biodex at a sampling frequency of 1000Hz and relayed to a computerised data acquisition system (Superscope II, Version 3.0, GW Instruments, MA, USA) for storage and subsequent processing. The

torque recorded during this motion was corrected for the effects of gravity and inertia (McHugh & Hogan, 2004). Thereafter, the difference in torque values at 60 and 0 msec were divided by the change in angle over the same time period to provide a value of active stiffness (Nm/deg). A custom made MATLAB software programme written by Antoine Nordez (University of Nantes, France) was utilized for this purpose. Participants undertook two trials at three submaximal levels of MVS (30, 50, and 70 %). The target torque was displayed on a screen to the experimenter and the subject and the perturbation trial did not commence until the subject was steady at the required torque level. Trials were conducted in a random order and the mean of the two stiffness values was used in subsequent analyses.

Surface electromyography (EMG) signals were recorded from pectoralis major using active electrodes (Delsys DE02.3; Delsys Inc., Boston, MA, USA) with an inter-electrode distance of 10mm and placed upon the skin as per SENIAM guidelines (Hermens & Freriks, 2005) and other studies (Krol, Sobota, & Nawrat, 2007; Suenaga, Minami, & Fujisawa, 2003). The EMG signals were amplified (x1000), band pass filtered (3Hz and 1kHz) and sampled at 1000Hz. Root Mean Square (RMS) values were calculated over five msec epochs with a one msec overlap. The criterion for observing increased EMG activity was two standard deviations above that recorded just prior to the perturbation as has been used in previous studies (Hundza & Zehr, 2007).

The reliability of these procedures was established during pilot testing of 15 healthy subjects. Stiffness was measured at 10%, 30%, 50%, 70% and 90% MVS. Subjects performed a total of twelve trials, four trials at 10%, and two

trials at the remaining percentage levels. All trials were performed in random order. Following data collection, test/re-test reliability values were established. The ICC for active stiffness regression line intercept was 0.929 (0.804, 0.968) while the ICC for the slope of the active stiffness was 0.824 (0.602, 0.928). Typical error for the slope and intercept data were 0.23 and 0.15 respectively. A Bland and Altman plot using the slope values from each trial was generated to test the repeatability of the two trials (see Figure 3.2). The graph was randomly scattered about the mean with no clustering, suggesting there was no bias between test 1 and test 2. The 95% limits of agreement indicate that the difference between the slopes of test 1 and test 2 were small.

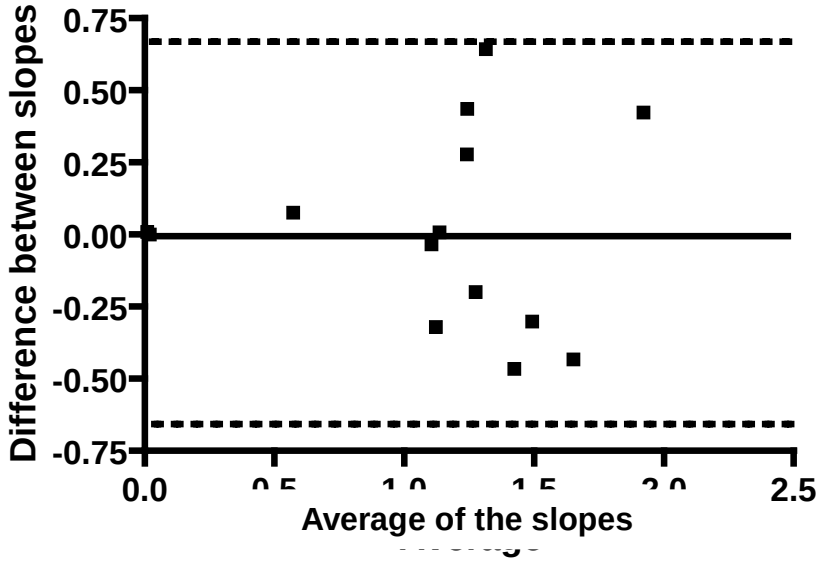


Figure 3.2: Bland Altman of slope data from trial one and trial two the difference between the slopes.

Pilot testing was also conducted with perturbations involving external rotation, in both the supine and sitting position. Low levels of reliability were found for perturbations in this direction and this procedure was subsequently discarded.

In an effort to minimise fatigue, the MVS levels used in the final testing were 30%, 50% and 70% MVS.

3.4 Data and Statistical Analysis

Statistical Analysis was undertaken using Graph Pad Prism (Version 4.00 (GraphPad Software, San Diego California USA)) and SPSS (Version 15.0 (SPSS Inc., Illinois, USA)). The dependent variables of interest were; maximal voluntary strength (Nm), active stiffness (Nm/deg), quality of life (measured with the WOSI), function (measured with the patient report section of the ASES) and perceived instability (measured with the SANE). Firstly, the appropriateness of utilizing parametric analysis was determined. Thereafter, the primary focus of the statistical analysis was the determination of differences in stiffness and strength across the shoulders. In regard to the stiffness measures, as it is well known that stiffness increases with increased levels of muscle activation, the statistical analysis was focused upon differences across shoulders at each level of muscle activation. This was undertaken with three dependent t-tests. A secondary analysis explored the relationships across selected dependent variables. In this regard, the Pearson Product Moment Correlation was used to examine the association between strength, stiffness (normalised to maximum strength), and quality of life, function and perceived instability. For all statistical analysis, significant differences were accepted at the alpha level of 0.05.

CHAPTER 4 – RESULTS

4.1 Introduction

This chapter is divided into five main sections. The first section provides a description of the participants. The second section presents the results from strength testing in the unstable and stable shoulder. Section three describes active stiffness in the unstable and stable shoulder. In the fourth section, quality of life and functional outcome scores are presented. The final section describes the association between stiffness and the number of instability episodes.

4.2 Participants

Thirty six participants responded to advertisements posted in gymnasiums and clinics throughout the Auckland area. Twenty subjects were excluded due the presence of bilateral symptoms, previous surgery, elbow pathology, or the presence of shoulder pain. The remaining 16 subjects met the inclusion criteria and took part in the present study. Participants had a mean age of 21.6 (SD=4.6) years, mean height of 179.4 (SD=6.1) cm and mean mass of 79.1 (SD=6.8) kg. The mean time since injury was 2.67 (range=0.5-10) months. All 16 subjects were right limb dominant, and the dominant limb was affected in 7 subjects. X-ray and MRI films were available for 6 of the 16 subjects. The number of instability episodes experienced after the initial injury ranged from two to twenty (mean=7, SD=6). The mean level of upper limb activity ranged from 6 to 20 (mean=13.75, SD=3.43), as measured by the Brophy Upper Limb Activity (Brophy et al., 2002) score. None of the subjects were involved in unilateral upper limb sports involving increased use of their dominant limb. With respect to further intervention, ten subjects were awaiting surgery for recurrent

anterior instability, whilst six subjects were satisfied with their conservative management and were not seeking any further treatment.

The mean WOSI score was 923 (SD=456.34) of a possible 2100. The range of scores varied from a minimum value of 296, to a maximum value of 1642. The range of ASES scores varied from 12 to 28, where 30 indicates the maximum level of function. The mean score of the ASES for the unstable arm was 22.7 (SD=4.8), and this was significantly lower ($p<0.05$) than for the stable arm (30; SD=0.0). The mean score for the SANE was 4.5 (SD=2.9), where the maximum score is 10, and the scores ranged from 2 to 9.5. Limb dominance was not significantly related to any of the outcome scores.

A significant positive correlation was demonstrated between the WOSI and SANE ($r=0.68$, $p<0.05$), while a significant negative correlation was shown between the ASES and SANE ($r=-0.57$, $p<0.05$), and the ASES and WOSI ($r=-0.82$, $p<0.05$). No significant association existed between the number of instability episodes and WOSI, ASES or SANE outcome scores ($p>0.05$).

4.3 Strength Tests

4.3.1 Maximal voluntary strength in stable and unstable shoulders

There was a significant difference ($p<0.05$), between isometric horizontal flexion strength in the stable and unstable shoulder. The mean was 39.8 Nm (SD=14.1) in the unstable shoulder and 45.1 Nm (SD=14.1) in the stable shoulder. There was no significant association between strength deficits in the unstable arm and the WOSI, ASES or SANE. Additionally, no relationship existed between limb dominance and strength.

4.3.2 Active stiffness and maximum voluntary strength

The association between active stiffness and MVS was examined using Pearson Correlation Coefficient. No significant association ($p>0.05$) was seen between maximum strength in the unstable limb and stiffness values at 30%, 50%, or 70% MVS.

4.4 Active stiffness of stable and unstable shoulders

A significant difference ($p<0.05$) was found between stable and unstable shoulders at the 30% MVS and the 50% MVS level (see Figure 4.2). No significant effect was found at 70% MVS ($p>0.05$). The mean values of stiffness in the stable and unstable shoulder at 30% MVS were 4.7 Nm/deg (SD=1.1) and 3.6 Nm/deg (SD=1.7) respectively (n=16). At the 50% MVS level, the mean values were 5.7 Nm/deg (SD=1.7) and 4.5 Nm/deg (SD=1.2) for the stable and unstable shoulder respectively (n=13). The mean values of stiffness for stable and unstable shoulders at 70% MVS were 5.9 Nm/deg (SD=0.17) and 5.5 Nm/deg (SD=0.1) respectively (n=12).

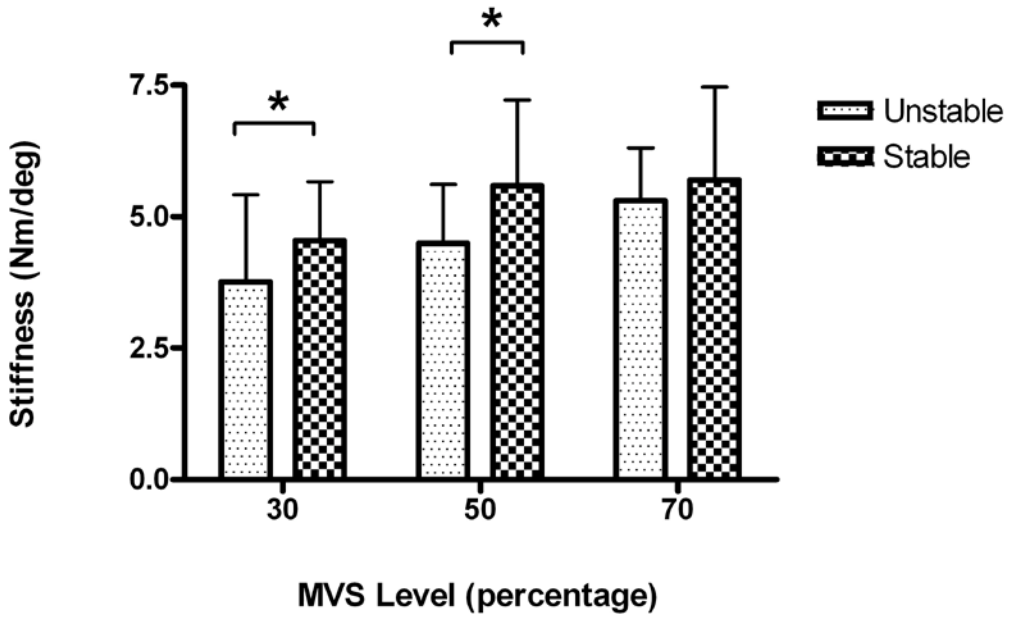


Figure 4.1: Active stiffness in the stable and unstable shoulder at 30%, 50% and 70% MVS.

Up to 58 msec after the perturbation, levels of EMG recorded from pectoralis major were not different from those recorded in the 100 msec prior to the perturbation. At 58 msec, increased EMG activity was noted. As stiffness measurements were calculated over a 60 msec epoch, the increased activity observed at 58 msec was not thought to be influential, particularly when consideration is given to electromechanical delay.

4.5 Active stiffness, quality of life, function and perceived instability

Stiffness values are influenced by the size of the muscles. In an effort to correct for this confounding factor, stiffness was normalised to maximal strength. There were no significant associations ($p>0.05$) observed between normalised stiffness values and the WOSI, ASES or SANE.

4.6 Active stiffness and number of instability episodes

The association between severity of instability and stiffness was also examined. No significant association ($p>0.05$) existed between number of instability episodes and stiffness, nor difference in stiffness values, at 30%, 50% or 70% MVS.

CHAPTER 5 - DISCUSSION

5.1 Introduction

This chapter is divided up into six sections. The first includes a discussion of the participants. The second section discusses maximal voluntary strength in the stable and unstable limb, and findings related to quality of life and function. Thirdly, a discussion concerning active stiffness is presented. This section also includes findings of active stiffness as they pertain to the WOSI, ASES and SANE questionnaires. The fourth section is concerned with the limitations of the study. Thereafter the recommendations for future research and conclusions are presented.

5.2 Participants

The subjects in the current study were typical of individuals with shoulder instability. Quality of life scores as measured by the WOSI score were comparable with pre-operative scores in other studies (Kirkley et al., 1999; Robinson, Jenkins, White, Ker, & Will, 2008). Limitations in function as measured by the ASES score were also similar to other studies (Michener et al, 2002). Shoulder activity levels were also similar to other studies (Brophy et al., 2009).

5.3 Maximal voluntary strength

The current study investigated horizontal flexion strength in the unstable shoulder, as shoulder dislocations occur not only because of a sudden perturbation into external rotation, but also because of excessive force or sudden traumatic load into horizontal extension (Kirkley et al., 1999;

VandenBerghe et al, 2005). The present study found a significant decrease in horizontal flexion strength, when compared to the contralateral stable shoulder. This was a novel finding. While there are many previous studies detailing decreased (Bak & Magnusson, 1997; Dauty et al., 2007; Tsai et al., 1991), or altered (Rupp, 1995) strength of external and internal rotation in the shoulder, an extensive review of the literature failed to reveal any other studies examining horizontal flexion strength in individuals with recurrently unstable shoulders. Strength values in the uninjured, stable shoulder were found to be similar to those of other studies (Flocks, 1995; Silva et al., 2006) investigating isokinetic horizontal flexion in stable shoulders .

5.3.1 Maximal voluntary strength, quality of life and functional limitation

No significant association existed between percentage strength deficits in the unstable limb and WOSI, ASES or SANE. Additionally, no significant association was demonstrated between strength when normalised to body weight and the WOSI, ASES or SANE. These findings concur with those of Tsai et al (1991) who also reported no association between shoulder strength and severity of impairment, as defined by the ROWE score. Similarly, Sachs et al (2005) noted no association between the lift-off test for subscapularis strength and function as measured by the ASES. However, Sachs et al (2005) did note a positive association between strength and WOSI scores. McDermid et al (2004), also studied the impact of rotator cuff pathology on quality of life and disability, and reported that internal and external rotation strength measures were related to disability, while only the presence of rotator cuff pathology (and not strength) was predictive of impaired quality of life. Given the contribution of numerous shoulder muscles to strength in horizontal flexion, it is difficult to determine

whether any specific muscle is more affected than others. However, as many shoulder tasks in sport and working activities require motion, at times combined with large of amounts of force in this plane, it would seem logical to recommend strengthening exercises to improve the performance in such tasks.

5.4 Active stiffness

It has been postulated by several authors (Akazawa et al., 1983; McNair et al., 1992; Myers, 2001; Sinkjaer et al., 1988) that intrinsic active stiffness may be the primary defence in protecting a joint from injury. This study builds upon the work of Myers (2001), who investigated shoulder stiffness of individuals with recurrent shoulder instability by perturbating the limb into external rotation. However, the stiffness values in this study were much higher than those of Myers (2001), and are more similar to that of Zhang et al (2000), who also perturbated the limb into horizontal extension. The increased muscle size of pectoralis major, relative to subscapularis, was thought to be the key component responsible for increased horizontal extension stiffness values compared with external rotation stiffness values.

As has been previously stated, shoulder dislocation occurs due to the combination of external rotation and horizontal extension (Kirkley et al., 1999; VandenBerghe et al, 2005). This study further explored the relationship between active stiffness and recurrent shoulder instability by perturbating the limb into horizontal extension. It was hypothesized that active stiffness would be decreased in unstable shoulders. A significantly lower level of stiffness was observed in unstable shoulders at 30% and 50% MVS while no difference was observed at 70% MVS. These findings suggest that less protection to such

perturbations is provided in the unstable joint at lower levels of muscle activation. While the mechanisms behind the loss of stiffness in horizontal extension are not able to be determined directly from the methods of the current study, there are a number of factors which may be responsible.

Firstly, several authors (Ahmad et al., 2003; Urayama et al., 2003) have documented increased length in the capsule and ligaments of the glenohumeral joint following dislocation. At low levels of muscle activation, these changes in length may increase laxity of the joint subsequently affecting the stiffness of the muscles, which at their attachments fuse with the capsule (Funk, 2005). At higher levels of muscle activation, these muscles may be able 'to take up the slack in the system' and create increased stiffness in the system (Huxel, 2005). Alternatively, more muscles may be involved in resisting motion at higher levels of contraction. It may be that at low levels of muscle activation, stiffness is provided predominantly by the rotator cuff musculature. Stronger more powerful muscles, such as pectoralis major, may be activated at higher levels of muscle contraction and provide increased stiffness.

Other authors (Rack & Westbury, 1984) have postulated that at low levels of muscle activation (less than 30% MVC), the tendinous component of muscle is much stiffer than other contractile components of the muscle. Thus the significant differences which existed at lower levels of stiffness in the current study may reflect deficits in the tendinous portions of the shoulder muscles, rather than the contractile elements of the tissue. These tendinous deficits may reflect a level of pathological damage in the labral tissue (such as a Bankart

lesion) or capsule-tendon aponeurosis which has resulted from repeated shoulder dislocations.

Another reason for the absence of a difference in stiffness at high levels of contraction may be the effect of co-contraction about the joint. Akazawa et al (1983) examined stiffness in the human thumb and found stiffness to increase with increased co-contraction of the flexor and extensor muscles. Similarly, Louie and Mote (1987) measured the ability of quadriceps–hamstring co-contraction to reduce knee laxity. Increased co-contraction increased joint stiffness and decreased joint laxity by 25%. In the present study, co-contraction may have been limited at low levels of muscle activation. At high levels of muscle activation, increased co-contraction may have led to similar amounts of stiffness across shoulders. This point highlights a limitation of the current study that EMG could not be utilized to measure levels of activation in all shoulder muscles. However, given the large number of muscles that contribute to shoulder stability, this would be a difficult task to undertake well.

5.4.1 Active stiffness, quality of life and functional limitation

While differences in stiffness were apparent across shoulders, these were not related to quality of life, function or perceived instability. This was a surprising finding. It was hypothesized that there would be a positive relationship between active stiffness and these outcomes. This hypothesis was based upon previous work in the unstable knee (McNair et al., 1992), which had found function to be positively correlated with stiffness. Movement at the knee however, is much more restricted to primarily one plane of movement compared to shoulder movement which occurs across three planes of movement. Thus it may be that

increased stiffness afforded by muscle activation might be limited in value if the shoulder joint is to move effectively across the multiple planes of possible motion needed for some functional activities. Even though no relationship between active stiffness and function or quality of life was observed, the loss of stiffness means that less protection is present when the joint is unexpectedly perturbated in this plane of motion. Therefore, it would seem prudent to recommend exercises that would remedy this deficit and lessen the chance of a dislocation.

5.4.2 Active stiffness and levels of maximum voluntary strength

Although not investigated with statistical analysis, the mean stiffness appeared to increase at each muscle activation level. This finding is in agreement with other studies of active shoulder stiffness (Huxel, 2005; Huxel et al., 2008; Myers, 2001; Zhang et al., 2000), all of which describe stiffness to increase with increasing level of contraction. This finding is also in agreement with studies of joint stiffness in other peripheral joints, which found that stiffness increased by as much as ten-fold in response to muscle contraction (Ma & Zahalek, 1985; McNair, 1991; Morgan, 1977; Sinkjaer et al., 1988; Weiss et al., 1988; Wilkie, 1950) .

5.5 Limitations of the present study.

This section will identify and discuss limitations in the present study with respect to the methodology used in strength testing, the presence of pathology in the affected shoulder, testing of active stiffness, and the effect of limb position, EMG, and questionnaires utilized.

5.5.1 Strength testing

The use of MVS as a measure of strength is accompanied by inherent flaws. In order to produce forces at MVS level, subjects need to be motivated, familiar with exercise and free from any pain-avoidance behaviour (Crombez et al., 1999). Additionally MVS can vary due to the level of verbal encouragement that the patient receives (McNair et al., 1996). None of the subjects reported pain during the isometric horizontal flexion task. It is possible however, that some subjects did not fully engage in the task because of 'fear avoidance' due to instability, despite consistent verbal encouragement. All of the subjects had suffered numerous previous dislocations or subluxations in their shoulder that resulted in significant episodes of pain or apprehension. The possibility of increased error due to apprehension or fear avoidance remains a consideration when testing in a group of subjects with shoulder instability.

5.5.2 Stiffness

The current methodological technique provides an indirect measure of stiffness as direct measurements of stiffness are only possible in studies involving animals or cadavers. While direct measurement of stiffness is useful in determining theories of stiffness as it applies to joint stability, indirect studies using pathological conditions provide clinically relevant evidence of stiffness which is more specific to clinical conditions. Additionally the possibility exists that experimentally created dislocation in cadavers may not replicate the situation in vivo. Thus research is necessary that accurately represents the pathological population in vivo.

5.5.3 Effect of limb position

For ethical reasons it was not possible to position the arm in 90 degrees of abduction and 90 degrees external rotation. Other studies (Huxel, 2005), have shown variations in muscle recruitment in different parts of the range of motion. Altering the limb position along a single plane will alter the length-tension relationship of the muscles involved, and may have an effect upon stiffness. Myers (2001), also commented upon the difficulty in obtaining a true apprehension position. Subjects in the study by Myers (2001), were positioned in 'pseudo apprehension' (90 degrees elevation with 30 degree horizontal flexion in the scapular plane). This position was reported to be 'ligament friendly' by decreasing the load upon shoulder ligaments (Poppen & Walker, 1978; Saha et al., 1983). Myers (2001), proposed that muscles in the dominant scapular plane may mask the possible effects of capsular redundancy or deficiency on muscle-joint complex stiffness.

Perturbating the limb into horizontal extension may have increased the contribution of pectoralis major relative to other rotator cuff muscles (e.g. subscapularis). There is limited literature available detailing pathological changes within pectoralis major as a result of traumatic dislocation. There is however, research documenting the pathological changes in subscapularis following dislocation (DePalma et al., 1967; Gamulin et al., 2002). Despite expected difficulties in establishing reliability during perturbations into external rotation, alternative values of active stiffness and possible correlations with quality of life and function may be present in this direction.

5.5.5 ASES and WOSI questionnaires

While the questionnaires chosen had received notable psychometric evaluation, they may not accurately reflect the quality of life and function in the participants. Questionnaires are only able to reflect the subjects' perceptions of their ability, while the performance of actual tasks may show a different picture.

5.6 Recommendations and future research

The findings of the current study contribute to the knowledge and understanding of active stiffness in the recurrent unstable shoulder. In light of this study's findings, this section will now discuss four key areas that have been identified for future research.

Despite the lack of an association found between stiffness and quality of life and function found in the present study, further exploration of the relationship between stiffness and function in the unstable shoulder is warranted. The present study examined stiffness in one plane of motion, as has been undertaken in other peripheral joints (Bryant et al., 2008; McNair et al., 1992). Due to the multi-directional nature of shoulder movement, it may be that stiffness of the shoulder during co-contraction of the rotator cuff musculature around the shoulder examined three dimensionally, would more accurately reflect the functional stiffness of the system and thus be related to quality of life and function. Investigations into measuring stiffness during shoulder co-contraction in a three dimensional manner should be explored to further examine this relationship.

Although early examinations of stiffness have been studied in the ankle and knee, few studies have examined therapeutic methods to increase stiffness in unstable joints. A variety of exercise types have been suggested. McNair et al (1992) recommended endurance type exercises based upon the work of Goubel and Marini (1987) and Kovanen et al (1984) and strength type exercises based upon the work of Pousson (1990). Isometric exercises have also been shown to increase stiffness in the patellar tendon of healthy knees (Kubo, Yata, Kanehisa, & Fukunaga, 2005). The effectiveness of a rehabilitation programme following an episode of instability would be enhanced, with the knowledge of those exercises that most improved stiffness.

Another area for future consideration is the consideration of shoulder perturbation with fine wire EMG. Previous studies (Myers, 2003) have demonstrated alterations in rotator cuff activity. While several authors have postulated that alterations exist to upset the delicate balance of force couples around the shoulder, there are few conclusive data demonstrating the existence of inhibited rotator cuff musculature in unstable shoulders. It is possible that subscapularis activity is inhibited; either because dominant pectoralis major/latissimus dorsi patterning alters the motor programme in such a way that subscapularis is no longer recruited in an efficient and timely manner, or because of pathological changes within the joint. These pathological changes include alteration of joint receptors and increased negative intra-articular pressure. Thus further investigation into the presence and effect of subscapularis inhibition is warranted.

Further studies should also include a group of subjects who have had a single shoulder dislocation with no further episodes of instability and compared to those who have failed conservative treatment. The inclusion of this group would allow the illustration of the adaptive responses that may be present in this population, and may show different stiffness values when compared with those who have failed conservative treatment. It may be that the group of single dislocators have developed the necessary feed-forward loop mechanisms to modify and regulate their stiffness. Differences between these two groups may hold the answer to successful conservative rehabilitation of the unstable shoulder.

5.7 Conclusions

The observed decreases in active stiffness and strength in the unstable shoulder provide new information to support the incorporation of exercises to remedy these deficits in an effort to provide increased protection to the joint, as well as improve performance of tasks that involve the affected muscles. The lack of a relationship between active stiffness and perceived quality of life and function would suggest that other factors (to which stiffness may contribute), play a greater role in allowing those with instability to return to their work and sporting activities. It may be that the multi-dimensional nature of shoulder movement requires a limit to the amount of stiffness allowed, if shoulder movements are to be undertaken efficiently and effectively.

APPENDICES

APPENDIX A

M E M O R A N D U M

Auckland University of Technology Ethics Committee (AUTEC)

To: Peter McNair
From: Madeline Banda Executive Secretary, AUTEC
Date: 30 October 2007
Subject: Ethics Application Number 07/161 Active stiffness in unstable shoulder.

Dear Peter

Thank you for providing written evidence as requested. I am pleased to advise that it satisfies the points raised by the Auckland University of Technology Ethics Committee (AUTEC) at their meeting on 10 September 2007 and that the Chair of AUTEC) has approved your ethics application. This delegated approval is made in accordance with section 5.3.2.3 of AUTEC's *Applying for Ethics Approval: Guidelines and Procedures* and is subject to endorsement at AUTEC's meeting on 12 November 2007.

Your ethics application is approved for a period of three years until 30 October 2010.

I advise that as part of the ethics approval process, you are required to submit to AUTEC the following:

- A brief annual progress report indicating compliance with the ethical approval given using form EA2, which is available online through http://www.aut.ac.nz/about/ethics, including when necessary a request for extension of the approval one month prior to its expiry on 30 October 2010;
- A brief report on the status of the project using form EA3, which is available online through http://www.aut.ac.nz/about/ethics. This report is to be submitted either when the approval expires on 30 October 2010 or on completion of the project, whichever comes sooner;

It is also a condition of approval that AUTEC is notified of any adverse events or if the research does not commence and that AUTEC approval is sought for any alteration to the research, including any alteration of or addition to the participant documents involved.

You are reminded that, as applicant, you are responsible for ensuring that any research undertaken under this approval is carried out within the parameters approved for your application. Any change to the research outside the parameters of this approval must be submitted to AUTEC for approval before that change is implemented.

Please note that AUTEC grants ethical approval only. If you require management approval from an institution or organisation for your research, then you will need to make the arrangements necessary to obtain this.

To enable us to provide you with efficient service, we ask that you use the application number and study title in all written and verbal correspondence with us. Should you have any further enquiries regarding this matter, you are welcome to contact Charles Grinter, Ethics Coordinator, by email at charles.grinter@aut.ac.nz or by telephone on 921 9999 at extension 8860.

On behalf of the Committee and myself, I wish you success with your research and look forward to reading about it in your reports.

Yours sincerely

Madeline Banda
Executive Secretary
Auckland University of Technology Ethics Committee
Cc: Margie Olds margie@flawlessmotion.com

APPENDIX B

Want to be involved in a study on……..

shoulder instability?

You are eligible if:
You are male, aged 16-40 years
You have had 2 or more dislocations / subluxations in your shoulder
You are not currently receiving treatment for your shoulder
You have no pain in your shoulder
You have no other diseases that may affect your sensation or muscles.

Subjects will be excluded if they have had previous shoulder surgery.

Where:
Study will take place at Health and Rehabilitation Research Centre, AUT Akoranga Campus, Northcote, Auckland

How much time?
The study will take no more than 1 hour of your time
Call Jane (Rehabilitation Centre Administrator) on 09-9219999 Ext 7194, or Margie (Researcher) (0215-73422) for more details

Shoulder Instability 921-999 x 7194	Shoulder Instability 921-999 x 7194	Shoulder Instability 921-999 x 7194	Shoulder Instability 921-999 x 7194	Shoulder Instability 921-999 x 7194	Shoulder Instability 921-999 x 7194	Shoulder Instability 921-999 x 7194

APPENDIX C

Participant Information Sheet

Date Information Sheet Produced:
23/08/2007

Do you need an interpreter?

English	I wish to have an interpreter	Yes	No
Maori	E hiahia ana ahau ki tetahi Kaiwhakamaori / Kaiwhakapakeha korero	Ae	Kao
Samoan	Oute mana'o ia iai se fa'amatala upu	Ioe	Leai
Tongan	Oku ou fiema'u ha fakatonulea	Io	Ikai
Cook Island	Ka inangaro au I tetai tangata uri reo	Ae	Kare
Niuean	Fia manako au ke fakaaoga e taha tagata fakahokohoko kupu	E	Nakai

Project Title

Active Stiffness in the Unstable Shoulder

An Invitation

You are invited to take part in a research study that is being undertaken by the Health and Rehabilitation Research Centre at the Auckland University of Technology. This information sheet explains the study to you, and you can then decide whether you would like to be involved. It is entirely your choice, and if you do agree to take part, you are free to withdraw from the study at any time without having to give a reason. If you do not understand any aspect of the study described below, please ask for clarification. You do not have to decide immediately about participating in the study. However, if all the subjects required are selected before your decision is made, you will not be included in the study.

What is the purpose of this research?

This research study examines the shoulders of patients who have had a dislocation and or repeated episodes of instability (e.g. your shoulder popping out of its socket). Many shoulders require surgery for shoulder instability. The aim of this study is to help improve physiotherapy treatment so that surgery may not be required. Specifically it aims to investigate the compliance (or stretchiness) of the muscles and ligaments in people who have had dislocations or subluxation episodes, and compare with people with normal shoulders.

The secondary purpose of this study is to investigate the relationship between levels of function in the shoulder and muscle / ligament compliance

This research is being performed as part of the requirements for Margie Olds to complete a Master of Health Science Degree in Physiotherapy. The data collected will be published in a thesis to be held at the Auckland University of Technology Library (Akoranga campus). In addition, the data collected from this study will be used to write a paper that will be submitted to a professional journal for possible publication. Finally, the data collected from this study may be used for presentations at professional conferences.

No material that could personally identify you will be used in any reports on this study unless your personal approval is given for the dissemination of results to

specific persons (please see the section below titled “How will my privacy be protected?” for more information on privacy issues).

How was I chosen for this invitation?

This study requires 29 volunteers who have had shoulder dislocations or subluxation in their shoulder and 29 volunteers who have no pathology in their shoulder.

Volunteers with shoulder instability will meet the following criteria:

- A history of 2 or more shoulder dislocations or subluxations

Exclusion criteria for all subjects:

You will not be able to participate in the study if you:

- Have had previous shoulder surgery,
- Have pain in the shoulder or neck area.
- Currently are under treatment for shoulder instability.
- Have conditions that may alter sensory or motor function e.g. diabetes, rheumatological disorders or peripheral nerve disorders.

What will happen in this research?

There are three main parts to the research

1. Questionnaire:

   Subjects will fill out two questionnaires that ask questions about the level of pain, instability and functional impairment that result from their shoulder pathology. Details of subjects’ gender, weight, height and age will also be recorded.

2. Maximal Strength Testing

   This session will begin with a 5-minute warm-up.

   To test the strength of your shoulder, you will seated, and strapped to a machine, with your arm positioned out to the side. You will then be required to push as hard as you can three times, against the machine.

3. Shoulder stiffness

   The final stage of the experiment involves you sitting and strapped to the machine. The machine arm will move backwards while you resist it forwards. This will be repeated at different percentages of your maximal strength. We will record how much your strength is, and the degree that the arm moves. This gives us a measure of how stiff the tissue is, and may be relevant to why your shoulder has dislocated more than once.

What are the discomforts and risks?

Your shoulder will be positioned with your arm at 90 degrees out to the side and your hand turned up towards the ceiling. This may be a position that makes you apprehensive about your shoulder re-dislocating.

The other risks are related to the strength testing procedures.

- There is the risk of delayed onset muscle soreness. As the name suggests this is soreness of the muscles that begins one or more days after exercise. This can occur after lifting heavy weights or doing exercises you are not used to. While this can be uncomfortable the symptoms usually go away after one or two days.
- There is the risk of developing a new injury.

- There is a minimal risk that your shoulder will suffer another subluxation or dislocation. In the unlikely event that your shoulder does dislocate, the following protocol will be adhered to.
    i. The subject will be removed from the machine
    ii. First aid will be applied by the researcher (Margie Olds), who is a registered physiotherapist.
    iii. If necessary, the subject will be transported to the nearest hospital / emergency department for medical assistance.

How will these discomforts and risks be alleviated?

All subjects will participate in a session to be familiarized with the equipment and the procedures and a standardized warm-up will be performed at the beginning of each session to minimize the risk of injury.

In order to ensure that your shoulder does not move out of its socket, your hand will be positioned in the middle of range. This will increase the support about your shoulder to ensure that it will not re-dislocate.

The researchers involved in the study will be available to subjects after completion of the study should any stress, harm or related concerns arise. The researcher who will be conducting the testing sessions (Margie Olds) is a registered physiotherapist with post-graduate training in sports physiotherapy and is capable of assessing injuries, providing first aid and suggesting appropriate options for further assessment and treatment.

What are the benefits?

Many subjects (up to 80%) suffer from repeated dislocations to the shoulder. This study will further knowledge of shoulder dislocations and recurrent instability, and improve physiotherapy treatments after dislocation.

This study also tests the strength of your shoulder. Feedback will be available to you at the completion of the research project regarding the strength and stiffness of your shoulder.

What compensation is available for injury or negligence?

Compensation is available through the Accident Compensation Corporation within its normal limitations.

How will my privacy be protected?

No material that could personally identify you will be used in any reports on this study unless your personal approval is given for the dissemination of results to specific persons. All subjects will be assigned a number and only the principal researchers of this study will have access to your name. All participant records will be kept in locked storage area by the principal researchers.

If you wish to have access to the results of this research, you are entitled to request a copy from Peter McNair. These copies will be available after the study is completed and published.

What are the costs of participating in this research?

There are no financial costs to you for this study. You will be reimbursed for travel costs to AUT ($15) where this research is being undertaken. It will take no more than 1 hour to complete this study.

What opportunity do I have to consider this invitation?

This invitation is open to you for the next 2 months. This opportunity is not available to those patients who have had shoulder surgery.

How do I agree to participate in this research?

If you would like to participate in this research, please call Jane Galle (Research Administrator) on (09) 921-999 ext 7194 or Margie on 0215-73422. They will take your details and we will call you to arrange a suitable time for you to come in for the study.

If you agree to participate in the study, please complete the attached consent form.

Will I receive feedback on the results of this research?

If you would like to receive feedback from this study, we will be sending out a report following the completion of this research. You will also have access to a bound copy of the thesis at AUT library.

What do I do if I have concerns about this research?

Any concerns regarding the nature of this project should be notified in the first instance to the Project Supervisor, Peter McNair, peter.mcnair@aut.ac.nz 921 9999, ext 7143

Concerns regarding the conduct of the research should be notified to the Executive Secretary, AUTEC, Madeline Banda, *madeline.banda@aut.ac.nz* , 921 9999 ext 8044.

Whom do I contact for further information about this research?

Researcher Contact Details:

Margie Olds C/- AUT

0215-73422

Project Supervisor Contact Details:

Peter McNair, *peter.mcnair@aut.ac.nz,* 921 9999 ext 7143.

Approved by the Auckland University of Technology Ethics Committee on 30/10/2007, AUTEC Reference number 07/161.

APPENDIX D

Consent/Assent Form

For use when laboratory or field testing is involved. Participants aged 16-18 years are required to submit this form as well as a parental consent form.

Project title: Active Stiffness in the Unstable Shoulder
Project Supervisor: Peter McNair
Researcher: Margie Olds

- ○ I have read and understood the information provided about this research project in the Information Sheet dated 11/05/2007
- ○ I have had an opportunity to ask questions and to have them answered.
- ○ I understand that I may withdraw myself or any information that I have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- ○ I am not suffering from heart disease, high blood pressure, any respiratory condition (mild asthma excluded), any illness or injury that impairs my physical performance, or any infection. I have had no previous shoulder surgery, ailments in sensory or motor function, e.g. diabetes, rheumatological disorders or peripheral nerve disorders.
- ○ I have no pain in the shoulder or neck area, and am not currently receiving treatment for shoulder instability.
- ○ If I withdraw, I understand that all relevant information will be destroyed.
- ○ I agree to take part in this research.
- ○ I wish to receive a copy of the report from the research (please tick one): Yes○ No○

Participant's_signature:.....
Participant's_name:.....
Participant's Contact Details (if appropriate):
……..
……..
……..
……..
Date:

Approved by the Auckland University of Technology Ethics Committee on 30/10/2007. AUTEC Reference number 07/161

Note: The Participant should retain a copy of this form.

APPENDIX E

Parent/Guardian Consent Form

For use in conjunction with either an appropriate Assent Form when involving participants aged 16-18 years whose age makes them vulnerable as concerns consent.

Project title: Active Stiffness in the Unstable Shoulder
Project Supervisor: Peter McNair
Researcher: Margie Olds

- ○ I have read and understood the information provided about this research project in the Information Sheet dated 23/10/2007
- ○ I have had an opportunity to ask questions and to have them answered.
- ○ I understand that I may withdraw my child/children and/or myself or any information that we have provided for this project at any time prior to completion of data collection, without being disadvantaged in any way.
- ○ If my child/children and/or I withdraw, I understand that all relevant information, or parts thereof, will be destroyed.
- ○ I agree to my child/children taking part in this research.
- ○ I wish to receive a copy of the report from the research (please tick one): Yes○ No○

Child/children's name/s :
……
……
Parent/Guardian's signature:
.....……
Parent/Guardian's name:
.....……
Parent/Guardian's Contact Details (if appropriate):
……..
……..
……..
……..
Date:

Approved by the Auckland University of Technology Ethics Committee on 30/10/2007
AUTEC Reference number 07/161
Note: The Participant should retain a copy of this form.

APPENDIX F

WESTERN ONTARIO

SHOULDER INSTABILITY

INDEX (WOSI)©

A disease-specific quality of life measurement tool for patients with shoulder instability

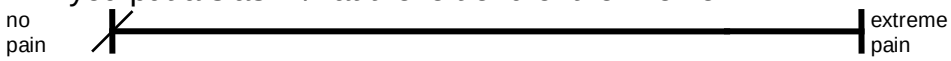
Suggested citation: The Development and Evaluation of a Disease-Specific Quality of Life Measurement Tool for Shoulder Instability: The Western Ontario Shoulder Instability Index. AJSM 26(6):764-772, 1998.

INSTRUCTIONS TO PATIENTS

In Sections A, B, C, and D you will be asked to answer questions in the following format and you should give your answer by putting a slash "/" acrossthe horizontal line.

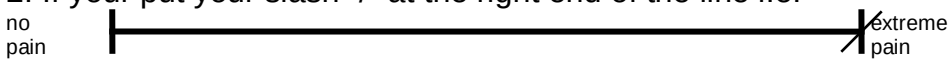
NOTE:

1. If you put a slash "/" at the left end of the line i.e.



then you are indicating that you have no pain.

2. If your put your slash "/" at the right end of the line i.e.



then you are indicating that your pain is extreme.

3. Please note:

a) that the further to the right you put your slash "/" , the more you experience that symptom.

b) that the further to the left you put your slash "/", the less you experience that symptom.

c) please do not place your slash "/" outside the end markers

You are asked to indicate on this questionnaire, the amount of a symptom you have experienced in the past week as related to your problematic shoulder. If you are unsure about the shoulder that is involved or you have any other questions, please ask before filling out the questionnaire.

If for some reason you do not understand a question, please refer to the explanations that can be found at the end of the questionnaire. You can then place your slash "/" across the horizontal line at the appropriate place. If an item does not pertain to you or you have not experienced it in the past week,

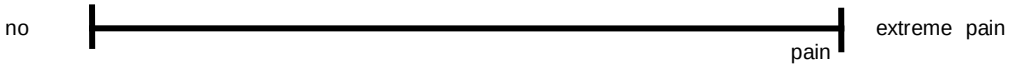
please make your "best guess" as to which response would be the most accurate.

Section A:
Physical Symptoms

INSTRUCTIONS TO PATIENTS

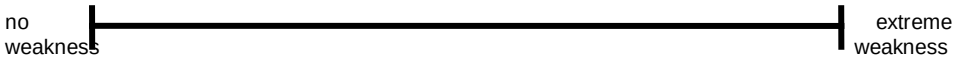
The following questions concern the physical symptoms you have experienced due to your shoulder problem. In all cases, please enter the amount of the symptom you have experienced in the last week. (Please answer with a slash “/" across the horizontal line.)

1. How much pain do you experience in your shoulder with overhead activities?



2. How much aching or throbbing do you experience in your shoulder?

3. How much weakness or lack of strength do you experience in your shoulder?



4. How much fatigue or lack of stamina do you experience in your shoulder?

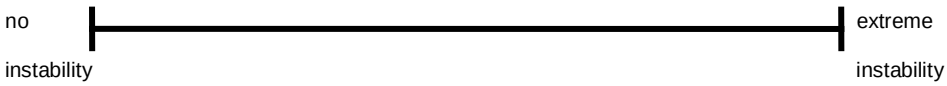
5. How much clicking, cracking or snapping do you experience in your shoulder?

Section A: Cont'd

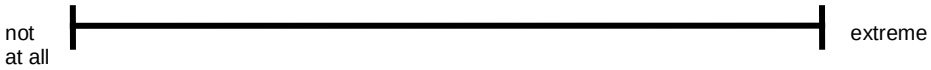
6. How much stiffness do you experience in your shoulder?

7. How much discomfort do you experience in your neck muscles as a result of your shoulder?

8. How much feeling of instability or looseness do you experience in your shoulder?



9. How much do you compensate for your shoulder with other muscles?



10. How much loss of range of motion do you have in your shoulder?

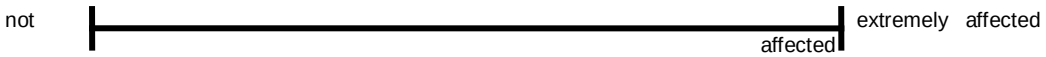
SECTION B: Sports/Recreation/Work

INSTRUCTIONS TO PATIENTS

The following section concerns how your shoulder problem has affected your work, sports or recreational activities in the past week. For each question, please indicate the amount with a slash "/" across the horizontal line.

11. How much has your shoulder limited the amount you can participate in sports or recreational activities?

12. How much has your shoulder affected your ability to perform the specific skills required for your sport or work? (If your shoulder affects both sports and work, consider the area that is most affected.)



13. How much do you feel the need to protect your arm during activities?

14. How much difficulty do you experience lifting heavy objects below shoulder level?

SECTION C: Lifestyle

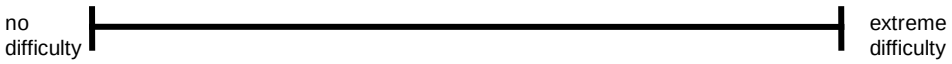
INSTRUCTIONS TO PATIENTS

The following section concerns the amount that your shoulder problem has affected or changed your lifestyle. Again, please indicate the appropriate amount for the past week with a slash “/" across the horizontal line.

15. How much fear do you have of falling on your shoulder?

16. How much difficulty do you experience maintaining your desired level of fitness?

17. How much difficulty do you have “roughhousing or horsing around” with family or friends?



18. How much difficulty do you have sleeping because of your shoulder?

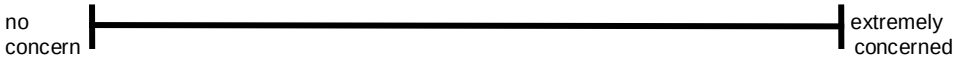
SECTION D: Emotions

INSTRUCTIONS TO PATIENTS

The following questions relate to how you have felt in the past week with regard to your shoulder problem. Please indicate your answer with a slash "/" across the horizontal line.

19. How conscious are you of your shoulder?

20. How concerned are you about your shoulder becoming worse?



21. How much frustration do you feel because of your shoulder?

THANK YOU FOR COMPLETING THE QUESTIONNAIRE

An Explanation of the Meaning of the Questions in the Western Ontario Shoulder Instability
(WOSI) Index

Section A: Physical Symptoms

Question 1.
Refers to any activity requiring you to raise your arm above shoulder level. i.e. putting dishes in a cupboard, styling your hair, swimming the front crawl, painting a ceiling or throwing a ball overhand etc.

Question 2.
Refers to a dull background pain as opposed to sharp pains that are quick or sudden.

Questions 3.
Refers to a lack of strength to carry out an action using your arm.

Question 4.
Refers to your shoulder becoming tired or not being able to do something for as long a period of time.

Question 5.
Refers to the noises that occur in the shoulder with motion.

Question 6.
Refers to the feeling of the joint not wanting to move, which is often experienced in the morning upon rising, after exercise or after a period of inactivity. This does not refer to a lack of range of motion.

Question 7.
Refers to the amount of tension, pain or spasm you experience in the muscles of your neck that seem to be caused by your shoulder problem..

Question 8.
Refers to your shoulder feeling like it is coming part way or completely out of joint, slipping down or sliding in different directions.

Question 9.
Refers to using the muscles in your arm or back to compensate for your shoulder when you perform movements or activities.

Question 10.
Refers to not having full movement of your shoulder in all or any direction(s).

Section B: Sports/Recreation/Work

Question 11.
Refers to having to restrict the amount that you can participate in an activity or if you have had to stop all together.

Explanation of Questions contd

Question 12.

Refers to any difficulty you have performing the skills that are required at work or in a sport or recreational activity.

Question 13.

Refers to consciously or unconsciously protecting your arm by keeping it close to your body, shielding it or wearing a brace.

Question 14.

This does not refer to lifting objects above your head but lifting something heavy below shoulder level eg. a bag of groceries, equipment at work, books, bowling ball.

Section C: Lifestyle

Question 15.

Refers to the fear of falling on your shoulder or onto your outstretched hand on that side.

Question 16.

Refers to the fitness level you maintained before your shoulder became a problem. Includes a decrease in cardiovascular fitness, strength level, or muscle tone.

Question 17.

Refers to any type of rough or vigorous play activity that you would normally engage in with your family or friends.

Question 18.

Refers to having to change your sleeping position, waking up during the night, trouble getting to sleep or waking up feeling unrested due to your shoulder.

Section D: Emotions

Question 19.

Refers to always being aware of your shoulder or taking it into consideration before doing anything

Question 20.

Refers to being concerned about your shoulder becoming worse instead of better or staying the same.

Question 21.

Refers to feeling frustrated because of your inability to do things you used to do or that you want to do but can't because of your shoulder.

APPENDIX G
SANE Questionnaire
Does your shoulder feel unstable (as if it is going to dislocate?)

How unstable is your shoulder (mark line?)

0 _____ 10

Very stable very unstable

ASES Questionnaire

Circle the number in the box that indicates your ability to do the following activities: 0= Unable to do; 1= Very difficult to do; 2= Somewhat difficult; 3= Not difficult		
Activity	Right Arm	Left Arm
1. Put on a coat	0 1 2 3	0 1 2 3
2. Sleep on your painful or affected side	0 1 2 3	0 1 2 3
3. Wash back / do up bra in back	0 1 2 3	0 1 2 3
4. Manage toileting	0 1 2 3	0 1 2 3
5. Comb hair	0 1 2 3	0 1 2 3
6. Reach a high shelf	0 1 2 3	0 1 2 3
7. Lift 10 lbs above shoulder	0 1 2 3	0 1 2 3
8. Throw ball overhead	0 1 2 3	0 1 2 3
9. Do usual work – List:	0 1 2 3	0 1 2 3
10. Do usual sport – List:	0 1 2 3	0 1 2 3

APPENDIX H

Brophy Questionnaire

Please indicate **with an "X"** how often you performed each activity in your healthiest and most active state, **in the past year.**

	Never or less than once a month	**Once a month**	**Once a week**	**More than once a week**	**Daily**
Carrying objects 8 pounds or heavier by hand (such as a bag of groceries)					
Handling objects overhead					
Weight lifting or weight training with arms					
Swinging motion (as in hitting a tennis ball, golf ball, baseball, or similar object)					
Lifting objects 25 pounds or heavier (such as 3 gallons of water) NOT INCLUDING WEIGHT LIFTING					

For each of the following questions, please **circle the letter** that best describes your participation in that particular activity.

1) Do you participate in contact sports (such as, but not limited to, American football, rugby, soccer, basketball, wrestling, boxing, lacrosse, martial arts, etc.)?

   A No
   B Yes, **without** organized officiating
   C Yes, **with** organized officiating
   D Yes, at a professional level (i.e. paid to play)

2) Do you participate in sports that involve hard overhand throwing (such as baseball, cricket, or quarterback in American football), overhead serving (such as tennis or volleyball), or lap/distance swimming?

   A No
   B Yes, **without** organized officiating
   C Yes, **with** organized officiating
   D Yes, at a professional level (i.e. paid to play)

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