ACTIVE STIFFNESS AND RECURRENT SHOULDER INSTABILITY

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2.4 Stiffness

2.4.1 Introduction

Some authors \(^1\,\,^2\) 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 or force, to the rate of change in angular rotation \(^3\), or length, and is the reciprocal of compliance \(^4\). Stiffer joints are more resistant to external force and the capsuloligamentous tissues are less likely to be injured when exposed to external forces \(^4\,\,^6\). 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 \(^7\).

2.4.2 Physiological factors influencing stiffness

2.4.2.1 Studies in isolated muscle

Early work \(^8\,\,^9\) examining muscle stiffness, focused upon isolated muscle. Wilkie \(^8\) described a technique to examine muscle stiffness called the ‘quick release’ technique.
It was based upon a model of muscle advanced by Hill \(^9\), 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 \(^{10}\), examined resting muscle to further examine elements of muscle which 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 \(^{11}\), described a technique to measure the increase in contractile component muscle stiffness immediately following a short stretch. Joyce et al \(^{11}\) noted that the tension developed during lengthening or shortening was modified by changes in length which had preceded those situations. Rack and Westbury \(^{12}\), 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\textsuperscript{13} 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\textsuperscript{14}, and Huxley\textsuperscript{15}, together with Hill’s\textsuperscript{9} 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\textsuperscript{13}.

Flitney and Hirst\textsuperscript{16, 17}, proposed that the less steep or shoulder section of the short range stiffness may be 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 \(^{17}\).

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 \(^{18}\) 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 \(^9\) model.

Work by Morgan \(^{18}\) and Walmsley and Proske \(^{19}\) suggested that tendon stiffness was relatively constant. However Rack and Westbury \(^{20}\) 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 \(^{21}\). The method was based upon the knowledge that muscle spindles were very sensitive indicators of changes in muscle fibre length \(^{22}\), 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 \(^{21}\) and Proske and Morgan \(^{23}\) 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 \(^{23}\) 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 \(^2\text{1}\). 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 low externally applied forces, which lengthen the muscle, will be more effectively transmitted to the muscle fibres and muscle spindles via the tendon. However, at high force levels the more compliant tendon lengthens, thus attenuating the forces reaching the muscle fibre. Additionally, as muscle fibres have been shown to have a viscous, as well as an elastic component, the movement of the muscle fibre will also lag behind the external movement \(^2\text{1}\).

Maganaris and Paul \(^2\text{4}\) utilised ultrasound to examine the mechanical properties of muscle and tendinous tissue. Based upon previously developed methodology \(^2\text{5}\), \(^2\text{6}\) Maganaris and Paul 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 increases curvilinearly as a function of displacement and strain.

It was further proposed by Maganaris and Paul \(^2\text{7}\) 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 to total mechanical work would increase.

Bojsen-Moller et al\(^2\) 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\(^2\) utilised 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. 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 further utilised 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 when measuring Achilles and patella tendon stiffness. 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\textsuperscript{30} 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\textsuperscript{31, 32} have made other adaptations to the original controlled and quick release methods of Hill\textsuperscript{9} and Wilkie\textsuperscript{8} 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\textsuperscript{31} 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 \(^{33}\) 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:

\[
K = 4\pi^2 mf^2 + \frac{c^2}{4m},
\]

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 \(^{34}\) 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, 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.\(^4\) 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 muscle 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 utilised to measure stiffness is applied vibration. Hunter and Kearney\(^35\), 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.\(^36\) 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 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.\(^\text{37}\), studied the regulation of multiple-joint stiffness by measuring human arm stiffness and electromyography (EMG) signals. EMG was assumed to reflect corresponding muscle stiffness and 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\(^\text{38}\), 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\(^\text{39}\), 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 \(^{40}\) 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 \(^{41}\) noted an
decrease in wrist displacement with increased wrist joint stiffness. As seen in other
studies \(^{42,43}\), 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 \(^{44}\).
This method is based upon the work of Morgan \(^{18}\) 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 \(^{44}\).

The current study builds upon previous studies \(^{40,44}\) 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. An additional advantage of this methodology was 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 utilised 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-tendinous of the Australian blue-tongued lizard. 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. 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 \(^{47}\). 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 \(^{48}\).

Later work by Kovanen et al \(^{49}\), 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 \(^{48}\) 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 \(^{48}\).
Other studies in humans \(^{31, 50}\) show an increase in collagen concentration and tensile strength as a result of eccentric training. MacDougall et al. \(^{50}\), examined seven male subjects after five to six months of heavy resistance training and noted an increase in strength, and cross sectional area in triceps brachii. Pousson \(^{31}\), also 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. \(^{51}\) examined the tensile stiffness of the patella tendon in the elderly population. Fourteen elderly patients exercised isotonically three times per week to load the patella tendon. Subjects were tested at baseline and again after 14 weeks of training. The stiffness of the patella 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 \(^{51}\).

Some studies \(^{52, 53}\) however, have found no change in tensile strength or stiffness after training. Kiiskinen \(^{52}\), studied immature mice and noted no change in tensile strength,
despite increased dry weight of Achilles tendon following 5-7 weeks of physical training. Woo, 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.

Earlier work from Purslow and Trotter 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. 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. Several authors 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.

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. 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 reliability during continuously contracting slow twitch muscle, while the arrangement of titin in fast twitch fibres optimizes powerful contractions. Several authors have proposed that the increased passive tension seen in slow twitch muscle is due to other intra-sarcomeric proteins such as desmin and dystrophin and other components of the dystrophin glycoprotein complex.

A recent study by Prado et al utilised 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.

2.4.4.3 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. Loren et al 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 known to influence the biomechanical properties of muscle. Walmsley & Proske, 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. Ljung et al, 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\textsuperscript{71} 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 +/- 0.05\( \mu \text{m} \)) when compared with normal subjects (2.20 +/- 0.04\( \mu \text{m} \)). 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.

### 2.4.4.5 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 \cite{Akazawa2005} 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 \cite{Akazawa2005}. Stiffness can also be augmented by the activity of synergists (e.g. scapular stabilisers), and is dependent upon the background torque \cite{Nielsen2012}.

Louie and Mote \cite{Louie2013} 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 \cite{Nielsen2012}, 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.

2.4.4.6 **Extrinsic mechanisms which mediate stiffness**

Sinkjaer and Hayashi \cite{Sinkjaer2006}, 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.\textsuperscript{4, 40, 41, 75, 76}

Johansson et al\textsuperscript{77}, provided evidence to support the role of joint receptors in mediating muscle stiffness at a joint via the gamma-muscle-spindle system. It was hypothesised 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\textsuperscript{77}. 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\textsuperscript{77}. The use of the joint receptors in such a manner, relies upon previous experience\textsuperscript{78}. 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\textsuperscript{79}. 
Smith \(^{80}\) 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 \(^{81}\), provided experimentally derived evidence to support the proposal of Smith \(^{80}\). 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 \(^{77}\). Swanik et al \(^{82, 83}\), investigated the stiffness, flexibility and EMG activity during a landing task in females with a deficient ACL. The ACL–injured group had a significant increase in preparatory activity in the lateral hamstring before landing compared with the controls. Females with deficient ACL had less hamstring stiffness and greater torque development for knee flexion. This study
supports others\textsuperscript{84-87} suggesting the presence of pre-programmed muscle activation strategies in order to compensate for injury in the limb.

2. \textit{Extrinsic mechanisms mediating stiffness and functional outcomes}

There are also numerous studies\textsuperscript{4, 88, 89} illustrating the importance of an interaction effect between stiffness, function, muscle activation timing and activation by central processing. McNair et al\textsuperscript{4} 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\textsuperscript{90} 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\textsuperscript{90}.

More recently Bryant et al\textsuperscript{91}, 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\textsuperscript{91}.
The relationship between stiffness and performance in eccentric, isometric and concentric activities in the upper limb was examined by Wilson et al. 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. Several other authors have also provided evidence that increased stiffness improves performance and functional outcomes due to increased rate of torque development and increased stability.

Thus a great deal of evidence exists to support the proposal that increased stiffness enhances 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 requires motion in three degrees of freedom, has a heavy reliance upon muscle activity for stability and requires a large ROM for 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 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, 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. 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. 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 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. Thus while investigations into passive stiffness in the shoulder
provides 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. 95 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 78, 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 than females in the respective passive and active conditions. Generalised joint laxity and strength were shown to predictors of 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 \(^7\), 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 \(^{96, 97}\). There are no previous studies investigating active shoulder stiffness in unstable shoulders during perturbations in horizontal extension. Given that previous studies \(^{98}\) 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 \(^{99}\), subscapularis atrophy \(^{100, 101}\), increased biceps latency and suppression of pectoralis major activity \(^{98}\), 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 \(^{102}\), some authors \(^{1, 2}\) 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 \(^{13, 27, 30, 44}\). 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 \(^{7}\) 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 relationship between stiffness in the unstable shoulder and function and quality of life also merit investigation.
References


