Study of the scapular muscle latency, shoulder kinematics and muscle activity in people with and without shoulder impingement

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Abstract

Background and significance: Shoulder impingement is a common shoulder pathology which is associated with changes in kinematics and muscle activity around the shoulder joint. The changes in muscle activity are theorized to be caused by changes in motor program strategies controlling the smooth and coordinated movements at the joints. Changes in muscle latencies, especially feed forward contractions, indicate alterations in these motor control programs. The purpose of the study was to assess for differences in the latencies and deactivation times of scapular muscles between subjects with and without shoulder impingement. **Research Methods:** Twenty five healthy subjects and 24 subjects with impingement were recruited. Scapulothoracic and glenohumeral kinematic data were collected using an electromagnetic system. Simultaneously myoelectric activities using surface electrodes from upper trapezius, lower trapezius, serratus anterior and anterior fibers of deltoid were collected as subjects raised and lowered their arm in response to a light cue. Data was collected during unloaded, loaded and after performing repetitive arm raising motion conditions. **Analysis:** The ratios of the number of feed-forward contractions during trials were compared by chi square analysis across groups and conditions. The other variables were analyzed using 2 or 3 way mixed model ANOVAs. **Results**: The percentage of trials showing feed forward contractions was higher for upper trapezius and lower trapezius in the unloaded condition and lower for serratus anterior in the condition after repetitive motion for the subjects with impingement as compared to healthy subjects. Subjects with impingement also demonstrated significantly earlier contraction of upper trapezius and an earlier deactivation of serratus anterior during lowering of the arm as compared to the healthy subjects. All subjects exhibited an earlier activation and delayed deactivation of lower trapezius and serratus anterior in conditions with a weight held in hand. The study found decreased scapular upward rotation, decreased posterior tilt and a less anterior plane of elevation in combination in subjects with impingement using logistic regression analysis. No significant group differences were found for muscle activity as a percentage of the reference contraction. **Discussion and conclusions**: The subjects with impingement showed some significant differences for muscle activation and deactivation times to indicate differences in motor control strategies. Rehabilitation measures should incorporate appropriate training measures in tandem with strengthening and stretching exercises to focus on improving movement patterns and muscle control.

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CHAPTER 1

INTRODUCTION

Background and significance: Overview of shoulder impingement

The shoulder joint complex consists of the sternoclavicular (SC), acromioclavicular (AC) and the glenohumeral (GH) joints. It enjoys greater range of motion than any other joint in the body. However, this high mobility results in challenges to the static and dynamic stabilizing structures of the shoulder complex. The motion at the glenohumeral joint depends on the smooth, coordinated motion of the scapula[1, 2]. The scapular motion, in turn, occurs due to the rotations at the SC and AC joint. Consequently, the shoulder depends on numerous muscles not only for generating these large amplitudes of motion but also for contributing to its dynamic stability during motion.

Shoulder impingement is defined as the compression and mechanical abrasion of the rotator cuff structures as they pass beneath the coracoacromial arch during the elevation of the arm[3]. The coracoacromial arch consists of the coracoid process, acromion, coracoacromal ligament and the AC joint. Traditionally, the contact of the soft tissue structures with the arch during motion was termed as impingement[4]. It is now more specifically termed as subacromial or external impingement. With the increased use of arthroscopy, tears and abrasions were identified on the under-surface of the rotator cuff tendons[5]. This contact of the soft tissue structures against the glenoid or labrum was termed as internal impingement. This entity can be clinically found on both anterior and posterior aspects though the latter has been strongly related to excessive humeral external rotations required during certain sporting activities such as baseball[6].

The large number of shoulder related problems necessitates an understanding of the mechanisms for altered kinematics for better design of treatment strategies. The most common etiological basis for rotator cuff tendinopathies is considered to be repetitive trauma[4]. Certain populations exposed to such cumulative trauma injuries include sportsmen and workers involved with overhead work, musicians and people using wheel chairs[7-9]. In an extensive survey about shoulder related problems, the authors found that impingement was the most common diagnosis[10]. Neer[4] described impingement as an encroachment of the supraspinatus outlet area which could progress to rotator cuff tears. An MRI study in asymptomatic individuals revealed that 50% of the population above 60 years of age have partial or complete rotator cuff tears[11]. When symptomatic they are a source of much functional limitation for the patient and may necessitate operative interventions.

There have been several studies which have tried to associate the deviations or alterations in the “normal” pattern of scapular or glenohumeral motion with shoulder impingement[12-16]. The study of these motion related abnormalities helps to describe probable mechanisms which may be associated with either causing or aggravating shoulder pathology. Nonetheless, according to another viewpoint, the alterations in movement may arise only as a compensatory mechanism to the pain and shoulder dysfunction[17].

Shoulder kinematics in healthy subjects

Scapular motion was traditionally measured in 2-dimensional (2D) studies during arm elevation. These studies aimed to measure the scapular contributions, especially upward rotation during the scapulohumeral rhythm. However, it is now well accepted that the scapular motion needs to be measured in all three dimensions especially when shoulder pathologies are associated with motions other than upward rotation. There have been studies to support the theories which associate certain scapular and humeral kinematics variables with the decrease in subacromial soft tissue clearance space. Some of them include an increased anterior tilt[18], increased upward rotation[17] and decreased humeral external rotation[19].

There is growing evidence that proves that the scapula usually upwardly rotates and tilts posteriorly as the arm elevates[2, 20, 21]. The descriptions about the scapular approximately vertical axis rotation (internal/external rotation) have been the most variable. As it is difficult to accurately measure scapular rotations at arm elevation angles beyond 120 degrees with surface sensors[22], there is less information about the pattern of motion throughout the end range of motion. Though, it has been demonstrated that the scapula externally rotates after arm elevation reaches around 90-110° of elevation by fixing sensors attached to pins inserted into the scapula[2, 21]. The clavicle shows a pattern of increased elevation, retraction and posterior long axis rotation as the arm elevates[2, 21, 23, 24]. At the same time, the AC joint demonstrates increased internal rotation, posterior tilt and upward rotation[21,25]. There is evidence for increased external rotation at the glenohumeral joint during arm elevation which is believed essential for clearance of the greater tuberosity away from the coraco-acromial arch[19, 21, 26].

Shoulder kinematics in shoulder impingement

To obtain a better understanding of the relationship between motion and shoulder impingement, the descriptions of the motions at these joints have also been studied in patients. Some differences seen relatively consistently across different studies involving subjects with impingement include an increased clavicle elevation and decreased scapular posterior tilt[12-14, 27]. The discrepancies arise when some studies report an increase in upward rotation[13] and others report a decrease in upward rotation values[12, 14, 27] for the impingement population. With the variability associated with the relation of scapular upward rotation to subacromial space reductions, it is unclear whether these deviations in patients are mechanistic causations to impingement or a positive compensatory change in patients. Other reported differences include an increase in scapular internal rotation[12, 16] in people with impingement.

The differences are not consistent across the kinematics studies which may be related to different methodologies and subject sample selections. The impingement syndrome consists of a very broad spectrum of disorders with a clinical inability to completely differentiate its overlap with glenohumeral instability and partial rotator cuff tears[28]. Hence, there is always a risk of analyzing a heterogeneous group of patients. Moreover, the etiology of impingement syndrome is multi-factorial. The possible causes for the presentation of symptoms range from anatomical factors such as abnormal acromial morphology[29] or AC osteoarthritis, to tightness in the pectoralis minor muscle[30], tightness in the posterior capsule[31], intrinsic cuff failures (degenerative changes of tendons), glenohumeral instability or symptoms secondary to micro trauma associated with repetitive strain or fatigue. It has also been challenging to screen subjects with impingement for either only subacromial or only internal impingement as there is a lack of sensitive clinical testing measures and both group of patients present with similar clinical symptoms. Furthermore, until recently there was less documented literature available for clinically evaluating scapular motion abnormalities or dyskinesia in subjects to allow inclusion of only those patients who may have a pathology associated with abnormal scapular kinematics. Hence there is a variability seen in the literature regarding the outcomes of studies which have compared scapular kinematics in people with and without impingement.

Nevertheless, the increase in clavicle elevation is a consistent finding across studies[13, 14]. It may be associated with the increase in upper trapezius (UT) activity also found across various studies[12, 32, 33]. Other consistent electromyographic findings across studies have been decreases in activity in serratus anterior muscle in people with shoulder dysfunctions[12, 33-35]. The arguments suggested for the associations of muscle activity changes in people with impingement include changes in muscle strength secondary to pain or fatigue, structural deficits due to tendon tears; and altered motor control strategies in patients.

Muscle activity in shoulder impingement

The upper trapezius activity found during the most studied motion of arm elevation does not exceed 25-30% of maximum voluntary contraction under unloaded conditions[12, 32]. These values are small enough not likely to be affected by strength differences across groups. However, these may be suggestive of a change in motor control strategy in patients which contributes to altered kinematics while using their arm overhead. Also, studies which involved repetitive shoulder motion in healthy and impingement populations, found that though strength deficits occur in both groups equally, kinematic changes were more dramatic in people with impingement[36]. Some studies have shown changes in kinematics[37] and improvement of strength[38] in subjects with full thickness rotator cuff tears after pain relief obtained by subacromial injections which suggest that pain seems to be an additional contributory mechanism to change in muscle activity rather than structural deficits. Pain has been associated with inhibition of muscles and changes in motor programs such that patients use altered movement patterns[39-42].

Physical therapy management of shoulder impingement related pain and dysfunction is often focused on stretching the tight structures (pectoralis minor/posterior capsule) and strengthening of other muscles (rotator cuff, lower trapezius and serratus anterior)[43-46], though no conclusive studies suggest weakness of these muscles. There has been some anecdotal evidence about lack of scapular muscle control and its association with shoulder pathology[45, 47]. Repetitive motion tends to aggravate the problems associated with extrinsic compression[48] and it is also postulated that repetitive motion makes the lack of scapular muscle control more visibly apparent[49].

Relevance of the proposed study

One of the few ways in which muscle/motor control is studied is through the study of muscle activation and deactivation time. This information along with the knowledge of the muscle amplitude through the range of motion can provide insights into any muscle inhibition or motor control abnormalities. Though previous investigations have made contributions to the issue of muscle latency in people with impingement, they are constrained by inadequate power and lack of comparative analysis of scapular and glenohumeral muscle latencies[50-52]. The study of the glenohumeral muscle (deltoid) latency can provide additional information about the relationship between the prime mover and the scapular stabilizer muscles. Also, to the best knowledge of the author, the effect of loading and repetitive motion has not yet been studied on motor latency of scapular muscles, especially in people with impingement. The primary purpose of the proposed study is to identify differences, if any exist, between relative muscle latency for trapezius and serratus anterior in patients with impingement syndrome as compared to an asymptomatic control group. Secondarily, the proposed study shall provide combined information about the relative amplitudes of muscle activity and shoulder kinematic descriptors for people with and without impingement during loaded arm conditions and after repetitive motions.

Hypotheses:

1. Under all conditions, the latencies of scapular muscles (upper trapezius, lower trapezius and serratus anterior) as compared to anterior deltoid will show a feedforward contraction only for healthy subjects and not for people with impingement syndrome.
2. The absolute latency of the muscles as measured from the light stimulus will be affected by group and condition as follows:
    a. The absolute latency of serratus anterior and lower trapezius will be significantly higher in people with impingement as compared to healthy individuals.
    b. The absolute latency of all muscles will be significantly delayed in both groups after repetitive motion as compared to the unloaded condition.
    c. Lower trapezius absolute latency will decrease with loading as compared to the unloaded condition of the arm in both groups.
3. The relative latency of serratus anterior, upper trapezius and lower trapezius as measured from the onset of anterior deltoid will be affected by group and condition as follows:
    a. The serratus anterior activation will be followed by a significantly slower activation of upper trapezius and then by lower trapezius in healthy individuals under all conditions.
    b. Under the loaded condition, the relative latency of scapular muscles will significantly decrease as compared to the unloaded condition for subjects with impingement.

    c. Across conditions, there will be a significant delay in relative latency of serratus anterior and lower trapezius and significantly shorter relative latency of upper trapezius in people with impingement as compared to healthy subjects.

4. The angular value of humeral elevation when each scapular muscle will be deactivated will be significantly lower in healthy subjects as compared to people with impingement. The difference between groups will be significantly lesser for loaded conditions and after repetitive motion.
5. Under the unloaded condition, there will be differences observed in kinematic descriptors for scapular tilt, internal rotation and upward rotation between subjects with and without impingement. Subjects with impingement will show decreased upward rotation, increased internal rotation and decreased posterior tilt in both elevation and lowering phases.
6. After repetitive motions, the scapulohumeral rhythm will show differences across groups such that there will be a higher slope of the regression line between glenohumeral elevation and scapular upward rotation in subjects with impingement after repetitive motion.
7. The EMG of the muscles will be affected by group and condition in the following ways:
    a. Under all conditions serratus anterior will show significantly decreased activity as a magnitude percentage of referenced contraction over motion increments from 30°-60°, 60°-90° and 90°-120° in subjects with impingement as compared to healthy subjects.

b. The lower trapezius will show significantly decreased activity in people with impingement as compared to healthy subjects during the lowering phase for the unloaded condition.

Definition of terms

1. **Dyskinesia**- Dyskinesia will be defined as an immature or excessive elevation or protraction or non-smooth motion during arm elevation, or the posterior prominence of the medial border and/or inferior angle.
2. **Muscle onset**- The instant (beginning of the 25 msec. period) when the average muscle activity exceeds the baseline activity (measured for 50 msec. before the trigger/light cue) by 3 standard deviations and is maintained till the muscle deactivates in the lowering phase.
3. **Absolute Latency –** The time period (msec.) for the muscle to get activated after the light signal is triggered.
4. **Relative latency**- The time period (msec.) between the onset of anterior deltoid and the referenced muscle.
5. **Feed forward contraction**- The onset of a muscle will be termed as feed-forward if it has an onset before or up to 50 msec. after the onset of anterior deltoid.
6. **Deactivation time-** The instant (beginning of the 100 msec. period) when the muscle activity falls below the sum of the mean and 3 standard deviations of the baseline activity (measured for 50 msec. before the trigger) and is maintained there after.

CHAPTER 2

LITERATURE REVIEW

The “Critical Zone” of the Supraspinatus Tendon

Other than motion related abnormalities, one of the commonly proposed mechanisms of development of supraspinatus impingement is lack of adequate blood supply to its tendon insertion. The earliest studies[53-58] involved microangiography and histochemical methods in cadavers to investigate the absence or relative dearth of vessels around the insertion site which was termed as the ‘critical zone’. Rathbun and Macnab[54] concluded from their cadaver study that the supraspinatus tendon insertion is better perfused in an abducted position than in a rest position. Lohr and Uhthoff[58] found that the articular side of the supraspinatus tendon was more sparsely perfused than the bursal side. These studies are questioned as they study distribution of dye in arteries instead of actual blood flow in vivo. The presumption that all small arterioles/capillaries get filled with the dye and can be adequately seen or measured is questionable.

Recently Rudzki and colleagues[59] have shown, in vivo, age and exercise related changes in the vascularity of the supraspinatus tendon as measured by contrast-enhanced ultrasound characterization of vascularity. They studied young and older (>40 years) healthy volunteers during rest and after exercise. They found an age related decrease and post-exercise increase in blood flow to supraspinatus. In a different analysis of the same data, Adler and colleagues[60] found differences in the areas of the tendon itself such that the medial articular side was least perfused as compared to lateral articular, medial and lateral bursal sides. These differences were increased after exercise. Levy and colleagues[61] measured the blood flow in healthy subjects and in people with impingement and rotator cuff tears intra-operatively using laser Doppler flowmetry. They found a significantly lesser flow in subjects with impingement than healthy controls and an increase in flow at the edges of the torn tendon. However, they did not find any area of hypoperfusion or any critical zone in the tendon. This method allowed the authors to see the flow in very small branches which normally would have been missed in the injection studies in cadavers. Swiontkowski and colleagues[62] conversely found that there is a hyperemic response to impingement which was attributed to the possible repair mechanisms of the body.

Overall, it can not be conclusively said that the hypoperfused or hypovascular area, if present, is directly associated with increasing the risk for development of tendon degeneration. Also, repetitive trauma to the tendon would most likely occur at higher elevation angles (at least beyond 30° of arm elevation[63]) when perfusion has been found to be better, which further weakens the argument for relating lack of blood flow to any subsequent tendon injuries.

The next few paragraphs describe normal shoulder kinematics and the changes seen in subjects with impingement.

Normal Kinematics

The scapula moves over the thorax during arm movement to orient the glenoid with the humeral head and to help maintain the length-tension relationship of the rotator cuff muscles such that it optimizes their function during arm motion[45, 47]. The focus of the early studies dealing with scapular motion was limited to describing the scapulohumeral rhythm during motion. Amongst the 2-Dimensional techniques, many studies were done by taking serial X- rays[15, 64-66] during shoulder motion. The technique involved static positioning of the arm at certain levels of humeral elevation .Therefore such studies are limited by static analysis which may not be completely representative of dynamic motion. They are also limited by miscalculations due to projection errors[67], and an inability to describe scapular motions other than upward rotation. In spite of the difficulties to make comparisons amongst these studies due to selection of different bony landmarks to define angular values and study of variable planes of motion, the results of the studies show a pattern of increasing scapular upward rotation with arm elevation. The classical description by Inman et al.[1] claimed a ratio of 2:1 between GH and ST motion for the complete range of motion which was later found to be different across portions of the range of motion[15, 66] and across different loading conditions of the arm[68]. These studies had focused on the motion of the scapula on the trunk which is not a true anatomical joint. Dvir & Berne[65] proposed that through the clavi-scapular link, the scapular motions are actually a combination of sternoclavicular and acromioclavicular joint motions. Nevertheless, due to practical implications to measure the AC joint motions and the difficulty to appreciate it clinically, the motion of the scapula has been traditionally defined with reference to the anatomical axes of the trunk even in 3-dimensional (3-D) studies. The scapula has been described to have an ability to upward/downwardly rotate about an approximately antero-posterior axis, anterior/posteriorly tilt about an approximately medio-lateral axis and internally/externally rotate about an approximately vertical axis[20-22, 27]. A study by Ludewig et al.[20] investigated 3-dimensional scapular motion and muscle activity at static humeral positions. The study included 25 healthy subjects. Motion was analyzed at static positions of rest, 90°, and 140° of humerothoracic elevation during scapular plane abduction after the subject was fixed to a control system to prevent changes in trunk position. Surface EMG was also recorded during motion from trapezius, levator scapulae and serratus anterior and analyzed as percentages of their maximum voluntary contractions. The study revealed that the scapula shows a pattern of increase in upward rotation, posterior tilting and external rotation as humeral elevation progresses. Also, the muscle activity progressively increases. The study makes important contributions but is constrained by static analysis and measurement at limited angles of arm elevation.

Another 3-dimensional analysis study with 25 healthy subjects was undertaken by McQuade et al.[69] to analyze the scapulohumeral relationship. Scapular plane abduction was studied during passive, active and loaded conditions. The scapular upward rotation and humeral elevation values were used to plot a relationship between the scapular and humeral contributions across different phases and loads. The angular phases during arm elevation were calculated as percentages of maximum arm elevation achieved by each individual. The inferences drawn from the results support the premise that scapular contributions increase with loading during arm elevation. This has been supported in other 2-D studies[68] and 3-D studies[70, 71].

There are always issues related to limited accuracy of skin fixed sensors. To overcome this limitation, researchers have used pins to attach sensors[2, 21, 72] or inserted tantalium balls[73] into bones. McClure et al.[2] used an electromagnetic sensor fixed to pins drilled into the scapula to track its motion. The study was conducted on the non-dominant sides of 7 out of the 8 healthy individuals where the kinematics of scapula and clavicle were analyzed during scapular plane abduction, flexion, and during humeral axial rotation at 90 degrees elevation. Data was interpolated at 5 degree increments and was averaged over three trials. The results inferred from the study[2] support the results from the earlier study by Ludewig et al.[20] which suggests that the scapula rotates upwardly, rotates externally along the vertical axis and tilts posteriorly as the arm elevates. The curves for scapular tilting and external rotation were curvilinear with an increase in slope predominantly after 90°, whereas, comparatively, upward rotation curves increased linearly with arm elevation. There were some slight differences (less than 5°) between the elevation and the lowering phases of motion. Differences between elevation and lowering of the arm have also been studied by Borstad and Ludewig[74] in people with and without impingement. They found similar differences (<5°) between groups, more during the lowering phase at higher elevation angles for scapular internal rotation and anterior tilt positions.

The translations of the scapula seen as upward-downward motion during shoulder shrugging are actually clavicular motions of elevation-depression. Similarly, apparent medial lateral translations of the scapular medial border are brought about by protraction/retraction motion at the sternoclavicular joint. The most predominant clavicular motion during arm elevation is however the long axis rotation of the clavicle[1, 21,75]. The sternoclavicular motions describe a pattern of increase in clavicle elevation, retraction and posterior rotation as the arm elevates[2, 21, 23, 75]. McClure et al.[2] studied the clavicular angles of elevation/depression and protraction/retraction indirectly by tracking the sternal notch and acromioclavicular joint during scapular plane elevation and flexion motions. The change in clavicle elevation position from rest to peak arm elevation was found to be around 10 degrees. The clavicle retraction (around 16 degrees change in position from rest to peak elevation) occurred predominantly at higher angles. Fung and colleagues[23] inserted pins into the bones of cadavers and found similar directions for clavicle motion during passive humeral elevation as found by the earlier study, however, the study found more curvilinear patterns. They also compared the rotations for arm elevations along different planes and found a trend of increased clavicle rotations for abduction than for scapular plane elevation or flexion. Ludewig et al.[75] described the clavicular data using surface markers across different planes and elevation angles in healthy people without shoulder pathology. The description is limited to 110 degrees of humeral elevation as further surface tracking would be rendered inaccurate due to skin-slip issues. Clavicular protraction/retraction was found to be the most variable motion. Ludewig and colleagues[21] tracked the clavicular motions by inserting pins into the bones of healthy subjects. There are differences as measured during active motion in this study and passive motion studied in cadavers by Fung and colleagues[23]. This study found that the clavicle motion showed a more linear change across elevation angles. The change in clavicle posterior rotation position was found to be most consistent across subjects and measured around 30 degrees. A study by Sahara et al.[25] found similar results using a vertically open MRI. They found that during arm abduction, the clavicular motion relative to the lung (trunk reference frame) showed 31° of retraction, 7° elevation, and 33° of posterior axial rotation.

The effect of clavicle rotations about the 3 axes does not transform directly into the same axis scapulothoracic rotations due to the clavi-scapular angles (~70 ° in the transverse plane). An increase in clavicle elevation angle contributes less to scapular upward rotation and more to anterior tilt[76]. Clavicle posterior rotation contributes mostly to scapular upward rotation and slightly towards posterior tilt. And lastly, clavicle retraction perhaps contributes mainly to scapular external rotation[76].

Owing to the difficulties associated with tracking clavicle motion, there are fewer studies that describe AC joint motion. Sahara et al.[25] tracked the clavicle and scapula during arm abduction in multiple static positions using a vertically open MRI. The study included 7 people who were scanned bilaterally as they moved their arm from a position of rest to maximum elevation. To describe the motion using Euler angle conventions, anatomical coordinate systems were defined from the 3-D reconstructions of the bones. The study found that the AC joint showed 16° of protraction (internal rotation), 22° of upward rotation, and 22° of posterior tilting during abduction. The authors also used the screw axis method in another study and described that the axis passed through the AC joint and coracoclavicular ligament during arm abduction[24]. Though static analysis was performed, the study makes important contributions to describe accurate AC joint motions. One of the recent works by Ludewig et al.[21] used bone pins to quantify the AC joint motion in 3 dimensions. The AC joint showed a pattern of increasing internal rotation, upward rotation and posterior tilt as the arm elevates. A trend towards increased AC internal rotation during flexion as compared to elevation in other planes was found[21]. Likewise, increased upward rotation and posterior tilt occurred during abduction as compared to other elevation planes[21]. The AC joint internal rotation can be viewed as an offset to the scapulothoracic external rotation achieved due to clavicular retraction such that the scapula can move smoothly over the thorax.

There is less literature describing the glenohumeral motion as compared to the descriptions of the humerus with reference to the trunk as the latter motion is better clinically appreciated. Further, accurate representation of axial rotation of the arm may be limited with surface sensors[77]. The humerus external rotation is considered important for clearance of the greater and lesser tuberosities under the coracoacromial arch as the arm elevates[19]. In a recent study using bone pins[21], it was found that glenohumeral external rotation is considerably larger during abduction as compared to flexion and scapular plane abduction. Also, there were differences observed in the amount of humeral elevation with respect to the scapula at fixed humerothoracic angles, suggesting that scapular contributions differed across different planes of arm elevation. Scapular contributions were greater for abduction than for flexion.

Overall, the available literature provides information about shoulder joint motion across different planes and elevation angles. There is considerable evidence that proves the scapular motion of upward rotation (~35°) and posterior tilt (~20°); clavicle long axis posterior rotation (~30°), elevation (~5°) and retraction (~15°); AC joint internal rotation (~8-10°), upward rotation (~8-14°) and posterior tilt motion (~10-18°) as the arm is elevated. But, there is less conclusive information regarding scapular internal/external rotation. This may be due to the inherent high variability for this motion seen in the population. The difference across planes appears as such that flexion requires slightly less scapular contributions, less clavicle elevation and axial rotation and less humeral external rotation as compared to abduction[2, 21, 23].

Kinematics and shoulder impingement

The main reason for which scapular motion is studied in subjects with shoulder related pathologies is the proposition that abnormal motions could compromise the subacromial space furthering mechanical abrasion of soft tissue as they pass under the arch or come in proximity to the glenoid[78]. The relationship between kinematics and the subacromial space has been studied by a few authors using MRI and contact force measurements. Solem-Bertoft and colleagues[18] measured the acromion-humeral distance and acromial angle (angle between the acromion process and the horizontal in the sagittal plane) in positions of scapular protraction/retraction. They placed sandbags under the scapula and between the scapulae to impose these positions which actually produced SC joint motions of protraction-retraction along with scapular anterior-posterior tilting. The findings suggest that the subacromial space reduces when the acromion tilts anteriorly as a result of a passively imposed scapular protraction. The study is limited as the analysis was made with only one slice in each plane, a small sample size and the use of artificially imposed positions which may not be completely representative of actual postural or scapular position abnormalities. Flatow et al.[19] studied the acromion-humeral interval and soft tissue contact in cadavers using stereophotogrammetry. The experiment included artificial muscle torque generation using cables attached to the tendons. The study concluded that contact forces develop on antero-lateral aspects of the acromion early in the range of humeral elevation and this contact area moves more medially with progressive elevation. On the humeral surface, contact shifts to more distal sites of the biceps and supraspinatus tendon regions (insertion sites) with elevation. Also, maximum proximity between the humerus and acromion occurred around 60-120 degrees of arm elevation. This is in agreement to the painful arc of motion described by shoulder pain patients[3, 4]. They also examined the effect of humeral internal rotation on the contact areas. They found that there was an increase and shift towards the posterior and distal contact areas even during resting position.

Another study by Karduna and colleagues[17] measured the effects on the subacromial space by passively translating the humeral head superiorly during fixed scapular rotated positions. They found that scapular internal/external rotations or anterior/posterior tilt positions of ± 5 and ± 10 degrees from a "resting" scapular position did not make any significant difference in the amount displacement to a preset force. However, they found that the amount of clearance increased with an increase in scapular upward rotation. The study is limited by lack of control of initial position, static analysis in isolated rotated scapular positions and lack of description of where the actual forces are developing on the acromion. Possibly, the humeral head collided against medial aspects of the acromion and therefore more contact was seen in scapular upward rotation positions. Bey et al.[63] calculated the acromio-humeral distance 3-dimensionally using fluoroscopy in subjects who had undergone rotator cuff repairs. They found that this distance decreased with humeral elevation with a minimum distance at 60° of GH elevation (~90° of humerothoracic elevation). One surprising result was that the supraspinatus was closest to the acromion much earlier in the range (27 – 36° of elevation) than conventionally considered (60-120° of painful arc of motion).

The above mentioned studies help to describe the consequences of the kinematic alterations on the subacromial space/ acromion-humeral distance. The space is presumed to decrease with certain kinematic parameters such as scapular anterior tilting, glenohumeral internal rotation and increase in arm elevation from resting position. However, most of these studies are limited by their analysis of passive, static positions[17-

[19]. The studies help to corroborate certain clinical phenomenon such as the presence of a painful arc in patients and provide explanations for the mechanistic/compensatory kinematic differences observed in patients. There is an association, though inconsistent between alterations of scapular kinematics and shoulder pathology consistent with shoulder impingement. The next several paragraphs describe this association in patients, studied in multiple studies using 2-dimensional, 3-dimensional electromagnetic, topographical and imaging techniques.

Endo et al.[27] investigated scapular positions during shoulder abduction at 0, 45 and 90 degrees of humeral elevation in 27 people with unilateral shoulder impingement comparing their painful side with the contra-lateral pain-free side. They used routine antero-posterior radiographs for analysis. They found a significant difference between the sides with the impingement side showing decreased upward rotation (at 90° only) and posterior tilt (at 45° and 90°). The authors used a unique technique to calculate 3-dimensional angles from planar radiographs which are nonetheless subject to projection errors[67]. These errors tend to increase especially with antero-posterior plane radiographs as the scapular plane lies approximately 30-40 degrees anterior to the coronal plane.

The study by Ludewig et al.[12] used electromagnetic tracking with 52 male construction workers which included 26 healthy subjects and 26 patients with impingement syndrome. The subjects were matched for exposure to overhead work and other demographic variables. The subjects with impingement showed decreased upward rotation and tilting at the end of the 90-120 degree phase of scapular plane abduction; increased medial rotation under loading with 5 and 10 lb loads; increased trapezius activity especially under loading at higher angles of humeral elevation and decreased serratus activity across all loads and phases. A study by Lukaseiwicz and colleagues[14] compared 20 healthy people with 17 patients with impingement syndrome. Comparisons were made at static positions of 0, 90 and maximum angles of humeral elevation in the scapular plane by repeated digitizing of landmarks to form anatomical coordinate frames. The 3 rotational projection angles for the scapula were calculated by finding angular values between respective positional vectors. Scapular translations were calculated by finding the difference in position of the scapular centroid. The study revealed a decrease in amount of posterior tilt, and increased superior scapular translation in symptomatic subjects. The latter finding can be interpreted as an increased clavicular elevation position.

Hébert et al.[79] studied scapular behavior in healthy people and patients with impingement. They tried to quantify relative contribution of scapular motions in 3 dimensions to total scapular motion. The data was collected at rest and at static positions of 70°, 90° and 110° during flexion and abduction when the subject was fixed to a control system. No differences were observed for resting positions of scapulae between the groups. Differences amongst impingement subjects were noted for tilting values against normative data collected from healthy individuals.

McClure et al.[13] compared 45 subjects with impingement with 45 controls for a 3-Dimensional analysis of scapular and clavicular motion during elevation of the arm in the scapular plane and flexion. They found that at higher angles, there were increases in upward rotation, and clavicular retraction and no differences in amount of tilting angles in the impingement group. Though these results are divergent to previous literature, the authors believed that the changes in scapular kinematics seen in their sample were important compensatory motions. This reasoning was also supported by the study of Karduna and colleagues[17] where they had found an increase in acromial contact forces with increased scapular upward rotation. The differences between studies are possibly due to differences in sample populations of patients. In spite of this, the study ascertained increases in clavicular elevation in patients during arm elevation which is a common finding across studies and it is a commonly noticed phenomenon clinically as a shoulder shrugging attempt during arm elevation[14, 80].

The different results seen across different studies may be attributed to different methodology and sample selection techniques. The literature regarding the accurate classification and diagnosis of impingement is ambiguous[81]. Shoulder impingement syndrome remains a wide umbrella of disorders which may include inflammation or degeneration of various tendons, partial rotator cuff tears or bursitis[3, 81]. As more recent studies reveal, there are differences in the site of abrasion (external/subacromial versus internal), location of impingement (anterior versus posterior), and mechanisms for pathology development (extrinsic versus intrinsic)[81]. These differences are further associated with different contributory mechanisms of causation ranging from anatomical causes[29], motion related mechanisms[12, 13, 27], and tissue property differences[48, 82]. These differences in the mechanisms, locations, and presentation of impingement pathology make the design and clinical sample selection criteria difficult. Also, interpretation of study results is complicated by the heterogeneity of the clinical population. Subsequently this can make the application of the results into designing interventions difficult.

It can be concluded that the subjects with impingement show kinematic differences of increased scapular anterior tilt and increased SC joint elevation[12-14, 27]. The scapular upward/downward rotation results differ across studies[12, 13, 16]. The differences in kinematics seen in patients may relate to differences in muscle action, strength and flexibility of soft tissues. It is therefore important to have an understanding of the muscles which move, restrict or control dynamic shoulder motion. The next section describes the role of different muscles in shoulder motion and the alterations of activity in pathological conditions.

Muscle action and kinematics

The contributions made by muscles have been studied in the past using various approaches including cadaver studies to enhance the knowledge of the anatomical and biomechanical functions of muscles[83], comparisons between active and passive arm motion[69, 84], study of EMG activity in muscles during arm motion[1, 12, 85], study of motion in patients who have nerve injuries[86, 87], kinematic studies after experimentally removing a particular muscle by nerve blocks[88] and study of shoulder models to calculate muscle moment arms (unpublished study). All the different approaches have helped to refine our knowledge about the functions of muscles. In shoulder related research, some authors have studied the kinematics and EMG activity simultaneously to relate the functional status of the muscle with movements of the scapula. The literature has been variable with different positioning of electrodes[12, 32, 89-91], normalization techniques[91,92], and poor controls for the factors associated with interpretation of EMG data such as length of muscles, velocity of contraction and type of contraction[93].

Despite using various approaches, there has been a general consensus regarding the functions and roles of scapular muscles during arm movements. The trapezius and serratus anterior have been recognized as muscles which can rotate and stabilize the scapula[65, 83, 85, 90]. The knowledge about their roles and dysfunction in shoulder related pathologies provide potential guidelines to interventions aimed at improving shoulder motion and function.

Studies[87, 94] have found in patients with trapezius paralysis that the shoulder girdle "droops" and scapular downward rotation and lateral translation occur. Also, with lack of trapezius activity to stabilize the scapula, intact rhomboids and levator scapulae may be rendered inefficient to rotate the scapula[87]. Inman et al.[1] claimed that we need coordinated activity in scapular muscles for smooth movement of the scapula during arm motions. They studied raw EMG data from various muscles and confirmed that muscle activity in various glenohumeral and scapulothoracic muscles increases with elevation of the arm. Some muscles are believed to be the prime movers for the scapula (trapezius, serratus anterior) whereas others (rhomboids, levator scapulae, etcetera) synergistically act to provide a stable base of support for the glenohumeral muscles[1]. Trapezius which inserts on to the spine of the scapula and acromion process is ideally suited for scapular stabilization as the instantaneous center of rotation of the scapula on the thorax has been found to move from the root of the spine towards the AC joint, nearly along the line of trapezius insertion[85]. The role of trapezius has been further investigated by studies which have calculated the changes in the moment arms of trapezius and serratus anterior using computer models during arm elevation in different planes (unpublished studies). These modeling studies found that the upper trapezius primary capability is elevation of the clavicle at the SC joint whereas the middle and lower trapezius primary capability is external rotation of the scapula. The lower trapezius can upwardly rotate the scapula at lower angles of humeral elevation but does not have much tilting capability. This is in agreement with the EMG studies which show an increased EMG activity in lower trapezius at higher angles as the muscle gets into a physiologically and bio-mechanically disadvantageous position[85]. The serratus anterior was found to have maximum torque capabilities to upwardly rotate but it can also posteriorly tilt and externally rotate the scapula. Hence this muscle can be viewed as the prime mover for scapular motion during arm elevation. The role of serratus anterior has been established further in many studies which have analyzed EMG activity during sports activities[95, 96].

Wiedenbauer & Mortenson[97] found that upper trapezius was most active during scapular elevation (actually SC elevation); overall trapezius was more active during abduction as compared to flexion and lower trapezius activity peaks later during the range of motion. Bagg and Forrest[85] studied the EMG activity in the three parts of trapezius and serratus anterior during scapular plane elevation in 20 healthy male subjects. They averaged integrated EMG signal collected from the muscles during 5 trials performed at a predetermined speed of arm elevation. Simultaneous kinematic analysis was done using a camera to capture scapular and humeral motion. They plotted the EMG signal across the range of motion to analyze muscular activity. They found that the muscles show an increase in activity as the arm elevates with some plateauing in the mid range for the upper and middle fibers of trapezius and serratus anterior. The lower trapezius on the other hand showed little activity till later in the range. These findings were correlated with the biomechanical findings of the shift in the instantaneous center of rotation for the scapula. The authors proposed that the trapezius fibers act as scapular rotators during different parts of the range in accordance to the varying biomechanical advantage the muscle may get during motion.

Overall, the upper trapezius primarily acting on the SC joint can elevate the clavicle; the middle and lower trapezius primarily acting at the AC joint can externally rotate the scapula; and the serratus anterior can cause scapular upward rotation, external rotation and posterior tilt (unpublished studies)[83, 90]. These functions of serratus anterior make its contribution to scapular kinematics very significant against development or worsening of shoulder impingement symptoms. Whereas the over activity of upper trapezius can be considered detrimental to impingement symptoms as increased clavicle elevation is associated with scapular anterior tilting.

Changes in EMG activity with pathology

Peat and Grahame[33] investigated trapezius, serratus anterior and deltoid EMG in people with and without shoulder pathology. They found that the upper trapezius showed an increased activity during arm elevation and lowering and decreased activity in serratus anterior at some humeral elevation angles in patients as compared to healthy controls.

Scovaozzo et al.[35] used EMG to evaluate muscle activity during swimming motions in athletes with and without shoulder pain. The study revealed a decrease in the activity of the anterior and middle fibers of the deltoid, subscapularis and serratus anterior during different phases of the swimming motion for the subjects with shoulder pain. The authors propose that the muscles showed decreased activity either due to shoulder pain or that pain caused the athletes to use alternative motions and muscles during the action of swimming. It can be concluded that people with pain used a different motor program for the task using different muscle activation patterns.

In a study comparing EMG amplitudes across the range of motion in people with and without glenohumeral instability, the authors[98] found that serratus anterior activity was reduced across all planes of elevation and elevation angles. Lin and colleagues[32] studied the upper and lower fibers of trapezius muscle in people with and without frozen shoulder syndrome during static elevated arm positions. They found increased upper trapezius activity across all planes and angles (60° and 120°) and increased lower trapezius activity only at 120° of elevation during the make test in the patient group as compared to controls.

Kelly and colleagues[99] studied several shoulder muscles in people with symptomatic and asymptomatic rotator cuff tears and healthy controls during functional tasks. Similar to past literature[32-33], they[99] observed an increase in upper trapezius activity during elevation of the arm and carrying tasks. Also, they found an increase in activity in supraspinatus and infraspinatus amongst symptomatic patients whereas subscapularis was found to be more active in asymptomatic people. It was interesting to find differences in EMG activity between asymptomatic subjects who earlier had successful pain relief with conservative management and symptomatic patients. This may be indicative of a continued change in motor control of scapular muscles after the episode of pain. The increase in upper trapezius activity has been a consistent finding across studies[20, 32, 33, 99] which may be associated with the increase in clavicular elevation or scapular upward translation found in many clinical and kinematic studies[13, 14, 80]. This increased upper trapezius activation may be viewed as a common compensatory strategy used by people with shoulder pain and pathology to elevate their arm. Nevertheless, the increase in clavicle elevation may cause an increase in scapular anterior tilt which may be viewed as a mechanism to either cause or aggravate the impingement symptoms.

Finally, Lin[34] reported similar results while investigating functional tasks in a poorly defined shoulder pain patient group who showed an increase in upper trapezius activity and decrease in serratus anterior activity as compared to healthy controls. Other authors have surmised that serratus anterior weakness or reduced activity may contribute to secondary impingement syndrome in the shoulder and any improvement in the function of the serratus muscle may help alleviate pain and dysfunction[96, 100, 101].

Changes in muscle activity have been linked to changes in the subacromial space[102]. Graichen et al.[102] studied the acromiohumeral distance using an open MRI system during elevated positions of the arm with and without active abduction forces in people with and without shoulder impingement. They concluded that muscle contraction further decreased the distance in patients as compared to their contra-lateral side in an elevated arm position (90°) whereas there was no significant effect of muscle contraction on acromio-humeral distance in healthy individuals.

Thus across studies, changes in the activity of the scapular muscles, especially trapezius and serratus anterior, have been associated with various shoulder related pathologies. Other than structural changes, motor control alterations have been proposed as a cause for these muscle activity changes[44, 49]. These may include a deactivation of the muscle such that it fails to get recruited on time, and/or fails to maintain its activation through out the range of motion as required; or conversely any hyperactivity of the muscles. The study of temporal recruitment of muscles has been undertaken to explore this possibility of altered motor control in patients[39-42]. There is evidence to support this relationship of altered muscle activation and pathology in different patient groups and recently similar studies have been undertaken for the people with shoulder pain and pathology. The following paragraphs are an overview of these studies which look at scapular muscle latency and shoulder pathology.

Latency of scapular muscles

It is postulated that the force couples generated by the trapezius and serratus anterior help to maintain a smooth scapular pattern of motion during arm elevation. Also, trapezius contraction is believed to help maintain the path of the instantaneous center of rotation of the scapula on the thorax[64]. Thus, abnormalities in scapular motion may be associated with altered muscular control (recruitment / deactivation) caused by pain, chronic fatigue or micro-trauma.

There have been a few studies which have looked at the relationship of scapular muscle latencies during elevation of the arm[50-52]. All the studies known to the author at this time have different methodological approaches and use of small sample sizes such that statistical power is often inadequate. One of the earliest works was done by Wadsworth and Saxton[52] in swimmers, where they compared the latency of the upper fibers of trapezius (UT), lower fibers of trapezius (LT) and serratus anterior (SA). These were compared across both the painful and the contra-lateral shoulders in 9 swimmers with symptoms of shoulder impingement and healthy controls matched for height, weight, training mileage and skilled hand preference. The EMG from the muscles was recorded while elevating the arm in the scapular plane at a speed of 40° per second. The onset time was estimated when the muscles reached 5% of their maximum amplitude from the moment the motion was detected by an inclinometer attached to the arm. Serratus anterior activation was however detected based on visual estimation because of presence of cardiac artifacts in the signal. The electrodes were placed at locations of maximum muscle bulk. Though no significant group differences were found, the study revealed that muscle latency of SA was delayed bilaterally in patients and that there was an increased variability associated with muscle latencies in subjects with shoulder pain, as demonstrated by larger within and between subject variance. Also, the order of muscle recruitment was UT activation followed by SA and then LT activation. The study may have failed to find group differences due to inadequate power owing to the small sample size (n=9 for each group). Also, the study does not provide information about the relative latencies of the scapular muscles as compared to the deltoid. Relative latencies of scapular muscles with reference to glenohumeral muscles can help to find any mechanistic connections between delayed recruitment and abnormal kinematics such as the reverse pull of the deltoid on the scapula (These are described later in a separate section).

Another recent study[50] compared two groups with and without shoulder impingement for scapular muscle latency bilaterally during scapular plane elevation of the arm. They found the same order of recruitment as found by Wadsworth et al.[52] such that UT was first to be activated followed by SA, MT and LT. They did not find significant group differences which can be also attributed to low sample size (n=10) and inadequate power. The authors do not mention how they statistically handle the trial to trial variability or interactions between the factors. Nevertheless, they report that side to side differences were observed for the impingement group only. They also looked at isokinetic strengths of shoulder axial rotator muscles at 60°/second and 180°/second speeds within an arc of motion between 40° of internal rotation to 50° of external rotation. The authors did not find any significant difference between both sides or between groups of subjects. This may indicate no difference in isokinetic performance of the shoulder rotators or a lack of power to find any significant differences. They provide no details as to why they chose to compare the injured side of patients with the non-dominant side of controls and vice versa. The speed of motion which can be considered as a confounding factor for EMG analysis of latency was not controlled in the study and subjects moved at a self determined comfortable speed. The authors have overstated that the results obtained by their study for controls are similar to those obtained by Wadsworth & Saxton[52]. They have calculated the latency from the moment the subjects were shown a light source whereas Wadsworth et al.[52] had used the moment of initiation of arm movement. It is known that visual reaction time (that is, time from stimulus to movement initiation) is approximately 150ms-200ms[103] and therefore direct comparisons to different reference actions are more difficult to make. Even when adjustments are made for the visual reaction time, the latency values obtained by Wadsworth and Saxton [52] are considerably smaller than that obtained by Moraes et al.[50]

Similar study by Santos and colleagues[51] compared the latency of scapular muscles during scapular plane elevation of the arm. The study compared 8 healthy swimmers and 8 patients with shoulder instability. The patients had negative tests for impingement during clinical testing but had significant issues related to instability of the shoulder. The authors used a different technique to estimate muscle latency as the time required for the muscles to be activated from the moment of initiation of movement described as 5% of peak movement velocity. As defined by this criterion, trapezius, serratus anterior and deltoid showed activity before movement initiation whereas other muscles (triceps, latissimus dorsi, pectoralis major) did not. The study also looked at humeral translations and other kinematic variables based on position vectors of arm and trunk. The validity of estimating translations of the humeral head using these position vectors is debatable as no subject stabilization was done. The speed of the movement was not controlled and subjects were asked to elevate their arm as fast as possible. The authors do not discuss statistical power which could be contributing to the absence of significant differences between the small groups.

The latency of muscles has also been investigated during sudden perturbations which probably are more indicative of the description of reflexive protective mechanisms rather than any programmed motor control strategy. The earliest work was done by Cools and colleagues[104] who studied latency of the scapular muscles during a sudden adduction perturbation or what can be called as a sudden drop of the arm from a 90° elevated position in a group of healthy individuals. Muscle latency was defined as the time needed for the muscle to reach 10% of its maximum voluntary contraction from the time movement was detected. The study also looked at muscle latencies after inducing fatigue by repeated abduction-adduction motions. The results showed no difference between the latencies for the three portions of trapezius muscle fibers. Fatigue caused a significant delay in firing of all muscles except lower trapezius without changing the recruitment order. The order of activation found by the authors was UT activation followed by MT and LT activation. These all were preceded by the activity in the prime mover, that is, by middle deltoid activity. The protocol used for fatiguing the muscles can be viewed as primarily acting on the deltoid muscle and secondarily also on the scapular muscles. Again as no EMG or kinematic descriptors are available other than latency, it is difficult to translate the results to describe meaningful mechanistic connections or motor control strategies. The authors did a similar study[105] using the same protocol to find differences between athletes with and without impingement. A sample of 39 patients and 30 controls was selected. The study compared the relative latency of the trapezius muscle across the two groups and shoulder sides comparing the injured side and non-injured side of patients with the dominant and non-dominant side of the controls respectively. The study found group differences with a delayed middle and lower trapezius onset in patients. Also, the relative latency of lower trapezius was significantly longer on the injured side as compared to the non-injured side in subjects with impingement.

Comparisons between various studies which look at scapular muscle latency have been difficult due to the use of different methods and techniques. Some studies have used a predetermined percentage of maximum voluntary contraction as the point of onset of muscle activity. This method has been criticized as it is sensitive to the peak amplitude and the rate of rise of EMG signal between different muscles[106]. A few studies have used more reliable computer algorithms to detect muscle onset time but have used different parameters for smoothing data or limits of variance for detection of onset. Studies have also differed as comparisons are made to either an external source (light) or movement initiation as detected by an inclinometer or a percentage of peak velocity. Relative latency of muscles as compared to the prime mover has not yet been investigated for self-initiated active arm movements.

It is evident that different investigators have used different methods, velocity of motion, sample populations, and different criteria to estimate muscle latency which makes it extremely difficult to make comparisons across studies. Nevertheless, it can be concluded that the muscle latency of scapular muscles is affected in people with impingement syndrome such that it may show increased variability, or delay in activation which may cause a change in recruitment order[52, 105].

Effects of fatigue

Shoulder impingement has been strongly associated with repetitive motions[4, 107]. This is concluded from the high incidence of shoulder related problems in people who use repetitive shoulder motions during sporting activity or occupational work. Therefore, it is presumed that fatigue of the muscles may change the kinematics at the joint such that it leads to reduced subacromial space or increased abrasion. Repetitive motion is also considered to precipitate shoulder impingement in people who have primarily more intrinsic causes such as altered acromial morphology or degenerative tissue changes[107].

Ebaugh et al.[108] studied the effect of muscle fatigue induced with repetitive motion on scapular and GH motions. Twenty healthy subjects were asked to handle small objects in the hand in elevated arm positions, perform resistive arm elevation and resistive diagonal arm motions until they perceived tiredness and could no longer continue the activities properly. The study found that the fatiguing protocol caused increased scapular upward rotation, external rotation and clavicle retraction, and decreased humeral external rotation. The study design was such that both deltoid and scapular muscles were fatigued so it is difficult to evaluate what factor exactly precipitated the changes. They found that the GH muscles were fatigued to a greater extent and therefore it is possible that there is a shift towards increased reliance upon scapular contributions to raise the arm overhead. Tsai et al.[109] used repetitive motion to selectively fatigue the GH external rotators (infraspinatus and teres minor). It is difficult to understand the premise for studying scapular motion after fatiguing GH muscles. McQuade et al.[110] found that subjects demonstrated increased scapular contributions during arm elevation after a fatiguing protocol.

It is claimed that people who use their arm in repetitive motions fatigue their muscles over time which brings about changes to compensate for the weakness associated with fatigue[111]. Su and colleagues[36] studied the effect of a routine swim practice on scapular kinematics in swimmers with and without shoulder impingement symptoms. The authors used an inclinometer to measure scapular upward rotation at rest, 45°, 90° and 135° of humerothoracic elevation. These measurements along with scapular strength measurements were made before and after the swim practice. The results show that there was a statistically significant reduction of strength as measured by a hand held dynamometer in both groups for upper trapezius (13%) and Serratus anterior (14%). The scapular kinematics did not differ between groups before the swim practice but there were significant reductions in scapular upward rotations at 45°, 90° and 135° after practice in persons with impingement. These results indicate that though strength reductions were not drastic (< 15%), there were differences in kinematics during arm elevation after performing repetitive motion. The decrease in scapular upward rotation is contrary to other results obtained after a fatiguing protocol[108, 110]. These differences may be due to differences in the activities and their duration used for fatiguing the muscles, different methodology used for measurement (inclinometer versus 3-D surface sensors) and differences in the sample population (symptomatic versus. healthy). Also noteworthy is that though muscles of healthy individuals fatigued, they did not demonstrate the significant decrease in upward rotation as shown in persons with impingement. The authors suggest that the pain induced during the activity may have brought about the changes.

Along with anecdotal information[16, 47] and the results obtained from these studies, it can be concluded that repetitive motion may help to reveal otherwise subtle kinematic differences. Also, the muscle weakness associated with fatigue may be insufficient to directly contribute to changes in kinematics, but may be involved with changing the motor control to selectively reduce or increase activity in some muscles.

Scapular Dyskinesia

The scapula follows the curvature of the thorax during motion. As it moves over this curved base, there has been evidence that it may not follow a smooth pattern and may visually show excessive prominence of the medial border or inferior angle[49, 112]. This has been termed as ‘dyskinesia’ or ‘dyskinesis’ and may be present in people with or without shoulder pathology. In nerve injury patients involving the serratus anterior or trapezius, this is described as ‘scapular winging’. It has been postulated by many authors that scapular dyskinesia is a sign of scapular dysrhythmia or incoordinated motion[45, 49] and therefore it is related to shoulder pathology.

Poppen and Walker[15] used serial X-rays to find that differences in the scapulohumeral rhythm in healthy and symptomatic subjects and found no specific differences of trends between groups. Warner et al.[16] demonstrated by 'Moire' technique that scapular asymmetry/winging may exist in a healthy population but is a considerably more notable phenomenon in pathologic groups especially during the lowering phase of dynamic motion. It has been documented that the alterations in scapular kinematics may be associated with lack of motor control of the scapular muscles[47]. This could be associated with either muscle inhibition decreasing the torque generated by the scapular muscle for stabilization or a reorganization of normal muscle firing patterns around the shoulder[47]. Kibler suggested that scapular symmetry should be noted during rest and dynamic motion especially during lowering of the arm[49]. The presence of these altered or dyskinetic scapular motions may be elicited better after fatigue or under loaded conditions[47, 49, 113]. Kibler devised a classification for scapular dyskinesia with 3 different types for inferior border prominence, medial border prominence and excessive scapular superior translation. There was moderate reliability amongst investigators regarding the diagnosis of dyskinesia based on this scale; and the scale has also not been validated[113]. Another means of assessment includes analysis of scapular landmarks in multiple positions called the 'lateral slide test' which has been criticized as it does not analyze dynamic motion and hence is inefficient to identify more meaningful and functional motion related abnormality[47, 114].

McClure and colleagues[112] studied reliability for diagnosing scapular dyskinesia in a population of 142 athletes performing flexion with a load in hand. They defined dyskinesia as an immature or excessive elevation or protraction, non-smooth motion during arm elevation, or the posterior displacement of the medial border and/or inferior angle away from the thorax. Two investigators analyzed the subjects in person and 6 other investigators analyzed video-recordings of motion. They found moderate inter-tester reliability for the in person and video raters (kappa values = 0.57, 0.54 respectively). The authors also undertook a study to check the validity of the testing protocol to confirm the differences as observed during testing by 3-dimensional analysis. The study included 66 athletes who were diagnosed as having either no, subtle, or frank scapular dyskinesia during weighted flexion and abduction motions. People who were diagnosed with frank dyskinesia showed less scapular upward rotation, less clavicle elevation and greater clavicle protraction at rest and during motion as compared to people without dyskinesia. However, no association was found between presence of dyskinesia and shoulder pain or pathology[115]. The depressed clavicle and shoulder girdle was similar to the 'SICK' scapula described by Burkhart and colleagues[80] in throwers. Though scapular winging was observed by the raters in people with dyskinesia, no differences were found in scapular external/internal rotation. This may be due to the inherent high variability for this motion within the population. Another reason for inability to find differences may be attributed to analysis of motion at limited angles of elevation. The in-coordination or dyskinesia may be more motion associated rather than positional information. A different analysis approach such as considering displacements over a range of motion may be required to make more valid comparisons. However, the present study presumed that visible dyskinesia in scapular motion could be regarded as a screening tool to clinically differentiate between patients who had more motion related shoulder impingement mechanisms from patients who had more anatomical/ tissue change related mechanisms. Also, the same visual screening was used to exclude healthy people who had dyskinesia so as to obtain a more homogenous comparison sample population.

Association of pain and kinematics and muscle activity for motor control

The effect of pain on strength, range of motion and functional status of patients has been studied in shoulder patients by the use of subacromial injections. Pain is known to cause inhibition of muscles, thus producing apparent weakness[116]. Ballantyne et al.[117] reported reduced EMG levels in swimmers with painful shoulders. Ben-Yishlay and colleagues[38] studied patients with impingement syndrome and/or rotator cuff tears before and after subacromial lidocaine injection. They found an improvement in strength and range of shoulder motion after injection. Interestingly, the rotator cuff tear patients also showed improvements in strength which suggests that the loss of strength seen at pre-injection may be due to pain induced muscle inhibition rather than structural deficits alone.

Steenbrink and colleagues[118] studied EMG activity levels in shoulder muscles during isometric contractions in multiple arm elevated positions. They found that shoulder adductors were more active before a subacromial lidocaine injection which may contribute to decreased abduction force and range of motion. This co-activation of adductors during arm elevation was interpreted as a strategy used by the patients to decrease the superior humeral head migration and subsequent pain. Thus pain was an important contributor to altered motor activity seen in patients. Another recent study[37] focused on the kinematic changes observed before and after subacromial injection in patients with rotator cuff tears. These authors studied 3-dimensional scapular and humeral rotations. They calculated a regression line for humeral elevation with scapular upward rotation across different phases of elevation and found that scapular contributions decreased after subsidence of pain due to lidocaine injection. It is a possibility that pain causes selective inhibition of muscles (GH rotators, Serratus anterior) and thus forces patients to use alternative strategies and muscles to elevate their arm. These results were further supported by Scibek et al.[119] when pain was associated with changes in scapulohumeral rhythm in subjects with rotator cuff tears.

There are many hypotheses which describe the effect of pain on motor control. Some commonly proposed mechanisms include changes in kinesthetic sensations, slow reaction times, motor neuron/cortical inhibition of muscles and pain induced fear avoidance[41]. Though there is inconclusive evidence to support one mechanism over another, it is generally believed that there is an association between pain and altered motor control[41].

Mechanistic connection between motor control and kinematics

The reason why motor control could be considered as an important contributor towards altered kinematics in people with shoulder impingement relates to the possibility that it leads to inadequate scapular stabilization. It can be hypothesized that if the scapular muscles have a delayed activation as compared to the prime movers of the humerus (deltoid and supraspinatus), the latter will exert a reverse action on the scapula. A reverse action of the deltoid may pull the scapula into anterior tilt and downward rotation which is opposite to the normal pattern of scapular motion needed to occur during humeral elevation. An unstable scapula will also affect the efficiency of the rotator cuff muscles. If the rotator cuff muscles are not able to generate adequate torque against the increasing activity of the deltoid, they will not be able to resist the superior migration of the humeral head potentially causing further reduction of the subacromial space. Inadequate scapular stabilization against the thoracic curvature which is probably visualized as an increased prominence of the medial scapular border and/or inferior angle can be considered as an increase in relative scapular internal rotation or anterior tilting. Though scapular internal rotation is relative glenohumeral external rotation which is essential during arm elevation, this motion can bring the posterior glenoid closer to the humeral head potentially precipitating posterior impingement problems. Although these are purely mechanical speculations of the possibilities, it is important to identify the possible association of motor control and shoulder impingement problems. This study aims to find the relation, if any, between kinematic patterns, EMG muscle activity amplitudes and latency differences between healthy controls and people with shoulder impingement. Though this will not help to identify what exactly causes shoulder impingement problems, the results could help to identify strategies for improved treatment of patients.

CHAPTER 3

RESEARCH METHODS

Subjects

The study used fliers and advertisement on the university premises, hospital, and other possible public places to publicize the study. Initially a convenience sample of subjects with impingement were screened and included in the study. The healthy control group was comprised of individuals who were matched for age, gender and hand dominance with the group of individuals with shoulder impingement. It is difficult to obtain a sample if we want to match for each variable exactly individually; hence the matching was done in a way to keep the groups similar and avoid any significant group differences.

It is known that not all subjects presenting with shoulder impingement would have motion related pathology, hence the subjects were screened for visible scapular motion abnormality or dyskinesia. Dyskinesia was defined as an immature or excessive elevation or protraction or non-smooth motion, or the posterior displacement of the medial border and/or inferior angle away from the thorax during arm elevation[112]. Subjects were tested during arm elevation and lowering in the sagittal plane with and without a 2-3 Kg weight in hand for a maximum of 10 repetitions. Subjects were classified as either having or not having dyskinesia. No distinction was made between subtle and frank dyskinesia. The people presenting with shoulder pain were included in the study if they successfully satisfied the following inclusion criteria (Appendix 1):

1. Full range of motion (up to 150°) at the shoulder joint as measured goniometrically during flexion.

2. History of pain or tenderness at the shoulder joint (C 5 dermatome) up to the deltoid insertion for at least 6 weeks before participation in the study, which was not associated with any traumatic insult to the shoulder joint.
3. A positive test for at least 2 of the following clinical diagnostic tests:
    a. Neer impingement test.
    b. Modified Hawkins Kennedy test (internal rotation of arm in 90° elevated position in scapular plane elevation).
    c. Elicitation of pain with passive humeral external rotation at elevation of 90° (posterior impingement test).
4. Elicitation of pain during any 1
    a. Jobe's (empty can) test.
    b. Resisted humeral external rotation at elevation of 90°.
    c. Active motion (painful arc of motion).
5. Visible dyskinesia on the painful side seen during screening as described above.
6. Ability to perform arm elevation with a 3 Kg weight in hand for at least 10-12 repetitions.
7. No obvious crepitus as determined by the examiner while performing passive motion.
8. No evidence of adhesive capsulitis such that there is no loss of active and passive range of motion especially in the direction of axial rotations in elevated arm positions.

Subjects without any shoulder pain or pathology were included if they had full (up to 150°) pain-free range of motion at the shoulder joint as measured goniometrically during flexion, no current shoulder joint pain or tenderness, no history of pain and tenderness in the shoulder joint (C5 dermatome up to deltoid insertion) lasting for more than 2 days, internal/external rotation range of motion grossly within normal limits and no scapular dyskinesia at least while raising or lowering their arm without an additional weight held in hand.

Subjects were excluded if they had

1. History of
    a. Fracture of the clavicle, scapula or humerus
    b. Dislocation of the AC or glenohumeral joint
    c. Full thickness rotator cuff tears.
    d. Diagnosed glenoid labral tears/SLAP lesions
2. Age below 18 and above 60.
3. Pain, tingling or burning sensation in distal upper limb region consistent with cervical radiating symptoms.
4. Neurological disorder such as traumatic brain injury, stroke, peripheral nerve injury or compression affecting the tested upper limb, myasthenia gravis, spinal cord injury, motor neuron disorders etc.
5. Fixed kyphosis or diagnosed scoliosis.
6. Body mass index (BMI) in $Kg/m^2 > 28$
7. Known tape allergy.

With these criteria the study tried to focus on individuals with shoulder impingement syndrome which is related to repetitive motion rather than traumatic injury to the shoulder joint. The other information which was collected for the study included hand dominance using the Oldfield's hand dominance test (Appendix 2), subject's history of involvement in overhead work and sports activity; and any investigative diagnostic tests that the symptomatic group subjects had undergone previously (Appendix 3).

Power analysis

The means and standard deviations for the absolute latency obtained from the pilot data (5 healthy subjects) were used to estimate the necessary sample size with an alpha level of 0.05 and power of 80%. Due to the lack of any available calculation for sample size estimation for repeated measures ANOVA with SAS (statistical analysis software), the sample was estimated for a 2 sample ANOVA. With an expected effect of 33% change for the subjects with impingement as compared to healthy subjects and within group standard deviation of 83 msec. (largest amongst all muscles), a sample of 24 per group would be required.

The sample size estimation for finding group differences for relative latencies was done after collecting data from 10 subjects with shoulder impingement and 5 healthy subjects from pilot data. The within group standard deviation of 63 msec. was used which was an average of the standard deviations for the three muscle relative latencies. If we would observe a 33% change from these numbers in predicted directions (decreased latency for UT and increased for LT and SA) for the subjects with impingement, a sample of 24 in each group would be adequate to find group differences This sample size is also adequate as shown by earlier literature [12] to show differences for the kinematic and EMG data.

Another estimation which was performed was to find the sample size using data from previous literature for finding group differences for absolute muscle latency[50]. If the study was to be replicated exactly and the means and standard deviations as found during the previous work [50] were to be used for sample size estimation, a sample of 18 subjects per group would have been required for finding group differences with alpha of 0.05 and power of at least 80%. Based on this set of power calculations, the targeted sample size of 24 per group was used.

Subject Information

Fifty five subjects were recruited for data collection. The data from 49 subjects was included in the study out of which 25 had no history of shoulder pain or pathology and 24 subjects had a history of shoulder impingement. Data from 4 subjects could not be used because of excessive noise in the EMG data; one subject could not complete the trials after repetitive motion due to pain; and data collection could not be completed for one subject due to technical issues (Figure 3.1).

The average age of the healthy subjects was 32.2 (9.8) years and 35.09 (12.5) years for the subjects with impingement (Table 3.1). The number of females was 13 in the healthy group and 10 in the impingement group. There were no significant group differences found for the demographic variables (Table 3.1). The DASH scores from the healthy subjects was 1.7 (range: 0-10.8) as compared to 16.8 (range: 3.3 to 35) for the subjects with impingement. The DASH score ranges from 0 to 100 with a higher number suggesting increased functional limitations. The average Penn shoulder score (out of 60) was 59 for the healthy subjects as compared to 49 for the subjects with impingement. Eleven of the subjects with impingement reported a history of being involved with medium to high levels of competitive sports and 3 subjects reported lifting heavy weights overhead for work. Amongst healthy subjects, 4 subjects reported participating in medium to high competitive levels of sports. The range of time since onset of symptoms before being tested was 6 weeks to 14 years (mean ± standard deviation = 2.6 ± 4.13 years, n=15). None of the subjects reported taking anti spasmodic or muscle relaxant drugs. Twelve subjects with impingement (50%) reported having difficulty sleeping on the affected side and 7 subjects with impingement (28 %) reported waking at night due to pain and discomfort in the shoulder.

Instrumentation

Kinematic data

Kinematic data was measured using the Flock of Birds (FOB) hardware[120] and MotionMonitor software (Innsport Sports Technology, IL). This electromagnetic motion tracking system allows simultaneous collection of position and orientation information from up to 7 sensors at the sampling frequency of up to 144Hz. The system has a reported accuracy within a range of 1.2 m from the transmitter to be 1.8 mm root mean square (RMS) for static position and 0.5° RMS for static orientation of the sensor. The (mini-bird) sensors are small with dimensions of 24 mm x 29 mm x 6.6 mm and contain 3 electromagnetic coils orthogonal to each other. One of the sensors is attached to a stylus with known offsets to digitize anatomical landmarks for building the joint coordinate systems.

The MotoinMonitor software is a data acquisition tool which helps to process the kinematic data synchronously with data captured from EMG amplifiers. It provides an immediate animation and graphical display of the tracked motion and later allows exporting data using various kinematic descriptors of rotation matrices and Euler angle sequences.

EMG data

Myoelectric signal data was captured using the EMG system (Therapeutics Unlimited, Iowa City, IA, USA). Silver/silver chloride bipolar active circular electrodes of diameter 8 mm and an inter-electrode distance of 2 cms were used for collecting electrical signals from the muscles with an on site gain of 35 times. The signal was further amplified using an adjustable gain setting, input impedance of >15 MOhms at 100Hz, CMRR of 87dB at 60 Hz, and noise <2.0 microvolts RMS referred to input. The signals from the onsite electrodes were filtered by the amplifier using a high pass filter of 20 Hz to reduce cable artifact.

This raw EMG was sampled at the rate of 2500 Hz using a 16 channel A/D board and MotionMonitor software. Raw signals were monitored on an oscilloscope (Tektronix Inc., OR, USA) throughout data collection. To remove noise signals collected due to electromagnetic pulses of the Flock of Birds system, the EMG raw signals were filtered using 8 notch filters after calibrating for them in MotionMonitor software. The process enables identification of the frequencies of the FOB that can create noise in the EMG signal and notch filters those frequencies from the recorded EMG muscle activity.

Procedure

The subjects were initially screened by a phone interview and later clinically to confirm either their inclusion or exclusion from the study. An informed consent was signed by all subjects that delineated the risks and benefits of their participation in the study (Appendix 4). Information regarding history of overhead work, athletic participation, any past clinical information on shoulder pathology as well as the DASH (Disabilities of the Arm, Shoulder and Hand) functional status measure (Appendix 5) and Penn shoulder scores (Appendix 6) were collected.

Surface EMG electrodes were placed over upper and lower fibers of trapezius, serratus anterior and anterior deltoid on each subject. For each of the following descriptions, the electrodes were placed parallel to muscle fibers such that the described point lay between the two electrodes used for that muscle. For upper trapezius, the electrode was placed over a point 2 cm lateral to the midpoint on a line joining the C7 spinous process and tip of the acromion process[91, 121]. For lower trapezius, the arm was elevated in scapular plane abduction to about 125° and the electrode was placed midway between the inferior angle of the scapula and the T7 spinous process[20, 108]. For serratus anterior, the electrode was placed over the 7th intercostal space, just anterior to the fibers of latissimus dorsi, after the subject elevated the arm up to 125° in scapular plane elevation[90]. This position was preferred over the 3rd intercostal space as there is less subcutaneous tissue over this site, less chance of cross talk from pectoralis major or latissimus dorsi and lower fibers are mostly involved with the chosen scapular kinematic parameters of the study (scapular upward rotation, posterior tilt and external rotation). For recording electrical activity from anterior deltoid, the electrode was placed 2 finger breadths below the acromion process[89]. A reference electrode was placed over the ulnar styloid process of the contralateral arm. The position of all electrodes (Figure 3.2 a and b) was confirmed by asking the subject to perform resisted contractions of the muscles to make the contraction visible. Also, crosstalk from neighboring muscles was checked by monitoring the oscilloscope signals while asking the subject to perform specific action to activate nearby muscles and not the muscle to be tested.

For normalization, the EMG data were collected over 2 trials when the subject performed maximum voluntary contraction (MVC) against manual resistance for 3 seconds. The mean of the activity for the peak moving window of 50 msec. was used for normalizing. Baseline activity was measured as the minimum of a 50 msec. moving window average computed from the rest file and was subtracted from the EMG activity as there was potential interference from the electromagnetic Flock of Birds system. Adequate rest periods of 30 seconds were given between trials to avoid fatiguing the muscles. For upper trapezius, the subject was asked to raise their arm in the direction of flexion at an angle of 60° against resistance applied at the distal upper arm while in a sitting position with the back stabilized[91, 92, 122] (Figure 3.3 a). The lower trapezius MVC was recorded in a prone position when the subject elevated and horizontally abducted their arm at 90° of elevation against resistance applied to the distal arm[90] (Figure 3.3 b). Resistance was applied to the distal arm for recording the MVC for serratus anterior when the subject performed scapular protraction (punch motion) at 60° of arm elevation in flexion (elbow flexed) (Figure 3.3 c). Resistance was applied to the distal upper arm when the subject performed arm elevation at 60° for recording MVC for anterior fibers of the deltoid[123] (Figure 3.3 a). The 60° elevation angle is the midpoint of the range being tested. Also, it was anticipated there would be less chance of reduced muscular effort or muscle inhibition due to pain at lower elevation angles for the subjects with impingement. Pain scores using the NPR scale (Appendix 7) were collected during each trial of MVC testing.

The electromagnetic sensors for tracking trunk motion were placed over the sternum, over the distal acromion for the scapular sensor and on a thermoplastic cuff worn over the distal arm for tracking humeral motion (Figure 3.2a). Then notch filters were calibrated for removing the noise obtained due to the electromagnetic train of pulses from the transmitter.

The subject stood in a “relaxed” standing position while certain anatomical landmarks were digitized to create the anatomical coordinate frames. This allowed transforming the data obtained from sensor tracking to more meaningful anatomically based joint coordinate systems (JCS)[124]. The trunk JCS was defined by digitizing the following points: spinous process of C7 vertebra (C7); spinous process of T8 vertebra (T8); deepest point of suprasternal notch; and the most caudal point of xiphoid process. The scapular JCS was defined by palpating: a point on the medial border of the scapula at the level of the spine also called the root of the spine; the most inferior point on the inferior angle of the scapula; and the posterolateral acromion process. The humeral JCS was defined by palpating the lateral and the medial epicondyle of the humerus (Appendix 8). The center of the humeral head was estimated by moving the arm through various small arcs of motion to define the pivot point using an optimization technique[125].

After digitization, the subject was instructed to stand in a neutral rest position for collecting a kinematic description of the rest posture with the arms relaxed at their side.

The attachment sites of the pectoralis minor muscle were also digitized in their respective sensor frames. The coracoid process was digitized in the scapular sensor frame and the 4th rib attachment site was digitized in the trunk sensor frame[30]. Then, the subjects practiced elevating their arm through their full range of motion in front of their body approximately in the sagittal plane at a speed such that it took 2 seconds to elevate their arm and 2 seconds to lower it. The subjects received a verbal command to be ready which was followed by a light signal. They were instructed to move as soon as they saw the light signal. Instructions were given to the subject to relax completely before each trial as the EMG signal before the trigger was used as baseline data for latency calculations. The subjects were also informed that the time between the 2 signals (verbal and light) may vary from 0.5 seconds to 4 seconds. This would reduce subject's anticipation to affect the reaction time. Also, the subject was informed about 'catch trials'. This means that the light signal may not trigger at all which avoids the effect of aging fore-periods (increasing anticipation of the subject with delay in cue causing decreased reaction times) affecting the reaction time[103]. Subjects were not constrained to assume any forced rotations of the arm and were instructed to move in their natural way. The loaded and unloaded trials were randomized using a coin flip. In case of loaded trials, the subject was given a weight in hand between 2 and 4 kg (according to their BMI and arm length) (Appendix 9). Five trials of each condition were recorded. After completing both sets of loaded and unloaded trials, the subjects raised and lowered their tested arm 10 times with the weight while no data was collected. This was followed by data collection for 5 trials continuing with the weight in hand. Information regarding any pain and discomfort was noted after each trial; the subjects rated their pain on a numerical pain score (Appendix

7). Also, the subjects were asked to rate their perceived exertion on the Borg's scale of perceived exertion (RPE) after each trial (Appendix 7).

Data reduction

The kinematic data was sampled at 100 Hz. The MotionMonitor software was used to define the JCS for the scapula, humerus and trunk using the digitized landmarks following the ISB recommended protocol[124]. The sensor data were transformed to the anatomical reference frames to allow clinical interpretation[124]. The anatomical X axis was pointing forwards, the Y axis upwards and the Z axis laterally outwards for the right side data analysis. The axes orientation for the left side was changed such that the X axis points backwards, Y axis upwards and the Z axis laterally outwards towards the left side. Euler angle sequences were used to describe the position and orientation of the segment at each frame following the ISB protocol[124] except for the humeroscapular descriptions. Appropriate conversions for the left sided data were made for further calculations. The YX'Z"" sequence was used to describe the scapular motions with respect to the trunk reference frame. It described the rotations in the order of internal/ external rotation, followed by upward/downward rotation and anterior/posterior tilt[126]. The humeral motions were described with reference to the trunk and scapula. The former was described using the ISB recommended YX'Y" sequence which defines the plane of elevation, elevation angle and then the axial rotation of the humerus. This sequence is limited by issues of singularity near positions of rest and full elevation and hence an alternative sequence was used to describe the humero-scapular motion. That was described by the X'Z'Y" sequence which defines the rotations in the order of arm elevation, plane of elevation (horizontal adduction/abduction) and then axial rotation (Appendix 8). The length of the pectoralis muscle was calculated as a Euclidean distance between the two attachment sites. It was then normalized to the height of the individual for subsequent data analysis.

The scapulohumeral rhythm was determined by calculating the slope of a line using linear regression across glenohumeral elevation and scapular upward rotation[127]. The slope was calculated in 30 degree increments (30°-60°, 60°-90° and 90°-120°) as well as from 30° to 120° of the elevation phase. The average value from 5 trials was used for all analyses.

The EMG data were sampled at 2500 Hz and then filtered by a low pass filter with a cut off at 500 Hz using the MotionMonitor software. Also, the data were filtered for electromagnetic noise due to the FOB system using the calibrated notch filters. MATLAB was used to full wave rectify and further smooth the data using a 50 Hz low pass 7th order Butterworth filter. For calculation of muscle latency, baseline EMG was calculated as the average of the 50 msec. before the light trigger. The evaluation of onset of muscle activity was calculated by using the algorithm described by Hodges & Bui[106]. It identifies the point where the mean of a moving window of 62 consecutive frames (25 msec.) exceeds the baseline activity by 3 standard deviations. Each onset time was checked visually to identify EMG trials disrupted by heart beat or other motion artifacts. The onset time was accepted only if the muscle maintained activity higher than the threshold level in subsequent windows (Figure 3.4). The relative latency of UT, LT and SA was calculated as a difference of their latency and that of anterior deltoid. The onset of a muscle was termed as feed-forward if it had an onset before or up to 50 msec. after the onset of anterior deltoid which is the prime mover (Appendix 10). Muscle onset before 50 msec. can not occur in a reaction to perturbation due to the electromechanical delay[42].

The switch off or the deactivation time for each muscle was identified in a similar way. The mean of 250 consecutive samples (100 msec.) was calculated from the point in time when the humerus was at a 120° elevated position during arm lowering. The deactivation time was identified when the mean of this moving window was lower than or equal to the sum of mean of the baseline activity and 3 standard deviations. Again, the muscle was only considered deactivated if the muscle activity after the switch off time continued to remain lower than the set threshold level in the subsequent 2000 (800 msec.) windows. The corresponding humerothoracic elevation angle was recorded for further analysis. In cases when the deactivation of muscles could not be identified at all, the lowest humeral elevation angle was considered as the angle for analysis (Figure 3.5). Each trial result was checked visually for motion artifact or noise due to the heart beat. In the presence of visible artifact, and if possible the algorithm was run for the windows after the artifact. The trials were not used if such artifacts were suspected to affect the outcome especially when the artifact occurred in the 50 msec. baseline window period or visually occurred to coincide with muscle activation (Figure 3.6a, b).

For quantification of the EMG data across the range of motion, the mean of the EMG activity over 30° increments (30°-60°, 60°-90°, 90°-120°) was calculated after full wave rectification and filtering. The baseline activity as measured during the 50 msec. moving window average from the rest file was subtracted from this. Then voltage values were normalized individually to the maximum of the mean muscle activity obtained over a 50 msec. moving window during the 2 normalization trials.

Validation of the Serratus Anterior Surface Sensor

Research methods: In a sample of 5 subjects with no history of shoulder pain or pathology, an additional experiment collected serratus anterior myoelectric activity using both fine wire electrodes and surface electrodes. Data from 3 males and 2 females with an average age of 35.6 years; height = 171.7 cms ; weight = 77.5Kgs; and BMI = 25.7 were collected. All were right hand dominant and the dominant side was tested in all subjects. The fine wire was inserted over the 6th or 7th rib and the surface electrode was attached over the 7th intercostal space at the mid-axiallry line just anterior to the latissimus dorsi muscle, after the person elevated the arm up to 125° in scapular plane elevation[90]. The connections of the fine wire to the preamplifier were not in optimal contact for the data collection of first 2 subjects and were subsequently changed for the last 3 subjects. The activity of latissimus dorsi (LD) was collected by a surface EMG electrode placed 3 finger breadths distal to the inferior angle along the posterior axillary fold parallel to the lateral scapular border[128]. A reference electrode was placed over the ulnar styloid process of the contralateral arm.

For normalization, the EMG data was collected over 2 trials while the subject performed maximum voluntary contractions against resistance for 3 seconds. The maximum of the average moving window activity over 50 msec. was used for the normalization reference. The MVC for the serratus anterior was collected as described earlier. For latissimus dorsi, the subject was asked to perform resisted adduction and extension of the arm while maintaining medial rotation starting from an adducted, extended and medially rotated position. Resistance was applied to the distal arm.

The procedure for motion trials (verbal cue followed by light cue) remained the same as for the full experiment. Data were recorded for unloaded and loaded trials while the subject raised their arm in front of their body approximately in the sagittal plane. In two subjects unloaded trials while maintaining humeral external rotation were also collected.

Data analysis: The EMG data was filtered and smoothed as described previously except that the fine wire EMG data were low pass filtered at 1000 Hz instead of 500 Hz. The latency for serratus anterior was estimated by the same computer algorithm explained earlier. The similarities between signals were assessed by the method used by Marshall & Murphy[129]. This study[129] compared the cross correlation coefficients[130] as obtained by correlation of a signal with itself (auto-correlation) and with another signal. Auto-correlation yields a correlation of 1 and time lag of 0. The cross correlation of two different signals gives an estimate of similarity. In the current study, the auto-correlation for surface serratus activity was estimated using the 'Xcorr' function in MATLAB (The Mathworks, MA). Additionally, cross correlation was calculated between signals of latissimus dorsi activity and that of serratus anterior activity as collected by surface ($\text{Xcorr}_{\text{LD-SAsurface}}$) and fine wire electrodes ($\text{Xcorr}_{\text{LD-SAfine wire}}$). For quantification of the EMG data across the range of motion, the means of the EMG activity over 3 successive windows each of 30 percent of the motion were calculated after full wave rectification and filtering. These values were normalized individually to the MVC and expressed as a percentage of MVC.

Data Analysis

The study primarily intended to determine if group differences existed for latency across the different conditions. The dependent variables for the study were the muscle absolute and relative latencies of UT, LT, and SA; humeral elevation angle corresponding to the time when the muscles were deactivated; scapular and glenohumeral 3-D angular kinematics; and normalized EMG activity of UT, LT and SA. The independent variables were the groups (subjects with and without impingement), conditions (unloaded, loaded and after repetitive motion), angles of arm elevation for kinematic analysis (rest, 30°, 60°, 90° and 120°), phases of motion (elevation and lowering), and motion increments of arm elevation and lowering for EMG analysis (30°-60°, 60°-90°, 90°-120°, 120°-90°, 90°-60° and 60°-30°). Amongst these factors, the group was a between subjects factor and the others were within subjects factors. This necessitated the use of mixed model ANOVAs. The level of significance was 0.05 for all tests.

Any differences between groups for age, height, weight, BMI and normalized pectoralis minor length, were tested using 2 sample t-tests. The difference between groups for Borg's scale of exertion and average velocity for elevation and lowering phases were estimated by a 2 way mixed model ANOVA across group and condition (unloaded, loaded and after repetitive motion). In case of significant interactions, the effect of group was analyzed using the Tukey-Kramer post-hoc analysis of 2-way interactions. The differences between groups for categorical variables such as distribution of gender, hand dominance, or tested side were done using chi squares analysis.

For all variables, the within subject trial to trial reliability was tested by calculating the ICC (intraclass correlation coefficient) (Model 3 and type (3, 1)) and

SEMs (standard errors of measurement)[131] (Appendix 11). After reliability testing, the mean of all available trials for each subject and condition was used for subsequent analyses.

Prior to performing further statistical tests, the assumptions of parametric statistics were tested. The normality of each variable for each cell (condition combination) in a 2 or 3 way ANOVA was tested using measures of skewness, kurtosis and standard normality tests (Shapiro-Wilk, Kolmogrov-Smirnov)[130]. If the data were not normally distributed, attempts were made to transform the data. If data transformations failed to render the data normal, outliers were identified using box-plots. The data from a subject was identified as an outlier if it was more than 3 times the inter-quartile range lower than the first quartile or 3 times the inter-quartile range higher than the third quartile. The data were re-analyzed for normality without the outliers, and if assumptions of normality could be satisfied, the outliers were removed for subsequent analyses of the data. If the normality assumptions could not be satisfied even after removal of outliers, non-parametric statistics were performed for that variable.

For hypothesis testing, generally 2 or 3 way mixed ANOVA models were used. In the case of significant interactions in a 2 way ANOVA, the follow up comparisons were made using Tukey-Kramer adjusted post-hoc analysis of 2-way interactions (comparisons at each level of interacting factors). In the case of significant 3 way interactions in a 3 way ANOVA, either the analysis was done at each level of the main factor of interest by multiple 2 way ANOVAs or the effects were analyzed at each interacting factor using contrasts. In the case where main effects of condition, phase, motion increment or angle were significant, follow-up pair wise comparisons were made using Tukey Kramer tests.

Hypotheses were tested in the following manner:

1. Hypothesis 1: This hypothesis predicted group differences for presence of feed forward contractions of scapular muscles. This was tested by checking the relative latency for UT, LT and SA during each condition. If the onset was before or up to 50 msec. after the onset of anterior deltoid, it was termed as feed forward. The total number of trials across subjects for each condition when the muscle showed feed forward contraction were counted and entered into a 2x2 chi square table. Chi square analysis was run across groups and individual muscle for all conditions (unloaded, loaded and after repetition motion) separately.
2. Hypothesis 2: This set of hypotheses predicted differences in absolute latencies and were tested by using a 2 way mixed model ANOVA across groups (healthy/impingement) and conditions (unloaded, loaded and after repetition) for each muscle (UT, LT and SA) separately. Subhypothesis a tested the main effect of group. In the presence of significant interactions with group, the effect of group was analyzed by follow-up pair-wise comparisons using Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

   Subhypothesis b tested the effect of condition, with a specific pair-wise difference predicted. In the presence of significant interactions with condition, the effect of condition was analyzed by follow-up pair-wise comparisons using Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

   Subhypothesis c tested the effect of condition (loading) on the LT absolute latency, with a specific pair-wise difference predicted. In the presence of significant interactions with condition, the effect of condition was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

3. Hypotheses 3: This set of hypotheses tested relative latencies of scapular muscles across groups and conditions. Subhypothesis a predicted latency differences across muscles. Group differences were still of interest, but not hypothesized. This subhypothesis was tested using a 3 way mixed model ANOVA across all muscles (UT, LT and SA), conditions (unloaded, loaded and after repetition) and groups (healthy/impingement). In the case of significant 3 way interactions, the effect of muscle (recruitment order) which was of main interest was tested for each group separately. In the case of subsequent 2 way interactions with muscle, the effect of muscle was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

   Subhypothesis b predicted an interaction between group and condition and subhypothesis c predicted a main effect of group. These were tested using a 2 way mixed model ANOVA across groups and conditions (unloaded and loaded and after repetitive motions) for each muscle (UT, SA and LT) separately. In the case of significant interactions the effects of group or condition were analyzed by follow-up pair-wise comparisons using Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

4. Hypothesis 4: This hypothesis was tested by multiple 2 way mixed model ANOVA for the humeral elevation angle of deactivation across groups (healthy and impingement) and conditions (unloaded, loaded and after repetitive motion) for each muscle separately. The hypothesis predicted a main effect of group and significant

interactions between groups and conditions. In the presence of significant interactions, the effects of group or condition were analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

5. Hypothesis 5: This hypothesis predicted an effect of group for the kinematic variables under the unloaded condition (group x condition interaction). This hypothesis was tested by a 3-way mixed model ANOVA across groups (healthy/impingement), conditions (unloaded, loaded and after repetition), and elevation angles (30°, 60°, 90° and 120°) for each 3-dimensional scapular variable (Scapular internal/external rotation, upward/downward rotation, tilt) across the phases (elevation/lowering) separately. In the presence of significant interactions of group with angle or condition, the effect of group was tested across each angle and/or condition using contrasts. In the presence of only 2 way interactions or to specifically determine any group differences during unloaded conditions, the effect of group, condition or angle was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.
6. Hypothesis 6: The hypothesis predicted group differences for scapulohumeral rhythm after repetitive motion (group x condition interaction). The hypothesis was tested by a 3 way mixed model ANOVA across groups (healthy/impingement), conditions (unloaded, loaded and after repetition), and angle increments. The dependant variables were the slope of the line from a regression of glenohumeral elevation and scapular upward rotation. In the presence of significant interactions of group with angle increment or condition, the effect of group was tested across each angle and/or condition using contrasts. In the presence of only 2 way interactions or to specifically

find differences in the condition after repetitive motion, the effects of group or condition were analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

7. Hypothesis 7: Subhypothesis a predicted group differences for SA muscle activity and was tested by a 3 way mixed model ANOVA across motion increments (30°-60°, 60°-90° and 90°-120°), groups (healthy and impingement) and conditions (unloaded, loaded and after repetitive motion) for each phase (elevation and lowering) separately. In the presence of significant 3 way interactions with group, the effect of group which was of primary interest was tested at each level of the interacting factors (condition or angle) using contrasts. In the presence of only 2 way interactions, the effect of group, condition or angle increment was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

Subhypothesis b predicted group differences for LT activity in the lowering phase only. It was tested using a the 3 way mixed model ANOVA across motion increments (120°-90°, 90°-60°, and 60°-30°), groups (healthy and impingement) and conditions (unloaded, loaded and after repetition) for the lowering phase. In the presence of significant interactions with group, the effect of group which was of primary interest was tested at each level of the interacting factors (condition or angle increment) using contrasts. In the presence of only 2 way interactions, the effect of group, condition or angle increment was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

Covariate analysis:

The following were considered as possible covariates: Age, BMI, normalized pectoralis minor length, pain level measures using a NPR scale, Borg's scale index, DASH scores and the speed of motion calculated as average humeral velocity for elevation and lowering phases. Correlations were computed between the dependant variables and covariates. In cases, when the correlation coefficients were significant and above 0.5 or showed patterns of group differences, the covariate was retained for further analysis using ANCOVA (analysis of covariance), otherwise the covariate was dropped from further consideration. When retained, the interaction of the covariate with all the factors in the 2 or 3 ways ANOVAs were checked for significance. In the case of no significant interactions, the main effect of the covariate was also tested. If the covariate showed a significant interaction with any other factor, it was nested within that factor for the analysis. The interpretation of results was done with and without the covariate in the model. For conditions where the presence of the covariate did not result in any substantive change in the interpretation of results (p value not changing significantly), the simpler model without the covariate was used.

Additional Exploratory Analyses

1. Effects of trials: The effects of trials on muscle latency were tested using 2 way mixed model ANOVAs across group and trial for each condition separately. The effects of trials on kinematic variables were tested using 3-way mixed model ANOVA across group (healthy and impingement), angle of elevation (30°, 60°, 90° and 120°) and trial for each condition and phase separately. In the case of

significant 3 way interaction, the effect of trial was tested at each angle separately by 2 way ANOVAs. In the case of significant 2 way interactions, the effect of trial was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

2. Relaxed standing position differences: The relaxed standing posture was recorded at the beginning and end of the data collection sessions. The kinematic variables were tested for group differences in the initial and final positions individually by 2 tailed 2 sample t-tests. Also, the scapular and glenohumeral kinematic variables at the initial rest position were compared with those at the final rest position using 2 way mixed model ANOVA across group and time of collection (initial/final).
3. Peak elevation kinematic differences: The scapular and glenohumeral kinematic variables were determined at peak elevation of each trial. Though there are known errors associated with surface tracking of scapular kinematic variables [22] and there were different peak elevation angles for each subject and trial, attempts were made to find differences which could be important for describing mechanistic causes of internal impingement. The kinematics at peak elevation were analyzed across groups and conditions by a 2 way mixed model ANOVA. In the case of significant interactions, the effect of group was analyzed using Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.
4. Glenohumeral kinematic variables: The 3D kinematic variables of glenohumeral plane of elevation, angle of elevation and axial rotation were tested for group differences across conditions (unloaded, loaded and after repetitive motion), and elevation angles (30°, 60°, 90° and 120°) by 3 way mixed model ANOVA for

each phase (elevation and lowering) separately. In the presence of significant interactions of group with angle or condition, the effect of group was tested across each angle and/or condition using contrasts. In the presence of only 2 way interactions, the effect of group, condition or angle was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.

5. <u>EMG analysis of upper trapezius activity</u>: This was tested by a 3 way mixed model ANOVA across motion increments (30°-60°, 60°-90° and 90°-120°), groups (healthy and impingement) and conditions for each phase (elevation and lowering) separately. In the presence of significant 3 way interactions with group, the effect of group, which was of primary interest, was tested at each level of the interacting factor (condition or angle) using contrasts. In the presence of 2 way interactions, the effect of group, condition or angle increment was analyzed by Tukey-Kramer adjusted post-hoc analysis of 2-way interactions.
6. <u>Analysis of fine wire EMG of serratus anterior as compared to surface</u>: The cross correlation coefficients for $\text{Xcorr}_{\text{LD-SA surface}}$ were compared to that of $\text{Xcorr}_{\text{LD-SA fine wire}}$ descriptively. The latencies as estimated by fine wire and surface electrodes for serratus anterior were compared descriptively. The muscle activity as a percent of maximum voluntary contraction was described for latissimus dorsi and serratus anterior across 30% of motion increments for the elevation phase.
7. <u>Logistic Regression</u>: All the kinematic variables, and the normalized pectoralis minor muscle length were included in a logistic regression analysis which modeled the log of odds of being in the impingement group for the unloaded condition. These analyses were done at 30°, 60°, 90° and 120° of humerothoracic

elevation angles. This is similar to a multiple regression model with the log of odds as the dependent variable. No interactions were tested. The odds ratios of all the variables with a significant Wald's chi square were analyzed to find the direction of differences between groups.

8. <u>Group comparison of variance</u>: The variances for relative muscle latencies during the unloaded condition were tested for equality across groups by a one sided F-test. The analysis was done with and without the outliers for each muscle.
9. <u>Analysis of subgroups within impingement group:</u>
    a. The subjects with impingement were sub grouped based on the history provided by the subjects and the clinical examination. They were divided based on the clinical impressions of the investigator into either having mainly internal or mainly subacromial impingement or a combination of symptoms. The subjects who possibly only had internal impingement were identified if they had a history of participating in overhead sports (volleyball, baseball, or cricket), were relatively younger (less than 40 years of age), had pain terminally with elevation, or tested positive with a posterior internal impingement test. Subjects who possibly had only subacromial impingement were identified if they tested negative on the above mentioned criteria. If they had an ambiguous history and presentation to clearly differentiate them into these categories of either internal or subacromial, they were sub grouped as ambiguous. The scapular kinematic variables were analyzed descriptively over all conditions and both phases separately.

b. The subjects with impingement were also sub grouped based on the level of involvement as indicated by the DASH scores. Subjects with DASH scores equal to or higher than 20 formed the high involvement sub group and subjects with DASH scores less than 20 formed the low involvement sub group. The scapular kinematic variables were analyzed descriptively over all conditions and both phases separately.

10. CHAPTER 4

RESULTS

Reliability analysis

The ICC and SEM results are provided in Table 4.1 and 4.2 The ICCs for scapular internal/external rotation ranged between 0.90 - 0.98; for scapular upward rotation between 0.80-0.90 and for scapular tilt between 0.87 – 0.98. The SEMs for all kinematic variables were below 3.5°. The ICCs for scapular muscle latencies were very low owing to the very high variability between trials. All ICCs for relative latencies across conditions and groups were less than 0.3. The ICCs for absolute latency of upper trapezius ranged from 0.17 to 0.50; for lower trapezius ranged from 0.20 to 0.46 and for serratus anterior ranged from 0.13 to 0.41 (Table 4.2). The SEMs for absolute latency of muscles ranged from 96 to 164 milliseconds and the SEMs for relative latency ranged from 73 to 135 milliseconds.

Trial to trial analysis

The trial did not have a significant effect on any absolute or relative muscle latency parameters. For kinematic variables, trial had a significant effect for analysis of scapular upward rotation, glenohumeral elevation and plane of elevation in the unloaded condition. The first trial was significantly different from the last three trials with an increase in scapular upward rotation, decrease in glenohumeral elevation angle and shifting of plane of elevation away from the sagittal plane. The differences in angular values were all less than 1.8°. These trends in differences for the kinematic parameters were captured by the condition effect as well and hence it was rationalized to use the average of the 5 trials for each condition. Average data from 5 trials was used for all subjects for analysis of kinematic and EMG variables except for latency analysis when some trials were discarded due to artifacts.

Covariate analysis results

Multiple correlations were calculated between demographic variables (age, height, weight and BMI), average pain scores during each condition, average Borg's scale score during each condition, average velocity of motion during elevation and lowering, height normalized pectoralis minor muscle length and the dependant variables (muscle latency and kinematic variables) (Appendix 12.A-F). The normalized pectoralis minor muscle length was the only variable with consistent moderate correlations (<0.5) (Table 4.3) for the analysis of scapular internal/external rotation and scapular tilting across groups. However, although the covariate was statistically significant, its inclusion in the model did not change the significance of other variables so as to change the interpretation of results. Hence simpler models without the covariate were retained (Appendix 13).

As the glenohumeral plane of elevation showed significant group differences, the humerothoracic plane of elevation at 90° was also checked to determine if it had any significant correlations with other kinematic variables (Appendix 12.G). However, due to low correlation values, it was not retained for further ANCOVA analysis.

Descriptive data (means/standard deviation) for all dependent variables by group and condition are presented in Tables 4.4.1 to 4.4.6

Results of Hypothesis Testing

Hypothesis 1

There were trials dropped due to presence of cardiac or motion artifact for estimation of muscle latency. The total number of lost trials for calculating relative latency was approximately 8 for upper trapezius, 10 for lower trapezius and 15 for serratus anterior across conditions (Table 4.5).

The data for relative latencies for muscles showed outliers which affected the normality of data and prevented effective transformation. Analysis of relative latency data after removal of outliers allowed us to perform parametric statistics and hence data from 2 subjects (different for each muscle) were excluded. So the total number of healthy subjects included was 24 for upper trapezius and lower trapezius analysis and 23 for serratus anterior analysis whereas the total number of subjects with impingement was 23 for all upper and lower trapezius analysis and 24 for serratus anterior analysis.

Results for these analyses are presented in Table 4.4.3. The upper trapezius showed feedforward contraction during 76% of unloaded trials in subjects with impingement, which was significantly higher than 57% of trials for healthy subjects (chi square = 9.44, p = 0.002). Similarly lower trapezius showed feedforward contraction during 63% of unloaded trials in subjects with impingement which was significantly higher than 46% of trials for healthy subjects (chi square = 6.6, p = 0.01). In trials after repetitive motions, serratus anterior showed feedforward contraction during 85% of trials in healthy subjects which was significantly higher than 73% of trials in subjects with impingement (chi square = 4.67, p = 0.03). All other condition comparisons were not significantly different between groups (Table 4.4.3).

Hypothesis 2

The data for absolute latencies were not normally distributed; hence, subsequent analysis on absolute latency data was done after a log transformation. Results for these analyses are presented in Tables 4.4.1, 4.6.1, 4.6.2 and Figures 4.1a-c.

Hypothesis 2 a

This hypothesis predicted differences between groups. There were no significant differences between groups for upper trapezius absolute latency. The absolute latency of lower trapezius showed a significant group by condition interaction (df = 2/94; F-ratio=3.11; p = 0.049) However, follow up analysis did not find significant differences between groups at each condition (Table 4.6.2)

The absolute latency of serratus anterior approached significance (p = 0.12) for group differences. The serratus anterior average absolute latency in subjects with impingement was 283.44 msec. as compared to 245.5 msec. in healthy subjects. Subjects with impingement showed a trend toward delayed recruitment of serratus anterior as compared to healthy subjects.

Hypothesis 2 b and 2 c

The hypothesis 2b predicted effects of condition and there were significant differences for absolute latencies of all three muscles. The absolute latency for upper trapezius increased significantly after repetitive motion (321.6 msec.) as compared to the unloaded condition (282.8 msec.) for both groups (df = 2/94; F-ratio = 4.04; p = 0.021). The absolute latency of upper trapezius during the loaded condition (308.3 msec.) was not significantly different from the other two conditions.

There was a significant interaction of group and condition (p = 0.049) for analysis of lower trapezius. The absolute latency for lower trapezius decreased from the unloaded condition (339.2 msec.) to the loaded condition (242.1 msec.) in healthy subjects as well in subjects with impingement (from 325.4 msec. to 265.1 msec.). However, the absolute latency for lower trapezius was decreased significantly from the unloaded condition to the condition after repetitive motion (248.1 msec.) for healthy subjects only and not for subjects with impingement. There were no significant differences for the variable between the loaded and after repetitive motion conditions across both groups (Table 4.6.2).

The absolute latency for serratus anterior significantly decreased in the loaded condition (248.4 msec.) and in the condition after repetitive motion (257.5 msec.) as compared to the unloaded condition (288.4 msec.) for both groups (df = 2/94; F-ratio = 7.61; p = 0.0008). There were no significant differences between the loaded condition and after repetitive motion condition (Table 4.6.2).

Hypothesis 3

Hypothesis 3 a

Results for these analyses are presented in Table 4.7.1, 4.7.2 and Figures 4.2 a-c. The hypothesis predicted a muscle order effect for recruitment of scapular muscles. A 3 way ANOVA was performed for relative latencies of scapular muscle across group and condition after excluding data from 6 subjects (2 outliers for each muscle). There was a significant interaction between muscle recruitment and condition (df = 4/164; F-ratio = 14.57; p<0.001). The serratus anterior (18.2 msec. after deltoid) and upper trapezius (17.7

msec. after deltoid) were recruited significantly before lower trapezius (64.6 msec. after deltoid) for the unloaded condition. There was no significant difference between upper trapezius and serratus anterior relative latency for the unloaded condition. In the loaded condition, the serratus anterior (2.4 msec. after deltoid) and lower trapezius (8.8 msec. after deltoid) were recruited significantly before the upper trapezius (60.3 msec. after deltoid) ($p<0.05$). This order of recruitment continued in the condition after repetitive motions with the serratus anterior (3.7 msec. before deltoid) and lower trapezius (22.2 msec. after deltoid) getting recruited significantly before upper trapezius (54.8 msec. after deltoid) ($p<0.05$). There were no significant differences between lower trapezius and serratus anterior relative latency in loaded and after repetitive motion conditions (Table 4.7). There were no significant differences in the order of muscle recruitment between the two groups.

Hypothesis 3 b

Results for these analyses are presented in Table 4.4.2, 4.8.1, 4.8.2 and Figures 4.2 a -c. The hypothesis predicted an interaction of group and condition for each muscle. The lower trapezius interaction came close to significance with $p = 0.08$. There was a significant main effect of condition for upper and lower trapezius. The relative latency of upper trapezius significantly increased from the unloaded (19.2 msec. before deltoid) to the loaded condition (71.7 msec. after deltoid) in both groups. The relative latency of lower trapezius significantly reduced from the unloaded (65.1 msec. after deltoid) to the loaded condition (6.8 msec. after deltoid) ($p<0.05$) in both groups. The serratus anterior showed a strong trend ($p = 0.052$) of decreasing relative latency from the unloaded condition (18.7 msec. after deltoid) to the loaded condition (5.6 msec. after deltoid) in both groups (Table 4.8.2).

Hypothesis 3 c

The hypothesis predicted a group difference between relative latency of muscles. No significant differences were found between groups for any of the 3 muscles (Table 4.8.1). However, the upper trapezius data was analyzed for each condition separately. This was done because the upper trapezius showed a high baseline activity in all conditions with weight held in the hand and hence the activation as detected by the algorithm (compared to a higher baseline) was delayed. Subsequently, it could be argued that the muscle was active even before motion began. The 2 sample t-test for upper trapezius relative latency during the unloaded condition showed a group difference (t-value=2.95; p = 0.005) for the subjects with impingement (4.24 msec. before deltoid) recruiting their upper trapezius significantly earlier than healthy controls (42.7 msec. after deltoid) (Table 4.4.1).

Hypothesis 4

Results for these analyses are presented in Table 4.9.1 and 4.9.2 and Figures 4.3a, 4.3b and 4.3c. This hypothesis tested the group difference for humerothoracic angle corresponding to deactivation of muscles across conditions. The humerothoracic elevation angle where serratus anterior was deactivated was significantly higher (~9°) in the subjects with impingement (36.4°) as compared to healthy controls (27.7°) across all conditions (df = 1/47, F-ratio = 6.69; p = 0.013). There was also a significant main effect of condition such that the serratus anterior muscle was deactivated much later in the range of arm lowering in the loaded condition (27.6°) and the condition after repetitive motions (29°) as compared to the unloaded condition (39.6°) (df = 2/94; F-ratio = 26.0; p<0.001) in both groups. There were no group differences for humerothoracic elevation angle associated with muscle deactivation of upper and lower trapezius. However, there was a significant condition main effect with both muscles showing significantly lower humerothoracic elevation angles before deactivation during the loaded condition (~20°) and after repetitive motions (~15°) as compared to unloaded trials (p<0.05) (Table 4.9.2).

Hypothesis 5

Descriptive results for these analyses are presented in Tables 4.4.4 and 4.4.5. The ANOVA results are described in Table 4.10.1 & 2 (elevation) and 4.11.1 & 2 (lowering) and Figures 4.4 a-f.

Scapular Internal/External Rotation

No significant interactions were found between normalized pectoralis minor length and any of the factors in the 3 way ANCOVA for scapular internal/external rotation during the elevation phase. Normalized pectoralis minor length was a significant covariate but the interpretation of results did not change with its presence in the model. Hence, the simple model was retained without the covariate. No significant group differences were found for scapular internal/external rotation during elevation of the arm. There was a significant 2 way interaction between condition and angle of elevation (df = 6/282; F-ratio = 20.02; p<0.001) such that scapular internal rotation during the unloaded condition was significantly less than scapular internal rotation during the loaded and after repetitive motions at 30°, 60° and 90° of humerothoracic elevation. There were no differences between the loaded and after repetitive motion conditions at all angles. The scapular internal rotation position at 30° was significantly lower (34.96°) than at 60° (37.55°) and 90° (38.35°) across all conditions. The scapular internal rotation position was different (~2°) between 60° and 90° only for the unloaded condition. The scapular internal rotation position at 120° was less (~3.2°) than that at 90° of humerothoracic elevation for all conditions.

During the lowering phase, normalized pectoralis length had a significant interaction with angle of elevation and hence it was nested with angle for ANCOVA (Appendix 13). The inclusion of the covariate did not change any interpretation of results and hence it was not retained in further analysis and a simpler model without the covariate was used. No significant differences were found between groups. However, the condition and angle of elevation factors demonstrated significant main effects. Scapular internal rotation during the unloaded condition (34.3°) was significantly less than scapular internal rotation during the loaded condition (36.8°) and the after repetitive motion condition (37.1°) (df = 2/94; F-ratio = 11.24, p<0.001). The scapula was in a significantly less internally rotated position at 120° (33.3) as compared to other angles (Table 4.11.1& 2 and Figures 4.4 a and b). Also, there was more internal rotation at 60° (37.8°) as compared to that at 30° (35.9°) of humerothoracic elevation.

Scapular Upward Rotation

There was a significant 3 way interaction between group, condition and degrees for the analysis of scapular upward rotation during the elevation phase (df = 6/282, F-ratio = 2.47; p value = 0.024). The follow up pair wise comparisons showed that the groups were closest to demonstrating significant differences for the unloaded condition at

120° of humerothoracic elevation (p = 0.076) with the healthy subjects showing higher (~3.35°) scapular upward rotation than subjects with impingement. The scapular upward rotation at 60° and 90° was significantly lesser (~2.5°) during the unloaded condition as compared to the loaded condition for the healthy subjects and at 90° and 120° for the subjects with impingement (~3°). The scapular upward rotation in healthy subjects showed no significant differences between the loaded condition and the condition after repetitive motion at all angles whereas the scapular upward rotation during the loaded condition was lesser (~2°) than that the after repetitive motion condition in subjects with impingement at 60° and 90° of humerothoracic elevation. For both groups, the scapular upward rotation during the unloaded condition was significantly lower from the condition after repetitive motions at 60°, 90° and 120° of humerothoracic elevation. Across all conditions and both groups, the scapular upward rotation increased from 30° (4.9°) to 120° (35.6°) of humerothoracic elevation (Table 4.10.1 & 2, Figure 4.4 c).

During the lowering phase, there was a significant interaction between condition and angle of humerothoracic elevation (df = 6/282; F-ratio = 12.01; p = <0.001). The scapular upward rotation was significantly higher (~1-2°) during the unloaded condition as compared to the loaded and after repetitive motion condition at 30° of humerothoracic elevation for both groups. The scapular upward rotation during the loaded condition was lower (~1.5°) that that during the condition after repetitive motions at 60° and 90° of humerothoracic elevation. The scapular upward rotation during the unloaded condition was higher (~2°-2.5°) than that during the condition after repetitive motion at 30° and 120° of humerothoracic elevation. Across all conditions and both groups, the scapular upward rotation decreased from 120° (36.2°) to 30° (5.3°) of humerothoracic elevation (Table 4.11.1 &2, Figure 4.4 d).

Scapular Tilt

The data was not normally distributed for scapular tilting, despite all attempted transformations (squares, cubes, exponential, log, inverse, square-root). The data from 3 subjects (2 subjects with impingement and 1 healthy) were outliers and hence were removed from further analysis which then rendered the data to have a normal distribution. Normalized pectoralis minor length showed a significant interaction with angle of elevation for only elevation phase analysis. When considered as a covariate, it was subsequently nested within angle. However, the inclusion or exclusion of normalized pectoralis minor length in the statistical model, although a significant factor, did not change the interpretation of results for group differences. The ANCOVA result is included in Appendix 13.

For the analysis of the elevation phase without the covariate, a significant interaction between condition and angle of elevation (df = 6/264; F-ratio = 4.52; p <0.001) was present. The scapular tilt during the unloaded condition was not significantly different from that during the loaded conditions at all angles. However, after repetitive motion, there was significantly more anterior tilt as compared to the loaded condition at 90° and 120° of humerothoracic elevation (p<0.05). Under all conditions there was a significantly increasing posterior scapular tilt from 30° (9° anterior tilt) to 120° (2.12° of posterior tilt) of humerothoracic elevation (Table 4.10.1 &2, Figure 4.4 e).

The 3 way ANOVA for the lowering phase analysis similarly showed a significant interaction between condition and angle of elevation (df = 6/264; F-ratio =

4.14; p<0.001). The scapular tilt during unloaded and loaded conditions showed a significantly less anteriorly tilted position as compared to the after repetitive motion condition for 60°(~1.1°), 90°(~1.5°) and 120°(~2°) of humerothoracic elevation. The scapular tilt during the unloaded condition was not significantly different from the loaded conditions at all angles except at 30° of elevation with the scapular anterior tilt being significantly higher (1.4°) during the loaded condition. Follow up pair-wise comparisons for angle effects showed that the scapula assumed an increasingly anteriorly tilted position while lowering the arm across all conditions (from ~4° posterior tilt to ~9° anterior tilt) (Table 4.11.1 &2, Figure 4.4 f).

Hypothesis 6

Results for these analyses are presented in Table 4.12.1 and 4.12.2a and b. The data was analyzed on log transformed data. For elevation phase analysis, a significant 3 way interaction was found between factors (df = 4/180; F-ratio = 2.67; p = 0.034). Hence pair wise comparisons were made using contrasts. During the increment between 30° to 60° and 60° to 90° of arm elevation, the slope significantly decreased from the unloaded (2.63) to the loaded (1.75) condition and after repetitive motion (1.66) condition for healthy subjects signifying increased scapular contribution. Similarly, for subjects with impingement the slope for the unloaded condition (1.75) was significantly less than that for the loaded condition (2.60) and the after repetitive motion condition (1.98). No significant differences were found between the loaded condition and the after repetitive motion condition across all increments and both groups. No differences across groups or conditions were found for the 90° to 120° increment.

Additional analysis on slope for elevation phase from 30° to 120° also showed no significant interactions but showed significant differences between the unloaded condition (2.40) and the other 2 conditions (1.89 and 1.81). The scapulohumeral rhythm values during the loaded and after repetitive motion conditions were not different from each other (Table 4.12.2b).

Hypothesis 7

A log transformation was used to result in a normal distribution of data. Descriptive results for these analyses are presented in Table 4.4.6. The ANOVA results are described in Tables 4.13.1 & 2 (elevation) and 4.14.1 & 2 (lowering) and Figures 4.5a-c.

Hypothesis 7 a

This hypothesis predicted group differences for serratus anterior activity. During the elevation phase, there was a significant interaction between condition and motion increment (df = 4/188; F-ratio= 30.13; $p<0.001$). Follow up pair wise comparisons showed that the unloaded condition was different from the loaded and after repetitive motion conditions at all increments (~25%) and the serratus anterior activity during the loaded condition was significantly lower than that after repetitive motions for the 30°-60° increment (3.5%). There were no significant group differences for serratus anterior activity (Tables 4.13.1 & 4.13.2).

For the lowering phase, there was a significant interaction between condition and motion increment (df = 4/188; F-ratio = 9.18; $p<0.0001$). Follow up pair wise comparisons showed that the unloaded condition was different from the loaded and after repetitive motion conditions at all increments (~13%) and the serratus anterior activity during the loaded condition was significantly lower than that after repetitive motion for the 60°-30° increment (<2%). There were no significant group differences across conditions and motion increments (Tables 4.14.1 & 2, Figure 4.5a).

Hypothesis 7 b

The hypothesis predicted a group by condition interaction for the lower trapezius activity during the lowering phase. There was a significant interaction between condition and motion increment (df = 4/188; F-ratio = 13.34; p<0.001). Follow up pair wise comparisons showed that the unloaded condition was different from the loaded and after repetitive motion conditions at all increments (~16%). There were no significant differences between the loaded condition and the condition after repetitive motions and no significant differences between the groups (Tables 4.14.1 & 2, Figure 4.5b).

Summary of Hypothesis Testing

Hypothesis 1: Under all conditions, the latencies of scapular muscles (upper trapezius, lower trapezius and serratus anterior) as compared to anterior deltoid will show a feedforward contraction only for healthy subjects and not for people with impingement syndrome. This hypothesis was partially supported for the after repetitive motion condition for serratus anterior relative latency where there were a significantly higher percentage of feed forward contractions in healthy subjects as compared to subjects with impingement. Significant group differences were also found for the upper and lower trapezius muscle during the unloaded condition but were in opposite direction to the hypothesis.

Hypothesis 2:

Hypothesis 2 a: The absolute latency of serratus anterior and lower trapezius will be significantly higher in people with impingement as compared to healthy individuals. This hypothesis was refuted. Serratus anterior absolute latency approached significance for group differences in the direction as expected with healthy subjects showing lower values than subjects with impingement.

Hypothesis 2 b: The absolute latency of all muscles will be significantly delayed in both groups after repetitive motion as compared to the unloaded condition. This hypothesis was partially supported. The absolute latency of upper trapezius increased after the repetitive motion condition whereas absolute latency of lower trapezius decreased significantly in the loaded and the after repetitive motion conditions in healthy subjects and absolute latency of serratus anterior decreased in both groups in the loaded and after repetitive motion conditions.

Hypothesis 2 c: Lower trapezius absolute latency will decrease with loading as compared to the unloaded condition of the arm in both groups. This hypothesis was supported. The absolute latency of lower trapezius decreased in both groups during the loaded condition as compared to the unloaded condition.

Hypothesis 3

Hypothesis 3 a: The serratus anterior activation will be followed by a significantly slower activation of upper trapezius and then by lower trapezius in healthy individuals under all conditions. This hypothesis was partially supported. The order of muscle recruitment for the unloaded condition was serratus anterior and upper trapezius followed by lower trapezius for both groups. For the other two conditions, the serratus anterior and lower trapezius preceded upper trapezius activation for both groups.

Hypothesis 3 b: Under the loaded condition, the relative latency of scapular muscles will significantly decrease as compared to the unloaded condition for subjects with impingement. This hypothesis was partially supported. The relative latency of upper trapezius increased and lower trapezius decreased in the loaded conditions for both groups.

Hypothesis 3 c: Across conditions, there will be a significant delay in relative latency of serratus anterior and lower trapezius and significantly shorter relative latency of upper trapezius in people with impingement as compared to healthy subjects. This hypothesis was partially supported. The relative latency of upper trapezius was significantly less in subjects with impingement as compared to healthy subjects. The hypothesis was refuted for lower trapezius and serratus anterior analyses.

Hypothesis 4: The angular value of humeral elevation when each scapular muscle will be deactivated will be significantly lower in healthy subjects as compared to people with impingement. The difference between groups will be significantly lesser for loaded conditions and after repetitive motion. This hypothesis was partially supported. The humeral angle corresponding to the muscle deactivation was higher for serratus anterior across all conditions in subjects with impingement as compared to healthy subjects.

Hypothesis 5: Under the unloaded condition, there will be differences observed in kinematic descriptors for scapular tilt, internal rotation and upward rotation between subjects with and without impingement. Subjects with impingement will show decreased upward rotation, increased internal rotation and decreased posterior tilt in both elevation and lowering phases. This hypothesis was refuted. The subjects did not show significant differences for scapular kinematic variables between groups across all conditions.

Hypothesis 6: After repetitive motions, the scapulohumeral rhythm will show differences across groups such that increased scapular contribution will be seen in subjects with impingement after repetitive motion. This hypothesis was partially supported. The scapulohumeral rhythm showed differences with an increased scapular contribution in the after repetitive motion condition for both groups.

Hypothesis 7

Hypothesis 7 a: Under all conditions serratus anterior will show significantly decreased activity as a magnitude percentage of referenced contraction over motion increments from 30°-60°, 60°-90° and 90°-120° in subjects with impingement as compared to healthy subjects. This hypothesis was refuted. The muscle activity of serratus anterior did not show significant group differences for elevation or lowering phases.

Hypothesis 7 b: The lower trapezius will show significantly decreased activity in people with impingement as compared to healthy subjects during the lowering phase for the unloaded condition. This hypothesis was refuted. The lower trapezius activity did not show significant group differences during the lowering phase across conditions.

Additional Exploratory Analyses

1. Relaxed standing position differences: There were no significant differences between groups in the scapular kinematic variables during the relaxed standing position as measured before or at the end of the data collection protocol (Table 4.15). The glenohumeral elevation angle was significantly different between groups with the subjects with impingement showing significantly higher GH elevation angle (10.1°) as compared to healthy subjects (5.4°) ($p = 0.02$) in the initial relaxed standing position. There were no significant differences between the initial and final rest position data for the scapular internal/external rotation, scapular tilt, glenohumeral plane of elevation and axial rotation values. However, the final rest position data had significantly less scapular upward rotation averaged over both groups (2°) than the initial rest position. Also, the glenohumeral elevation was significantly less elevated in the final rest position (~2°) than in initial rest position data.

2. **Peak elevation kinematic differences**: Results for these analyses are presented in Tables 4.16.1 & 2. There were no differences between the groups for the scapular upward rotation and tilting at peak humerothoracic elevation. The scapular internal/external rotation showed a trend toward difference ($p = 0.07$) with the subjects with impingement showing more internal rotation (31°) than healthy subjects (25.7°). There was a significant effect ($p<0.001$) of condition across both groups for scapular tilt with tilt position during the unloaded and the loaded conditions being significantly more posterior (~6-7°) than during the condition after repetitive motions (3.1°).

There was a significant effect (p = 0.006) of condition across both groups for glenohumeral elevation with the unloaded condition showing significantly more elevation (100.3°) than the loaded condition and condition after repetitive motions (~97°).There was a significant interaction between group and condition for the analysis of glenohumeral plane of elevation. The groups were different after the repetitive motion and loaded conditions with the subjects with impingement closer to the scapular plane (~6-9° anterior to the scapular plane) than healthy subjects (~11.5° anterior to the scapular plane). There was a significant effect (p<0.001) of condition across both groups for glenohumeral axial rotation with the unloaded condition showing significantly less external rotation (56.8°) than the loaded condition (60.1°) and condition after repetitive motion (59.2°).

3. Glenohumeral kinematic variables

Descriptive statistics results are described in Tables 4.4.4. Results for these analyses are presented in Tables 4.17.1 & 2 (elevation) and 4.18.1 & 2 (lowering) and Figure 4.6 a-f.

a. <u>Glenohumeral angle of elevation</u>: There was a significant interaction between condition and angle of elevation (df = 6/282; F-ratio = 33.26; p value = <0.001) for the analysis of the elevation phase. Follow up analysis showed that the glenohumeral elevation was higher (~4-5°) during the unloaded condition as compared to that during the other 2 conditions at 90° and 120° of elevation. There was no difference between the loaded condition and after repetitive motion conditions at all angles (Tables 4.17.1 & 2).

There were also significant interaction effects between condition and angle of elevation (df = 6/282; F-ratio = 26.74; p value = <0.001) during the lowering phase.

Follow up analysis showed that the glenohumeral elevation was lower (~2°-5°) during the unloaded condition as compared to the other 2 conditions at 30°, 60° and 120° of humerothoracic elevation. There were no differences between the loaded condition and after repetitive motion conditions at all angles (Tables 4.18.1 & 2)

b. Glenohumeral Plane of elevation: During arm elevation and lowering, there was a significant group effect and a significant interaction between condition and angle of elevation. During both phases, the subjects with impingement had their arm significantly less anterior to the scapular plane as compared to the healthy subjects (~5-6°). For both phases, the glenohumeral plane of elevation during the unloaded condition was significantly more anterior (~2° to 10°) to the scapular plane as compared to the other 2 conditions at all angles. The plane of elevation was significantly more anterior (1.5°) during the loaded condition as compared to the condition after repetitive motion for the elevation phase (Tables 4.17.1 & 2). The loaded condition and after repetitive motion condition were not different from each other for any angles during the lowering phase (Tables 4.18.1 & 2).

c. Glenohumeral axial rotation: For the elevation and lowering phases, there was a significant interaction between condition and angle of elevation. For the elevation phase, the axial external rotation during the unloaded condition was significantly lesser than that during the other 2 conditions at 30° (~10°) and 60° (~5°) of elevation. The axial external rotation was greater (~2 °) during the loaded condition as compared to the after repetitive motion condition at 90° and 120° of elevation (Tables 4.17.1 & 2). For the lowering phase, the external rotation during the unloaded condition was significantly less (~3°-10°) as compared to the loaded condition at all angles but it was significantly less as compared to the after repetitive motion condition only at 30° (9.1°) and 60° (5.6°) of elevation. The axial rotation progressively increased with increasing arm elevation across all conditions and phases. There were no significant group differences (Tables 4.18.1 & 2).

4. EMG analysis of Upper Trapezius: The results for these analyses are presented in Tables 4.13.1 & 2 (elevation) and 4.14.1 & 2 (lowering) and Figure 4.5 c. The analysis was performed on log transformed data after the data from one subject was excluded. It was an extreme outlier due to considerably weak contraction during the recording of the MVC. There was a significant interaction between condition and motion increment (df = 4/184, F-ratio=12.29; p-value<0.001). Pair-wise comparison showed that the muscle activity was significantly less (~25-30%) during the unloaded as compared to the loaded condition and after repetitive motion condition at all increments. The loaded condition was not different from the after repetitive motion condition for the 90°-120° increment. The upper trapezius muscle activity significantly increased (3-8%) from 30°-60° to the 60°-90° increment across all conditions. The muscle activity was not significantly different between the 30°-60° and 90°-120° increment for the loaded and after repetitive motion condition. There were no significant group differences (Tables 4.13.1 & 2).

For the lowering phase analysis, the muscle activity was significantly less (~13%) during the unloaded condition as compared to the loaded condition and the after repetitive motion condition at all motion increments. There were no significant differences between the loaded condition and the after repetitive motion condition. There were no significant group differences (Tables 4.14.1 & 2).

5. Analysis of fine wire EMG of serratus anterior: The latencies between the surface and fine wire EMG signals were estimated for 2 subjects only as the baseline activity was visually determined to be excessively high in the other subjects. The latency as estimated by the surface signal was consistently delayed in one subject (mean 10 msec.; range: 5-25 msec.) as compared to the fine wire signal. For the second subject, the average difference between the latencies was close to zero but the difference ranged between -32.4msec. to 38.8msec. The cross correlation coefficients were analyzed for 4 subjects. The data from one subject could not be analyzed for cross correlations as there was a loose connection between the fine wire and preamplifier electrode and the activity during arm lowering was not collected. The cross correlation coefficients were similar between $\text{Xcorr}_{\text{LD-SA surface}}$ (0.79) and $\text{Xcorr}_{\text{LD-SA finewire}}$ (0.78) (Appendix 14, Figure A). The percentage MVC activity of latissimus dorsi averaged across all subjects during the unloaded condition was 1.3%, 2.7% and 3.5% across motion increments respectively. The percentage MVC activity of latissimus dorsi averaged across subjects during loaded conditions was 1.8%, 3.6% and 6% across motion increments respectively (Appendix 14).

6. Logistic Regression: For the unloaded condition at 60° of humerothoracic elevation, scapular tilt, scapular upward rotation, glenohumeral elevation and glenohumeral plane of elevation showed significance for predicting group differences (Tables 4.19a and b). The probability of being in the impingement group was significantly less if the subjects had an increased scapular anterior tilt (chi square = 6.8, Odds ratio = 0.34; p value = 0.009) and more anterior plane of elevation (chi square = 8.3, Odds ratio = 0.49; p value = 0.004), and greater if they had decreased scapular upward rotation (chi square = 5.7, Odds ratio = 3.62; p value = 0.017). These directions of differences are consistent with the descriptive results of the variables (Tables 4.4.4 & 5). The probability of being in the impingement group was also significantly higher if the subjects had a lower glenohumeral elevation (chi square =5.25, Odds ratio = 3.3; p value= 0.02) which was the only outcome which did not agree with the descriptive result for the variable. Descriptively, the subjects with impingement showed a higher glenohumeral elevation angle. This difference may be explained by the strong association between this variable and scapular upward rotation. As both were included in the model, the estimate of the co-efficient for glenohumeral elevation was affected by scapular upward rotation. The scapular upward coefficient can be interpreted as the influence of scapular upward rotation after adjusting for glenohumeral elevation.

For the unloaded condition at 90° of humerothoracic elevation, scapular tilt, and glenohumeral plane of elevation were retained in the model. The probability of being in the impingement group was significantly lower if the subjects had an increased scapular posterior tilt (chi square = 3.87, Odds ratio = 0.66; p value = 0.049) and more anterior plane of elevation (chi square = 6.83, Odds ratio = 0.85; p value = 0.009).

For the analysis at 30 and 120 degrees, no kinematic variable could distinguish group differences significantly (Appendix 15).

7. Group comparison of variances: The result of this analysis is described in Table 4.20. No significant differences were found between the variances of upper trapezius relative latency with or without the data from 2 subjects which were considered as outliers.

Significant differences were found between the variances of lower trapezius both with and without the data from 2 subjects which were considered as outliers (p = 0.006 and p<0.001). The variance of the healthy subjects was higher (SD = 129.4 versus 72.4 with all subjects; and 92.8 versus 39.4 without outliers) than that of subjects with impingement.

Significant differences were found for serratus anterior relative latency variance only in the condition after removing data from 2 outliers (p = 0.01). The subjects with impingement had higher variance (SD = 78) than healthy subjects (SD=44).

8. <u>Analysis of sub groups within the impingement group</u>

a. <u>Based on type of impingement</u>: The results of this analysis are described in Appendix 16 a. Seven subjects were included in the sub group who possibly presented with only internal type of impingement, 8 subjects were included in the subgroup who possibly presented with only subacromial type of impingement and the remaining 9 were sub grouped as ambiguous. The internal impingement sub group presented descriptively with a more internally rotated scapular position (~3-4°) than the healthy and other subgroups during both phases and all conditions and angles. All impingement sub groups presented with lesser scapular upward rotation (~2-3°) than the healthy subjects for the unloaded and loaded conditions. Slightly larger differences (~5-6°) were found for the unloaded condition during the lowering phase. However, the subacromial only sub group presented with higher scapular upward rotation (1-2°) than the healthy group for the after repetitive motion condition. The internal impingement only subgroup had higher (~3-4°) posterior tilt than other impingement sub groups and the healthy subjects at 120° for both phases during the unloaded and loaded conditions. The subacromial impingement only and ambiguous sub groups presented with lesser scapular posterior tilt (~2-3°) than the healthy subjects for the after repetitive motion condition.

b. Based on level of involvement: The results of this analysis are described in Appendix 16b. Scapular internal rotation was not different between the subgroups during both phases and all conditions except the after repetitive motion condition when the lower involvement sub group had higher internal rotation than the other subgroup (~3-4°). The lower involvement sub group also tended to have lesser scapular upward rotation across all conditions and phases as compared to the high involvement group. There were some differences at 120 degrees for scapular tilt values (~2°) with the lower involvement sub group showing less scapular posterior tilt (Appendix 16 b).

CHAPTER 5

DISCUSSION

Validity of surface electrodes for serratus anterior EMG

The current study found that a surface EMG electrode provides a good estimate of serratus anterior muscle activity. The latissimus dorsi activity across motion increments during elevation of the arm in the unloaded condition (1.3-3.5%) and loaded conditions (1.7-6%) was insignificant as compared to serratus anterior activity (6-24% for unloaded and 10-37% for loaded condition). Hence any cross talk from latissimus dorsi, even if present in the surface EMG electrode signal for serratus anterior would be minimally affecting the interpretations. The cross correlation (~0.8) between the latissimus dorsi signal and the fine wire electrode signal of serratus anterior suggests that there is co-contraction of muscles rather than cross talk. These values are similar to those obtained for the cross correlation values between latissimus dorsi and surface serratus anterior signal (Appendix 14). There were some differences observed in the latencies as estimated by the fine wire and surface electrode (mean differences: 0-10 msec.). These would be predictable as the signal is picked up by the fine wire electrode earlier than the surface electrode. However, a few trials also found a delayed estimation of latency in the fine wire signal as compared to the surface which could be attributed to the difference in the baseline activities of the signals. Overall differences in latencies were quite small relative to the within subject trial to trial variability and the between subject variability, suggesting surface electrode use is not a substantive contributor to the variability seen.

A definite advantage of fine wire electrodes would be a relatively lesser effect of cardiac artifacts in the signal. However, there are challenges with maintaining proper contact with the preamplifier throughout the range and number of trials. Also, the electrode wires can move during the course of the movement or with repeated movements, altering the pickup area.

Comparison with past studies

The current study explored the possibility of differences in muscle latency and deactivation time in people with and without impingement which may be suggestive of differences in motor programs and may suggest any mechanistic relation to development or aggravation of shoulder impingement. As previously indicated, there are only a few studies with comparable data for shoulder muscle latencies.

Comparisons of absolute latency of scapular muscles as reported by Wadsworth et al.[52] to the current study are difficult to make due to differences in the method of estimation of latency. Wadsworth et al.[52] estimated latency from the time of onset of motion in contrast to a light cue. They found that serratus anterior was recruited after upper trapezius and followed by lower trapezius activation in healthy controls. The current study found that there were no differences between activation of serratus anterior and upper trapezius in the unloaded condition. The lower trapezius in their study[52] was activated approximately after 15° of humeral elevation. No attempt was made to make a similar comparison in this study as it is known that people of different sizes and body mass have a different initial position at rest. However, consistent with the earlier study, the current study found that lower trapezius was activated after the other scapular muscles. The Wadsworth study[52] found a significantly higher between subject variability in subjects with impingement. The current study found a difference in variance only for lower trapezius and serratus anterior. However, the results for lower trapezius are in contrast to those found by Wadsworth et al.[52] as the current study found a higher between subject variance for healthy subjects. When data from the adjusted sample (after removing outliers) was analyzed, the serratus anterior variance in subjects with impingement was higher than healthy subjects concurring with the results of Wadsworth et al.[52] The current study did not find any differences in the within subject (trial to trial) variance as measured by SEM and ICC values across the two groups when compared descriptively.

The study of Moraes et al.[50] used a light cue to calculate absolute latency of trapezius and serratus anterior and hence the results are more appropriate to compare to the current study. The values obtained from their study and the current study yield similar results for the data from healthy subjects for the onset of upper trapezius and serratus anterior. The current study found 299 ± 84 msec. as compared to 280 ± 70 msec. reported by Moraes et al.[50] for upper trapezius absolute latency in healthy subjects; and 296 ± 130 msec. as compared to 320 ± 50 msec. for serratus anterior absolute latency in healthy subjects. The values for subjects with impingement were not so similar with Moraes et al.[50] reporting 540 ± 120 msec. and 630 ± 130 msec. for the upper trapezius and serratus anterior absolute latency. The current study found much lower values with 311 ± 174 msec. for upper trapezius and 327 ± 136 msec. for serratus anterior absolute latency in subjects with impingement. Moreas et al.[50] found that lower trapezius on average activated nearly 1-1.5 seconds after the light cue. The current study did not find such delayed responses. There were 2 subjects in the current study that had substantially higher values that rendered the data not normal and hence their data were excluded. Their average lower trapezius absolute latency values during the unloaded condition were 560 msec. and 902 msec. If these two subjects were to be included, the average absolute latency for the groups would increase in healthy subjects from 357.5 msec. to 379.3 msec. (~23 msec. difference) and in the impingement group from 334.5 to 343.9 msec.(~9 msec. difference). These adjusted average values are still small as compared to the results from Moraes et al.[50]. This may be due to a difference in electrode placement site for lower trapezius. Moraes et al.[50] used visual inspection and site of maximum bulk to place their electrode which may have been different from the site used in the current study (between the spine and inferior scapular angle) affecting the latency results. These differences could also be attributed to a slower speed of elevation used in the Moraes et al.[50] investigation as they asked the subjects to move with a self selected comfortable speed.

Moraes et al.[50] did not find any group differences possibly due to inadequate power owing to a sample size of 10 subjects in each group. In the current study the serratus anterior showed a trend for group differences ($p = 0.075$) with people with impingement tending to show a delayed contraction. The current study also failed to find significant group differences because the muscle latencies were analyzed across all conditions which increased the between subject variance. The other difference between the two studies is that the current study descriptively found a larger within group between subjects variability for serratus anterior absolute latency (~130 msec.) in healthy subjects and a smaller within group between subjects variability for lower trapezius absolute latency (118 msec.) as compared to 410 msec. for the subjects with impingement.

To find any effect of hand dominance, both earlier studies[50, 52] compared side to side differences in subjects and did not find significant differences between the sides of both groups. The current study did not look at evaluating latency for both sides of the subjects. However, there were equal numbers of right and left dominant subjects and equal numbers of dominant and non-dominant sides tested in both groups. This would remove the possibility of hand dominance affecting the current results.

The anterior deltoid absolute latency was estimated by Hodges and Richardson[42] in a study to investigate spine muscle latency. They had found anterior deltoid to be active around 188 msec. after a light cue. This is less than the latency found in the current study (281.5 msec. averaged over both groups). One of the reasons may be a difference in the method for data analysis. Hodges et al.[42] used an increase of 2 standard deviations from baseline as the threshold to define activation whereas the current study used the criteria of 3 SDs. Another reason for the longer latency could have been that people with shoulder pain would tend to be slower to react overall as a protective phenomenon may be due to muscle guarding and pain. However, the current study did not observe group differences for anterior deltoid latency (294.5 msec. in the impingement group versus 265.1 msec. in the healthy group). The variability in the current study was much higher than that reported by Hodges et al.[42] who performed 10 repetitions in their experiment and took an average of 10 trials. This possibly decreased the variability of muscle latency for all studied muscles in their study[42]. Only 5 trials of motion were collected under each condition in the current study.

Interpretation of results

Within and between subject variability

Trial to trial variability was estimated using ICCs (3, 1) and SEMs. The trial to trial ICC values have never been previously reported for muscle latency data. These reliability values were found to be extremely poor across conditions (ICC values <0.3) which indicates high within subject or between trial variability for both groups. The standard errors of measurements were approximately 100 msec. for all relative muscle latencies. There was no pattern of trial variation, however, with no statistical significance observed for trial for any latency parameter.

The high variability between trials can be explained partially by the following factors. The electromagnetic system created electrical interference with the EMG data collection. Though multiple notch filters were used, the possibility of random artifacts due to the electromagnetic system can not be completely negated. Another phenomenon observed in the current study was that muscles occasionally showed a spurt of minimal activity after which the same muscle showed activity less than the defined threshold. The muscle then activated to show continued contraction and maintained activity beyond the threshold (Figure 5.1) throughout the rest of the motion trial until it deactivated. The onset of the muscle was defined in the current study as the time after which the muscle maintained contraction beyond the threshold for a continued time period. So the observed phenomenon would not identify as muscle activation for the initial “spurt” of minimal activity. Such a phenomenon has not been described in any of the muscle latency literature but anecdotal confirmation of similar behavior was noted through discussion with other researchers. As it is a possibility that other studies have ignored such a phenomenon and have estimated latency at the initial point when the muscle shows the first spurt of activity, their latency estimates would be lower than that found by the current study[42]. It can be argued that if latency is only an indication of a motor program, the first spurt of activity should be considered. But in the current study, we defined meaningful activation of a muscle only as the point after which it maintained its activity beyond the threshold. This may have also affected our analysis results by increasing the within subject variability. As the variability is large, taking averages across trials ensured that the mean represents the behavior of the muscle more appropriately than an individual trial. However, a higher number of trials per condition would have ensured a better representation.

This high variability between the trials may suggest that the same motion may be performed using different recruitment strategies. This suggests that either there is a lack of a fixed motor control program governing the recruitment order; or there is a flexibility of using different orders that allows subjects to avoid detrimental effects of overuse. Usually motor programs have relative timing as an invariant parameter such that despite changes in the overall speed and force, there are no changes in the order of events[103]. Wadsworth et al. reported a higher variability in the symptomatic group as compared to the healthy group and the current study found similar differences for serratus anterior and contrary results for lower trapezius. Wadsworth et al.[52] speculated that the high variability would increase the risks for injury in subjects who are exposed to repetitive trauma. The effects of increased variance between trials needs to be addressed in future studies. With lack of differences for consistent patterns of recruitment order in subjects with and without impingement, it is difficult to conclude on the association of high between trial variability and pathology.

Lower Trapezius absolute latency

The lower trapezius absolute latency showed group by condition interaction with the unloaded condition significantly later than the after repetitive motion condition only for the healthy subjects. The subjects with impingement failed to show the earlier recruitment of lower trapezius for the after repetitive motion condition. But on the contrary, the chi square analysis revealed that people with impingement had a significantly higher percentage of feedforward contraction of lower trapezius during the unloaded condition. It may be possible that people with impingement use a strategy of using the upper and lower trapezius earlier in unloaded conditions. Healthy subjects, however, tend to use the muscle earlier with increased challenge of lifting a weight after having performed repetitive motions. This change was not seen in people with impingement which may be suggestive that they can not make the necessary adjustments, perhaps because they fatigue the muscle. Cools et al.[104] found the reaction time for activation of lower trapezius in healthy volunteers before and after a fatiguing protocol. They found after fatigue that there was an increase in latency for all other fibers of trapezius except the lower trapezius. The current study also found that lower trapezius was recruited earlier after repetitive motion in healthy subjects. Ebaugh et al.[108] studied the effects of a fatiguing protocol involving some static and some dynamic tasks on scapular muscles of healthy individuals and found that the lower trapezius muscle did not show any EMG signs of fatigue with activities involving raising the arm. Histologically, the lower trapezius has a higher percentage of type 1 or fatigue resistant type of fibers whereas the descending component of upper trapezius has a higher percentage of type 2 fibers[132, 133]. It can be speculated that people with impingement tend to fatigue their lower trapezius due to earlier activation with less stressful tasks and fail to recruit this muscle earlier after performing repetitive motions as seen in healthy subjects.

Exploratory analysis

There are no published studies as yet which have studied relative latencies of scapular muscles as compared to the prime mover. The current study did not find any differences between the groups for upper trapezius latency across conditions. However, there was a significant difference between the conditions such that the muscle was found to be delayed in conditions with a weight in hand. This happened because the muscle showed a higher baseline activity when a weight was held in hand, thus increasing the threshold to determine activation. This may have happened to stabilize the shoulder girdle against the downward pull of the weight. Hence the computer algorithm which used the standard deviation from the baseline activity found a considerably delayed onset (Figure 5.2). Some other strategies which may be used to estimate latency in data with high baseline activity could be increased smoothing or using a lower threshold (1 SD instead of 3 SD). Though these strategies would yield results closer to that obtained by visual estimation, it can also be argued that these parameters (smoothing or threshold levels) should be the same across all muscles and groups in an analysis. Conversely, it can also be argued that the muscle is already active before initiating the movement. Hence, this study explored the possibility of differences in upper trapezius relative latency during the unloaded condition only. A two sample t-test was performed between groups and a significant difference was found such that people with impingement showed a significantly lesser upper trapezius relative latency than healthy subjects (Table 4.4.2/Figure 4.2a). This earlier latency of upper trapezius is concordant with results of many earlier studies which have shown higher amplitude of activity of upper trapezius in people with impingement [12, 33]. It is possible that people with impingement depend on their upper trapezius to elevate their clavicle in an attempt to elevate their arm[134]. This may be indicative of a different motor strategy used by people with impingement.

One of the other reasons for not being able to find group differences for relative muscle latency was that the power analysis was initially done with variance for the unloaded condition only. However, the final analysis included muscle latencies for all conditions which substantially increased the pooled standard deviation for group comparisons (Table 4.4.2). Hence additional exploratory analysis was performed only for the unloaded condition for lower trapezius and serratus anterior as well.

The group differences as identified by a 2 sample t test approached significance ($p = 0.053$) for lower trapezius relative latency with the subjects with impingement showing an earlier activation (44.3 msec.) as compared to healthy subjects (85.8 msec.). This is in concordance with the chi square analysis performed on trials showing feed forward contraction.

The groups did not show a significant difference during the unloaded condition for serratus anterior relative latency.

Deactivation Times of Scapular Muscles

Very few studies have investigated muscle deactivation times. Deactivation time is estimated by computer algorithms similar to those used to estimate latency. Radebold et al.[135] used an algorithm which estimated muscle deactivation of spinal flexors and extensors as the first frame of the moving window average of 44 msec. when activity reduced by the defined threshold of baseline plus 1.4 SD. The study[135] used a larger window width than that used for estimating onsets (25 msec.). We also used a moving window width of 25 msec. for calculating the onset but using the same width for estimating deactivation time was resulting in considerably earlier deactivation than would be made by visual estimation. Hence the window width was increased to 100 msec. The algorithm was then run for another 2000 windows after the first detected reduction in muscle activity below threshold (baseline plus 3 SD) to completely ensure that the muscle activity continued to be below the threshold.

All muscles showed contraction longer until the arm was lowered further in the condition with a weight in hand. This may show that during lowering of the arm, the scapular muscles need to control the scapula against the continuing torque of the deltoid which would be increased in the weighted conditions. The serratus anterior showed a group difference across all conditions such that people with impingement deactivated the muscle significantly earlier in the range of elevation. This is in agreement with the clinical finding of the current study and earlier studies[16] that dyskinesia was mostly present in subjects with impingement in the lowering phase, especially at lower elevation angles. Biomechanically, the possibility of impinging the supraspinatus tendon against the acromion is greatest at lower elevation angles (~30-40° of humerothoracic elevation)[63]. At higher angles, the tendons are rotated past the acromion and there is more bone to bone (humeral lateral edge to acromion) approximation. The serratus anterior is believed one of the most important muscles for scapular mobility and control[47, 65, 134] and its earlier deactivation during lowering of the arm may affect scapulothoracic motion in a detrimental way. The lack of posterior tilting and upward rotating torques normally produced by serratus anterior may alter scapular kinematics in a way as to cause further impingement of the rotator cuff tendons.

The current study provides a unique opportunity to look at kinematics simultaneously with muscle activity. During the eccentric phase in both groups, there was an increase in scapular anterior tilt and internal rotation positions after repetitive motion which is consistent with the finding of observing increased dyskinesia or prominence of the scapular medial border/inferior angle. However, the muscles tended to continue their contraction longer in this condition after repetitive motion which seems to be contradictory to the kinematic result. The serratus anterior was the only muscle which showed a group difference for deactivation which can partly explain the changes in scapular kinematic parameters for that group. It is a possibility that scapular kinematic position variables studied at 30 degree increments of 120°, 90°, 60° and 30° are not sensitive for finding group differences whereas muscle humeral angle corresponding to deactivation times is a more sensitive indicator. The only variable which came close to showing group differences was scapular upward rotation in the eccentric phase ($p = 0.12$). It is a possibility that scapular variables may show differences before and after scapular muscle deactivation times (especially serratus anterior) which is visibly observed as dyskinesia but this phenomenon can not be estimated by a positional kinematic analysis at 30 degree intervals.

Very few studies which have used muscle latency as an indicator for evaluating motor control programs have tried to relate latency changes to mechanistic reasons for development of pathology. The literature concerning delayed activation for certain core stabilizers of the spine has found that such delayed responses are present in low back pain subjects regardless of the nature of pathology[42]. It was argued that the delayed activation of the stabilizing muscles left the spine unprepared for the external perturbations[136]. However, the muscles do activate even though late and so it is topic of continued argument as to how the delay of activation in microseconds may mechanistically affect the stability of the spinal structures. It is a possibility that this delay in activation is indicative of the altered motor programs used by people with pain[42]. It is difficult to draw parallels in studies of scapular kinematics for more than one reason. The scapular muscles are not static stabilizers of the scapula but they are responsible for both stability and the correct mobility of the scapula. Their delayed or earlier activation may or may not be related to mechanistic causation of shoulder impingement but may be suggestive of the changes in motor control programs caused by pain. To further investigate such a scenario, it may be more meaningful to study the latency of muscles in subjects with impingement after removing pain by anesthetic injections.

The association of altered motor programs and pathology also needs to be addressed in future studies. The presence of differences between subjects with and without impingement for muscle recruitment or deactivation patterns indicates alteration in the motor control strategies. However, it can not be concluded whether these changes are causative mechanisms or compensatory changes. It is possible that these changes cause certain changes in the scapular kinematics such as to compromise the potential space for the rotator cuff tendons. However, it is equally possible that either pain or fatigue associated with repetitive use brings about these changes in subjects with impingement as compensatory strategies.

The current study has hypothesized that failure to recruit the proximal scapular group of muscles before the prime mover or anterior deltoid could indicate a lack of or alteration of motor control strategies. However, the results indicate that subjects with impingement tend to recruit the trapezius muscle earlier than the healthy subjects. This may also suggest alterations of the motor program such that it suggests over reliance or earlier activation in certain muscles. The motion of raising and lowering the arm is accomplished by a coordinated and organized muscle activity maintaining the relative balance between the muscle groups. The over reliance on one group or muscle over others affects this intricate balance and suggests alteration instead of a complete failure of a motor program.

Kinematics

Reliability and trial effects

Very good to excellent[131] ICCs (3, 1) were found for all scapular kinematic measures indicating high trial to trial reliability. The highest SEM values (beyond 3°) were found for scapular upward rotation (Table 4.2). The effect of trial was also calculated using a repeated measures ANOVA with trial as a within subject factor. It was significant in both concentric and eccentric phases for scapular upward rotation;

glenohumeral elevation and glenohumeral plane of elevation such that there were differences from the first to the third trial onward in the unloaded condition (1-2°). As this can be interpreted as an effect of performing repetitive motion also captured by the effect of another condition, no further analysis was done. If the analysis were to be performed between the first 2 trials of the unloaded condition and after the repetitive condition, the differences in the conditions would have increased slightly (~1°). Average data from all trials were used for statistical analysis.

Scapular kinematics

Correlations between various demographic factors such as age, height, weight and BMI and scapular kinematic variables were calculated. These correlations tended to be very low (Appendix 12 F) and showed no pattern between groups or condition and hence were not further considered as covariates for further analysis. As the plane of elevation was different between groups, correlations were estimated between the plane of elevation at 90 degrees of humerothoracic elevation and kinematic variables (Appendix 12 G). The only strong correlation which was observed was with glenohumeral plane of elevation which is a variable which depends on humerothoracic plane of elevation and scapular internal/external rotation. And so, it was not retained for further analysis.

The current study found a non significant pattern of decreased upward rotation (~2-3°) in subjects with impingement across all conditions, especially at higher elevation angles. There were no group differences for scapular internal/external rotation and tilting. However, the subjects with impingement showed a tendency for higher internal rotation at peak elevation (Tables 4.16.1 & 2). Also, logistic regression analysis showed that the subjects with impingement tended to have lower scapular upward rotation and increased anterior tilt at 60° for the unloaded condition.

The lack of larger scapular differences might be attributed to the difference in plane of elevation for the groups. Though the groups were instructed to raise their arms in flexion, it was found that subjects with impingement raised their arm closer to the scapular plane than true flexion. Also, subjects from both groups drifted away from flexion (sagittal plane) when challenged with weight in hand. The possible explanation for this phenomenon is that glenohumeral muscles would be most efficient during scapular plane elevation and hence when challenged with a weight, subjects would tend to move closer to this plane. It may also be a compensatory technique used by subjects with pain to move away from positions of impingement. Multiple correlations of humeral plane with other kinematic variables were calculated but as they were very low and did not tend to show any pattern, humeral plane was not considered as a covariate for further analysis. This suggests that the planar elevation difference between groups cannot explain a lack of group kinematic differences for other variables.

The functional rating scores for the subjects with impingement (DASH=16.8 and Penn shoulder score=49.1) indicate that the level of symptomatic involvement was not very high. The highest possible DASH score is 100 (more dysfunction) and a highest possible Penn shoulder score is 60 (less dysfunction). It is a possibility that the subjects with impingement did not experience much pain and discomfort (average NPR scores-1 to 2.3 across conditions) during the motion trials. If the symptomatic group had subjects who were more severely involved, it might have helped to find more significant group differences.

Lastly, lack of homogeneity amongst groups can not be denied. The subjects with impingement included those who may have either had subacromial impingement or internal impingement, or both types together. No investigation was done to confirm the site of impingement and the lack of objective clinical methods for this differentiation could result in having a group of subjects who had different mechanisms or compensations related to shoulder impingement. In the current study, an exploratory attempt was made to subgroup the subjects based on history and a clinical examination. As the numbers of subjects in each sub group was low, it was difficult to perform any statistical analysis on the subgroups but the data was analyzed descriptively. There were some small differences within the sub groups with subjects behaving slightly different than the whole impingement group so as to attenuate any significant group differences with the healthy subjects. In the presence of subjects who could have different mechanisms for development of impingement, an analysis on the means would tend to dilute any group differences if present. A possible mechanistic factor which could be associated with subacromial type of impingement is an increased anterior tilt [18] and a possible explanation for internal type of impingement could be decreased scapular upward rotation. If the symptomatic group had a combination of subjects with both type of impingement, it is a possibility that the whole group did not behave in a consistent way to be significantly different from the healthy subjects.

Also, earlier studies have found contradictory results regarding group differences for scapular kinematics. Studies have found either a decreased[12, 27] or increased upward rotation[13] in people with impingement as compared to healthy controls. Some studies[14, 79] did not find any group differences. Also, scapular tilt results have been contradictory with some studies showing a decrease in posterior tilt[12, 14, 27] and one other study showing increased posterior tilt[13]. These disparities in results signify differences in the way people with impingement move. Their disparities have been associated with differences in the compensation or causation mechanisms of impingement, different techniques of data collection (radiography versus electromagnetic versus optotrak systems versus Moire technique), different motions (multiple static versus continuous), and different subject samples (only males versus both genders). So future studies need to address this issue of finding objective ways to differentiate subjects based on their mechanisms of impingement and clinicians need be careful to apply the results of research studies which may have included subjects with these different mechanisms of impingement.

The effects of weight on kinematics

There have been various studies which have studied scapular kinematics in unloaded versus loaded conditions[68-71]. DeGroot[71] studied the scapular positions and scapulohumeral rhythm in 7 static positions from initial (0°) to peak elevation (180°) with multiple weights in hand (0, 0.9, 1.9 and 2.9 Kgs). They did not find any differences in any 3D positions or scapulohumeral rhythm with load in hand. The subjects in their study[71] held static positions for prolonged periods as the examiners palpated for anatomical landmarks in various positions. The authors speculated that the lack of difference across conditions was due to the high variability at higher angles which was due to difficulty in palpating landmarks and difficulty of subjects to hold static elevated positions. Doody et al.[68] used goniometry to estimate the scapulohumeral rhythm with and without a weight in hand. The study[68]found that the scapular contributions increased during the biomechanically most challenging interval (60°-90°) and in the loaded condition. Ludewig et al.[12] studied the effect of load in people with and without impingement. They[12] found that a load in hand had a significant interaction with either group or angle of elevation only for scapular internal rotation such that people with impingement showed more internal rotation in loaded conditions than the healthy subjects. Pascoal et al.[70] investigated the effect of loads (0 to 4 Kgs) on scapular positions in 3D in 3 different planes (flexion, scapular plane and coronal plane abduction). They found increased scapular upward rotation in the loaded condition in abduction and flexion. McQuade et al.[69] studied the effect of load on scapulohumeral rhythm and found that the scapular contributions increased with load initially in the range of motion (up to 40%). The current study found that scapular upward rotation increased significantly at higher angles (60°, 90° and 120°) and internal rotation increased (at 60° and 90°) with load in hand. The current study observed an increase in upward rotation as measured as position change across conditions and as a change in scapulohumeral rhythm. There was an increased scapular contribution in loaded conditions as compared to the unloaded conditions. The trends in the current study of increase in scapular upward rotation agree with those observed with earlier studies[68-70]. The current study results are different from those obtained by Ludewig et al.[12] for the current study did not find group differences or interaction with load conditions. The differences in kinematics observed between unloaded and loaded conditions in the current study are in the range of 2-3°. The effect of load is analyzed in a 3 way ANOVA across group, condition and degrees and hence the effect of load was calculated averaged across both groups increasing the sample size to 49. This is larger sample size than other previous[69-71] studies and hence the current study found small differences between conditions which were possibly missed in other investigations.

The effect of fatigue

Many studies have looked at the effect of fatigue on scapular kinematics. The study by McQuade et al.[110] asked subjects to raise their arm with weight for multiple repetitions till they could not continue further and checked fatigue by decrease in MVC. Ebaugh et al.[108] used a fatigue protocol with multiple activities till the subject could not continue or showed compensatory strategies and analyzed median power frequency in data analysis to confirm effects of fatigue. The study by Tsai et al.[109] used an isokinetic dynamometer and measured torque to check fatigue in glenohumeral external rotators. Ebaugh et al.[108] reported that the fatiguing protocol used in their study caused increased scapular upward rotation (at 60°, 90° and 120°), external rotation (at 90° and 120°) and clavicle retraction (at 60°, 90° and 120°), and decreased humeral external rotation. Su and colleagues[36] found that the scapular kinematics did not differ between impingement and healthy groups before a swim practice but there were significant reductions in scapular upward rotation at 45°, 90° and 135° after practice in persons with impingement. McQuade et al.[110] found that subjects demonstrated increased scapular contributions during arm elevation after a fatiguing protocol. Tsai et al.[109] found decreased posterior tilting (up to 90°), external rotation (up to 120°) and upward rotation (up to 60°). There is a difficulty in comparing the results from the current study with the earlier studies as the after repetitive motion trials were collected with a weight in hand. Also, it can not be conclusively said that subjects perceived fatigue and for which muscles as it was not measured.

The trends for scapular kinematics from unloaded to loaded conditions continued in the after repetitive motion condition. If the experiment were to be designed to find the effect of fatigue on muscle latency, the subjects would have needed to do multiple repetitions till they no longer could continue, include some means of checking reduction in strength (dynamometer) or change in median power frequency, measure latency after fatigue with no load in hand and subsequently analyze how different muscles fatigued to the activity and associate that with changes in latency. The current study limited the number of repetitions to avoid further irritation to the subjects with pain. The effect of fatigue could be investigated in future studies.

Pectoralis Minor length

The correlations between normalized length of the pectoralis minor and scapular kinematics showed a pattern such that correlation coefficients were negative with scapular internal/external rotation and positive with scapular tilt. This indicated that increased pectoralis minor rest length was associated with less scapular internal rotation and greater posterior tilt as would be assumed by its anatomical alignment[30]. The height normalized pectoralis minor length was retained as a covariate for scapular tilt analysis between groups and conditions. The covariate had a significant main effect and a significant interaction with angle of elevation and hence it was nested within the factor. This also showed significant effects signifying that the effect of pectoralis length differed across elevation angles but did not affect analysis of group differences (Appendix 13). In the current study subjects were excluded if they had postural kyphosis as estimated by visual examination, and hence larger differences in pectoralis minor length were not present. However, the significant interactions of the muscle length with the scapular variables suggest that it should be an important factor to consider for future studies and physiotherapeutic interventions for shoulder impingement patients.

Glenohumeral Kinematics

The subjects were instructed verbally to raise their arm in front of their body (approximately in the sagittal plane) and no other means were used to guide them strictly. The people with impingement raised and lowered their arm on an average 21° anterior to the scapular plane, whereas the healthy subjects raised their arm around 26° anterior to the scapular plane. Theoretically, this difference could occur due to changes in the scapular plane, or humerothoracic plane of elevation. An analysis was then done to determine any group difference of humeral plane at 90° in the thoracic reference frame. Subjects with impingement moved further away from the true sagittal plane with a difference of 8° during the unloaded condition and ~4-5° different during the loaded condition. There were no scapular internal rotation differences between groups suggesting that the difference in glenohumeral plane of elevation was present due to lifting the arm in a plane away from the sagittal plane. This may be a protective/compensatory mechanism used by people with pain to avoid more painful positions. Most impingement tests[137, 138] intend to increase the contact of rotator cuff muscles either with the coracoacromial arch or glenoid in flexion[137]. It is therefore a possibility that subjects tend to avert these positions by moving away from true sagittal plane elevation. However, the healthy subjects also drifted towards the scapular plane in loaded conditions and it is possibly mostly due to increasing the efficiency of glenohumeral muscles.

Recently another study (unpublished) found that subacromial volume decreased most substantially during arm elevation in the scapular plane with humeral internal rotation. During flexion, possibly posterior translation protects against impingement. It can be speculated that the tendency of subjects with impingement to move closer to the scapular plane is a contributory mechanism to the development of pathology. Neither of these possibilities can be proven by the current or previous studies and future studies could investigate the association of plane of elevation and pathology.

Summarizing the glenohumeral kinematics from the current study, significant interactions were present between condition and angle of elevation for all glenohumeral variables such that there was a decreasing glenohumeral elevation angle (indicating more scapular contribution), decreasing plane of elevation (indicating moving away from true flexion) and increasing external rotation in the loaded condition and after repetitive motions as compared to the unloaded condition.

Clinical Implications

The current study observed some group differences between people with and without impingement. The results could help physical therapists to focus their treatment for improved scapular control during the eccentric lowering phase in patients by training of the serratus anterior muscle to maintain its contraction longer. As this muscle is believed extremely important for maintaining correct scapular motion on the trunk, training of this muscle especially in the lowering phase might improve the kinematic behaviors such as reducing dyskinesia. This could be achieved by using some EMG feedback techniques such that patients learn to maintain their muscle activity as they lower their arm. As studies have found that greater rotator cuff approximation to the acromial undersurface occurs at lower elevation angles[63], controlling scapular mobility during arm lowering could possibly decrease rotator cuff impingement. The current study did not include high speed motions such as throwing where the stronger eccentric contractions of the glenohumeral muscles might pull the scapula further into undesirable positions (anterior tilt/ internal rotation) in the absence of stabilizing forces from serratus anterior.

A phenomenon previously noted by previous investigators is an increased use of upper trapezius in subjects with shoulder impingement[12, 33]. This is kinematically associated with an increase in clavicle elevation. It is a common clinical finding to visually observe patients attempting to shrug the shoulder while elevating their arm. The current study did not find group differences for muscle activity but found group differences in relative upper trapezius latency with subjects with impingement recruiting the muscle earlier than controls. Physical therapists could consider attempting to train patients with impingement to voluntarily reduce their upper trapezius activity before and during arm motions. This could be initiated by first making the subjects perceive the difference of the relaxed versus contracted state of upper trapezius. Then training initially could be started with EMG biofeedback as subjects could be taught to maintain a low activity in upper trapezius as the arm is elevated. Then the challenges could be increased with increasing speed of motion or adding a weight and continuing to maintain lower activity of the muscle. This could be complemented with visual feedback from mirrors or tactile feedback such that subjects would recognize their faulty shoulder shrugging (clavicle elevation) motions while moving their arm in space.

Physical therapy approaches to treatment for impingement subjects involve strengthening of serratus anterior, trapezius, and other rotator cuff muscles but in most cases[43, 44, 46, 139] the amount of weight or repetitions are not adequate for muscle hypertrophy based strength changes. The strength changes associated with muscle hypertrophy occur when muscles contract against resistance equivalent to 80-90% of one repetition maximum for multiple repetitions (8-12)[140]. But usually the recommended exercises for shoulder impingement rehabilitation includes 3 sets of 10 repetitions with a moderately low weight (variable as used in previous studies), multiple angle isometric holds with or without weight, exercises against body weight (push-ups) or theraband exrercises[43]. These exercises are recommended either daily or in sessions (2-3) per week[43]. It is evident that the recommended exercises are moderately low intensity exercises not intended for muscle hypertrophy. The approach of rehabilitation should consider modification from strength training such that the outcome of therapy is not focused on changes in strength but rather changes in movement patterns or muscle behavior and building the appropriate motor program. Physical therapists could use EMG biofeedback measures to train patients to use their serratus anterior and learn to relax and use less of upper trapezius[46, 139]. Additionally, endurance training exercises should be considered for the lower trapezius muscle, because the subjects with impingement failed to recruit the muscle after the repetitive motion condition as done by healthy subjects which possibly can be explained by lack of endurance. This endurance training might be achieved by considering exercises with multiple repetitions and low intensity.

The results of the current study also support continued therapeutic focus on stretching the pectoralis minor muscle. Although not associated with group differences, this muscle was again seen in the current study to be associated with scapular tilting and internal rotation in directions that are presumed detrimental[30].

There was a trend towards less upward rotation (~3°) in people with impingement as compared to controls especially at higher angles. Some authors speculate[13, 17] that it is a compensatory mechanism used by patients to increase the subacromial space whereas others consider it to be a mechanism which causes impingement by reducing the space. Although the impact of altered scapular upward rotation has been speculated on, only one published study has assessed the association of altered scapular upward rotation and the subacromial space[17]. Karduna et al.[17] attempted to measure the displacement of the humeral head toward the acromion in various scapular orientations in a cadaveric study and found that an increase in upward rotation was associated with decreased space for superior displacement. However, there were concerns with interpreting these results due to not having a standard starting position for all comparisons in the study. Also, the most approximation occurring at the tested 90° position is likely contact with the lateral humerus instead of the rotator cuff tendons[63]. So, it can not be conclusively proven from the study if a decrease in scapular upward rotation is detrimental or not. Also, it is a possibility that changes in scapular upward rotation at higher angles of elevation affects the contact of rotator cuff tendons with the glenoid which is not yet studied. If improving the magnitude of upward rotation in impingement subjects is a therapeutic goal, strength training of the serratus anterior might help to avoid the reduction in upward rotation seen in the current and previous studies[12, 27] and thus help to reduce possible abrasion against the glenoid or coracoacromial arch.

Limitations of the current study

The current study has several limitations. One of the limitations was the loss of trials due to cardiac or motion artifacts (Figure 3.6a and b). On average across conditions, the number of lost trials in each group for upper trapezius was 7, for lower trapezius was 10 and for serratus anterior was 15. The muscles were close to the chest wall and hence cardiac artifacts were observed in many trials. This theoretically could be reduced in future studies by using fine wire electrodes instead of surface electrodes especially for serratus anterior. The surface EMG signal quality deteriorates in the presence of high subcutaneous tissue or fat which could as well be avoided by using fine wire electrodes. Hence only subjects with BMI less than 28 were included in the study. Nevertheless, there are difficulties with using fine wire electrodes relating to difficulty in maintaining the electrode connections with the preamplifier and fixed electrode spacing over many trials especially as the arm makes contact with the electrode wires at lower elevation angles. Also, the number of trials recommended should be higher than 5 per condition to provide a more representative average value. Other studies investigating muscle latencies have used up to 10 trials[42]. As the current study was looking at more than one condition involving lifting weight for multiple repetitions (up to 15), the number of trials per condition was limited to 5 to avoid risk of further irritation and pain for subjects with impingement.

Surface tracking for kinematic descriptors is prone to error due to skin slip. However, only subjects with BMI less than 28 were included in an attempt to reduce this error. There were no differences for weight or BMI between subjects in both groups and hence any error associated with surface skin slip can be considered non-systematic and random. It would not create group differences but it may obscure those that do exist. With the exception of the analysis at peak elevation, the remaining analysis was limited to 120° of humerothoracic elevation below which the errors associated with skin slip are less[22].

There were no data collected in the current study for clavicular motion and hence no acromioclavicular motion data available. The scapular motion with a trunk reference frame is not true anatomical joint motion. Actual sternoclavicular and acromioclavicular joint motion might help to better understand the associated changes in muscle activities. For example, a change in upper trapezius activity directly acting at the sternoclavicular joint could be directly associated with the change in the kinematics at that joint. However, accurate surface tracking of the clavicle has validity and reliability limitations[75]. Also, for making comparisons to published literature, most past studies investigated the scapulothoracic motions and it was considered appropriate to study the scapula in a trunk reference frame.

The speed of motion and plane of elevation were not strictly controlled in this study. There were no differences found in the speed of elevation between groups, nor any moderate or strong association of speed with the dependent variables (Appendix 12 B, C and E).

There was a significant difference in plane of elevation between groups. Hence it was considered as a covariate for kinematic analysis. It did not have significant impact on the interpretation of results and hence was not retained in further analysis.

The electromagnetic system can interfere with the EMG data collection. Multiple notch filters were used to remove noise artifact. Also, the subjects were positioned in such a way that the electrodes were at a maximum possible distance away from the electromagnetic system. However, for obtaining data from subjects with a higher BMI, higher gains were used which amplified the signal as well as the noise from the electromagnetic system. This interference could affect the latency estimation as it may cause intermittent artifacts in the signal. Another possible disadvantage of the interference could be inaccuracies in the analysis of muscle activity (up to 5%). Along with notch filters, the baseline activity magnitude obtained from the rest file was subtracted from the activity files. It would be impossible to collect electromagnetic kinematic data simultaneously with EMG data without having these limitations and appropriate measures were taken to avoid any systematic errors between groups or conditions with the data analysis.

The position for estimating the maximum voluntary contractions was done at 60° of humerothoracic elevation in an attempt to avoid the painful arc, which has been traditionally defined as the range when pain due to subacromial impingement is experienced[4]. However, some of the subjects with impingement experienced significant pain (average NPR score ~2; range = 0-7) which would affect their capability for producing strong voluntary contraction. This would cause higher estimation of their muscle activity as a percentage of MVC and might affect group comparisons as the percentage of MVC would tend to be higher in subjects with impingement who demonstrated a weaker MVC.

Finally the motions tested in the current study may not be representative of functional or overhead sports tasks and hence generalization of results to other motions and conditions needs to be done cautiously.

Future Implications of current study

The results of the current study may help to improve future studies which intend to investigate muscle latency of scapular muscles and investigate this relationship of motor control and pathology. There can be ways in which to avoid several of the limitations observed in the current study. The protocol changes which could be considered to decrease within subject variance include increasing the number of trials, reducing loss of trials due to cardiac artifacts by using fine wire EMG and collecting kinematic data using either a camera system or imaging technique instead of an electromagnetic system to avoid interference. In addition, subjects could be matched on exposure along with other variables such as hand dominance, height, weight, etcetera.

The lack of a precise clinical criterion to identify dyskinesia in subjects limits the ability to include only those subjects who have impingement due to motion abnormalities. Subjects can have impingement due to anatomical abnormalities (hooked acromion)[29] or secondary to instability. It is a possibility that the group of subjects with impingement was heterogeneous and thus as many clear and consistent group differences were not obtained. Future studies need to address the issues related to developing valid and reliable clinical measures for assessing motion related pathologies in the shoulder.

Also, it should be acknowledged by clinicians that not all patients with shoulder impingement would have motion related pathology or that they would respond positively to therapy focused at corrections of scapular motions. And hence, amongst the inclusions criteria, a positive scapular muscle assistance test (SAT)[47] could be included. The test is considered positive if the patient perceives reduction in symptoms after the examiner assists the scapula into more posterior tilt and upward rotation.

There is also a lack of validated and confirmed clinical tests to differentiate internal and external impingement subjects. The current study potentially included both types of subjects and it is a possibility that people with different types of impingement have different underlying mechanisms for the development of the pathology. These differences and their associated mechanisms or compensations need to be understood further by future studies.

However, the results of this study can form a basis for further investigations. The effect of pain on motor control strategies could be studied. It is believed possible to alleviate pain in subjects with true subacromial impingement by using subacromial injections. Muscle latencies and deactivation times could be studied before and after such injections. As another line of investigation, the latencies could be studied only in healthy individuals between those who have dyskinesia and those who do not to investigate if latency and muscle deactivation times differ in people with different kinematic patterns. Also, this study could be expanded to determine latency differences in rotator cuff muscles.

Similar studies could also be completed using different populations such as subjects with rotator cuff tears. Other motions and speeds of motion could be tested for finding differences in motor control strategies. Future studies could investigate the effect of any intervention (exercises, biofeedback training) on muscle latency and kinematics and investigate if interventions translate to relief of symptoms and impaired functional status in patients.

Summary and Conclusions

The study examined the differences of muscle latency, muscle deactivation times, scapular kinematics and associated muscle activity in people with and without shoulder impingement. The data from 25 healthy subjects and 24 subjects with shoulder impingement was included. The study was done under three different conditions: unloaded, loaded and after repetitive motions. The muscle latency of the scapular muscles was analyzed with reference to an external light cue and to the activation of anterior deltoid. The humerothoracic angle corresponding to the deactivation time for all muscles was evaluated. Also, scapular and glenohumeral kinematic variables were analyzed for the elevation and lowering phase. Simultaneously muscle activity as a percentage of MVC was described for the elevation and lowering phase. There were no differences between the groups based on demographic variables. The results of the hypothesis are summarized at the end of the results section.

The study also validated the use of surface electrodes for recording the serratus anterior muscle activity. The results of the study helped to identify that the variability of muscle latency is variable between trials even for healthy subjects. This was shown by the low ICC and high SEM values. This is in contrast to the good to excellent reliability values for the kinematic variables analyzed at 30 degree increments.

The subjects with impingement showed an earlier activation of upper and lower trapezius for the unloaded condition and later activation of serratus anterior in conditions after repetitive motions. The subjects with impingement also showed an earlier deactivation of the serratus anterior muscle across conditions as compared to the healthy subjects. This has important clinical implications as motion related abnormalities or dyskinesia is a phenomenon better perceived during the lowering phases. Also, subtle changes in the scapular kinematics at lower angles could affect the proximity of the rotator cuff tendons to the acromion.

Across all conditions, all muscles showed an earlier activation and a delayed deactivation during the conditions with weight in hand. The condition after repetitive motions did not significantly contribute to show group differences except for lower trapezius absolute latency which decreased in healthy subjects but not in subjects with impingement.

The predictors for group differences for kinematic variables were scapular upward rotation, scapular tilt and glenohumeral plane of elevation as determined by logistic regression at 60°. The subjects with impingement showed lesser scapular upward rotation, more anterior scapular tilt and a less anterior plane of elevation. The loaded and the after repetitive motion condition showed differences from the unloaded condition for almost all variables. The scapular upward rotation increased, posterior tilt decreased and internal rotation increased. The glenohumeral plane of elevation became less anterior with the conditions with weight in hand in both groups. The muscle activity defined as a percentage of MVC did not show group differences across conditions.

TABLES

Table 3.1 Descriptive statistics for the demographic variables and functional rating scores

	Healthy (n=25)	**Impingement (n=24)**	**P value**
Age	32.20 (9.8)	35.09 (12.5)	0.38
Gender	13 Females, 12 Males	10 Females, 14 Males	0.47
Height (cms)	171.50 (8.5)	173.77 (10.3)	0.41
Weight (kgs)	67.85 (9.9)	70.85 (11.2)	0.33
BMI	23.16 (2.6)	23.34 (2)	0.78
Hand Dominance	23 Right Sided	23 Right sided	0.58
Tested Side	8 Left, 17 Right	7 Left, 17 Right	0.83
DASH scores	16.84 (9.6)	1.67 (3.1)	<0.001
Penn Shoulder Score	49.1 (6.5)	59.1 (1.8)	<0.001

Table 4.1 ICC and SEM for scapular kinematic variables

Variable	Condition	Group	Phase	ICC	SEM
Scapular Internal/External Rotation	Unloaded	Healthy	Elevation	0.94-0.95	1.5°-2.2°
	Unloaded	Healthy	Lowering	0.90-0.92	2.2°-2.6°
	Loaded	Healthy	Elevation	0.90-0.92	2.1°-2.8°
	Loaded	Healthy	Lowering	0.93-0.94	1.7°-2.7°
	ARM	Healthy	Elevation	0.91-0.97	1.4°-3.0°
	ARM	Healthy	Lowering	0.94-0.95	2.0°-2.8°
Scapular Upward Rotation	Unloaded	Healthy	Elevation	0.84-0.91	1.8°-2.9°
	Unloaded	Healthy	Lowering	0.80-0.83	3.1°-3.5°
	Loaded	Healthy	Elevation	0.85-0.88	2.1°-2.3°
	Loaded	Healthy	Lowering	0.86-0.90	2.3°-2.4°
	ARM	Healthy	Elevation	0.88-0.90	1.9°-2.5°
	ARM	Healthy	Lowering	0.87-0.90	2.7°-3.2°
Scapular Tilt	Unloaded	Healthy	Elevation	0.95-0.98	1.6°-2.0°
	Unloaded	Healthy	Lowering	0.91-0.97	2.2°-2.6°
	Loaded	Healthy	Elevation	0.95-0.98	1.6°-2.1°
	Loaded	Healthy	Lowering	0.94-0.97	1.8°-2.0°
	ARM	Healthy	Elevation	0.97-0.97	1.5°-2.1°
	ARM	Healthy	Lowering	0.95-0.98	1.7°-2.2°
Scapular Internal/External Rotation	Unloaded	Impingement	Elevation	0.94-0.95	1.8°-2.4°
	Unloaded	Impingement	Lowering	0.94-0.96	2.0°-2.3°
	Loaded	Impingement	Elevation	0.93-0.98	1.4°-3.2°
	Loaded	Impingement	Lowering	0.94-0.97	1.7°-2.9°
	ARM	Impingement	Elevation	0.94-0.97	1.8°-2.1°
	ARM	Impingement	Lowering	0.94-0.97	1.9°-2.7°
Scapular Upward Rotation	Unloaded	Impingement	Elevation	0.86-0.89	1.8°-2.7°
	Unloaded	Impingement	Lowering	0.90-0.93	2.3°-3.0°
	Loaded	Impingement	Elevation	0.90-0.91	2.0°-2.6°
	Loaded	Impingement	Lowering	0.89-0.90	2.7°-3.3°
	ARM	Impingement	Elevation	0.90-0.93	2.2°-2.6°
	ARM	Impingement	Lowering	0.90-0.92	2.4°-2.9°
Scapular Tilt	Unloaded	Impingement	Elevation	0.91-0.96	1.6°-2.7°
	Unloaded	Impingement	Lowering	0.87-0.96	2.3°-2.8°
	Loaded	Impingement	Elevation	0.93-0.96	1.7°-2.8°
	Loaded	Impingement	Lowering	0.93-0.97	1.9°-2.6°
	ARM	Impingement	Elevation	0.95-0.98	1.9°-2.5°
	ARM	Impingement	Lowering	0.91-0.98	2.4°-2.6°

Where, ARM = after repetitive motion condition

Table 4.2 ICC and SEM for latency

Variable	Condition	Group	ICC	SEM (in msec.)
Upper Trapezius (Absolute latency)	Unloaded	Healthy	0.17	121.7
	Loaded	Healthy	0.49	118.5
	ARM	Healthy	0.50	142.6
	Unloaded	Impingement	0.35	118.5
	Loaded	Impingement	0.36	114.2
	ARM	Impingement	0.26	123.5
Lower Trapezius (Absolute latency)	Unloaded	Healthy	0.20	159.6
	Loaded	Healthy	0.42	96.0
	ARM	Healthy	0.27	144.5
	Unloaded	Impingement	0.28	135.7
	Loaded	Impingement	0.41	97.0
	ARM	Impingement	0.46	124.6
Serratus Anterior (Absolute latency)	Unloaded	Healthy	0.22	129.7
	Loaded	Healthy	0.39	109.9
	ARM	Healthy	0.13	155.4
	Unloaded	Impingement	0.25	164.2
	Loaded	Impingement	0.41	108.0
	ARM	Impingement	0.29	136.4
Upper Trapezius (Relative latency)	Unloaded	Healthy	0.13	101.0
	Loaded	Healthy	0.30	121.6
	ARM	Healthy	0.29	112.5
	Unloaded	Impingement	NS	78.9
	Loaded	Impingement	0.16	102.7
	ARM	Impingement	0.13	79.7
Lower Trapezius (Relative latency)	Unloaded	Healthy	0.20	135.2
	Loaded	Healthy	0.17	103.0
	ARM	Healthy	0.43	95.3
	Unloaded	Impingement	NS	98.0
	Loaded	Impingement	0.20	92.1
	ARM	Impingement	0.29	105.3
Serratus Anterior (Relative latency)	Unloaded	Healthy	NS	80.3
	Loaded	Healthy	NS	110.7
	ARM	Healthy	NS	73.4
	Unloaded	Impingement	0.17	117.0
	Loaded	Impingement	NS	122.4
	ARM	Impingement	0.12	111.2

Where, NS = non significant F-ratio for the one way ANOVA,
ARM = after repetitive motion condition.

Table 4.3 Correlation coefficients between scapular kinematic variables and normalized pectoralis minor length

Group	Condition	Angle of elevation	Scapular IR/ER	Scapular UR	Scapular tilt
Healthy	Unloaded	30°	-0.37	-0.06	0.40
		60°	-0.36	0.07	0.41
		90°	-0.32	0.14	0.43
		120°	-0.23	0.19	0.35
	Loaded	30°	-0.37	-0.12	0.38
		60°	-0.33	0.11	0.41
		90°	-0.32	0.14	0.42
		120°	-0.25	0.05	0.39
	After repetitive motion	30°	-0.27	-0.14	0.39
		60°	-0.21	0.06	0.40
		90°	-0.14	0.00	0.40
		120°	-0.13	-0.09	0.35
Impingement	Unloaded	30°	-0.24	-0.21	0.21
		60°	-0.29	-0.26	0.33
		90°	-0.37	-0.20	0.41
		120°	-0.44	-0.05	0.45
	Loaded	30°	-0.27	-0.01	0.35
		60°	-0.29	0.01	0.46
		90°	-0.34	0.05	0.54
		120°	-0.39	0.19	0.55
	After repetitive motion	30°	-0.35	-0.12	0.29
		60°	-0.36	-0.08	0.40
		90°	-0.42	-0.04	0.47
		120°	-0.48	0.02	0.48

Where, Scapular IR/ER = Scapular internal/external rotation and
Scapular UR = Scapular upward rotation

Table 4.4.1 Mean and confidence limits for absolute latency of muscles (in milliseconds)

Muscle	Group	Unloaded condition	Loaded condition	After repetitive motion condition
Upper trapezius	Healthy (n=25)	286.89 (262.4-31.7)^{a}	298.32 (272.8-326.2)^{ab}	301.21 (275.5-329.3)^{b}
	Impingement (n=24)	278.92 (254.6-305.5)^{a}	318.55 (290.8-348.9)^{ab}	343.33 (313.4-376.1)^{b}
Lower trapezius	Healthy (n=25)	339.22 (310.8-370.2)^{a}	242.10 (221.8-264.2)^{b}	248.06 (227.3-270.7)^{b}
	Impingement (n=24)	325.40 (297.6-355.8)^{a}	265.08 (242.4-289.8)^{b}	297.62 (272.2-325.4)^{ab}
Serratus anterior	Healthy (n=25)	273.83 (253.4-295.9)^{a}	229.39 (212.3-247.9)^{b}	238.03 (220.3-257.2)^{b}
	Impingement (n=24)	303.84 (280.7-328.9)^{a}	268.95 (248.5-291.1)^{b}	278.64 (257.4-301.6)^{b}
Anterior deltoid	Healthy (n=25)	251.54 (235.6-268.5)^{a}	237.14 (222.1-253.2)^{a}	247.44 (231.8-264.2)^{a}
	Impingement (n=24)	277.97 (260.0-297.2)^{a}	262.47 (245.5-280.6)^{a}	284.58 (266.2-304.2)^{a}

Note: Different letters (‘a’, ‘b’) are assigned to signify differences across conditions ($p<0.05$). Hence, assignment of the same letter signifies no difference between the conditions and assignment of letters (‘ab’) signifies no difference relative to either ‘a’ or ‘b’.

Table 4.4.2 Mean and standard deviation for relative latency of muscles

Muscle	Group	Unloaded condition	Loaded condition	After repetitive motion condition
Upper trapezius	Healthy (n=24)	42.69 ± 61.8^{a*}	85.35 ± 98.8^{b}	67.99 ± 89.6^{b}
	Impingement (n=23)	-4.24 ± 45.5^{a*}	58.12 ± 70.9^{b}	61.64 ± 48.5^{b}
Lower trapezius	Healthy (n=24)	85.83 ± 92.8^{a}	0.01 ± 67.6^{b}	12.41 ± 19.7^{b}
	Impingement (n=23)	44.31 ± 39.4^{a}	13.55 ± 65.2^{b}	22.29 ± 92.9^{b}
Serratus anterior	Healthy (n=23)	8.88 ± 44.2^{a}	-3.05 ± 64^{a}	-12.82 ± 43.4^{a}
	Impingement (n=24)	28.58 ± 77.5^{a}	14.32 ± 75.8^{a}	1.52 ± 67.8^{a}

Note: Different letters (‘a’, ‘b’) are assigned to signify differences across conditions (p<0.05). Hence, assignment of the same letter signifies no difference between the conditions.

* signifies group difference (p <0.05) for a condition.

Table 4.4.3 - Percentage of trials with feed forward contractions

Muscle	Group	Unloaded condition	Loaded condition	After repetitive motion condition
Upper trapezius	Healthy (n=24)	56.64 %*	45.45 %	53 .04 %
	Impingement (n=23)	76.15 %*	48.54 %	49.54 %
Lower trapezius	Healthy (n=24)	45.45 %*	74.07 %	72.64 %
	Impingement (n=23)	62.73 %*	69.23 %	66.36 %
Serratus anterior	Healthy (n=23)	77.78 %	74.51 %	85.15 %*
	Impingement (n=24)	69.90 %	71.85 %	72.90 %*

* signifies group difference for a condition (p <0.05).

Table 4.4.4 Mean and standard deviation for scapular kinematic variables during elevation and lowering phases

Condition	**Phase**	**Angle**	**Scapular IRER**		**Scapular UR**		**Scapular tilt**	
			Healthy (n=25)	Impingement (n=24)	Healthy (n=25)	Impingement (n=24)	Healthy (n=24)	Impingement (n=22)
Unloaded	Elevation	30°	33.09° (6.2°)	32.02° (7.4°)	-5.84° (5.8°)	-4.24° (4.6°)	-9.24° (6.3°)	-8.78° (3.8°)
		60°	36.05° (6.9°)	34.88° (8.0°)	-14.68° (6.1°)	-13.04° (5.5°)	-6.08° (7.3°)	-5.26° (4.6°)
		90°	37.55° (7.8°)	36.88° (8.6°)	-25.72° (6.7°)	-22.82° (6.3°)	-3.30° (9.5°)	-1.96° (6.2°)
		120°	34.65° (8.6°)	35.32° (9.8°)	-35.28° (6.7°)	-31.93° (7.9°)	2.41° (12.3°)	3.36° (8.6°)
	Lowering	30°	35.09° (7.5°)	33.26° (7.7°)	-9.14° (6.7°)	-5.01° (7.3°)	-8.23° (6.3°)	-7.70° (4.8°)
		60°	36.37° (7.5°)	35.31° (8.4°)	-19.78° (7.2°)	-15.43° (8.9°)	-4.63° (8.6°)	-3.98° (6.5°)
		90°	35.48° (8.1°)	35.21° (9.0°)	-31.09° (7.3°)	-25.44° (9.8°)	-0.77° (11.8°)	0.25° (8.1°)
		120°	31.32° (8.8°)	32.27° (10.1°)	-37.90° (7.3°)	-33.28° (9.1°)	4.01° (13.2°)	5.90° (9.3°)
Loaded	Elevation	30°	35.74° (6.6°)	35.77° (8.5°)	-5.38° (5.1°)	-3.68° (5.9°)	-8.65° (6.2°)	-9.13° (4.1°)
		60°	37.98° (7.7°)	38.58° (9.4°)	-16.62° (5.7°)	-13.95° (6.7°)	-4.94° (7.2°)	-4.74° (4.8°)
		90°	38.17° (8.1°)	39.26° (10.7°)	-28.22° (5.7°)	-25.72° (7.4°)	-1.98° (9.7°)	-1.30° (6.8°)
		120°	33.97° (9.5°)	36.16° (11.7°)	-36.65° (6.4°)	-35.24° (8.6°)	2.63° (12.5°)	3.35° (8.7°)
	Lowering	30°	36.97° (6.3°)	36.95° (9.1°)	-5.33° (6.5°)	-2.73° (7.8°)	-9.79° (6.6°)	-8.99° (4.7°)
		60°	38.55° (7.4°)	39.01° (10.1°)	-17.89° (6.6°)	-15.14° (8.7°)	-4.95° (8.5°)	-3.70° (6.1°)
		90°	37.47° (8.7°)	38.11° (11.1°)	-29.52° (6.0°)	-26.66° (9.1°)	-0.97° (11.5°)	1.23° (7.8°)
		120°	32.86° (10.3°)	34.37° (11.4°)	-36.59° (7.2°)	-35.49° (9.6°)	3.61° (12.8°)	5.75° (8.5°)
After repetitive Motion	Elevation	30°	36.50° (7.8°)	36.63° (8.3°)	-5.14° (5.6°)	-5.23° (6.6°)	-9.61° (6.7°)	-9.15° (6.3°)
		60°	38.39° (8.2°)	39.43° (9.1°)	-17.29° (6.0°)	-15.97° (6.9°)	-6.49° (7.9°)	-5.78° (6.5°)
		90°	37.86° (8.3°)	40.38° (10.4°)	-29.67° (6.1°)	-27.73° (7.6°)	-4.06° (10.7°)	-3.61° (8.0°)
		120°	33.37° (9.7°)	37.25° (12.1°)	-38.04° (7.7°)	-36.60° (8.8°)	0.45° (12.6°)	0.55° (10.9°)
	Lowering	30°	36.49° (8.4°)	37.01° (8.4°)	-5.66° (7.7°)	-4.03° (7.7°)	-10.57° (7.2°)	-9.65° (6.4°)
		60°	38.57° (9.1°)	39.20° (9.6°)	-18.90° (8.1°)	-16.65° (8.0°)	-5.86° (9.1°)	-5.09° (7.3°)
		90°	37.64° (10.2°)	39.08° (10.8°)	-31.06° (7.9°)	-27.82° (8.4°)	-2.33° (11.6°)	-1.11° (8.8°)
		120°	32.98° (11.0°)	35.92° (12.0°)	-38.09° (8.1°)	-35.93° (9.2°)	2.13° (13.1°)	3.01° (10.1°)

Table 4.4.5 Mean and standard deviation for glenohumeral variables during elevation and lowering phases

Condition	**Phase**	**Angle**	**Angle of Elevation**		**Plane of Elevation**		**Axial Rotation**	
			Healthy (n=25)	Impingement (n=24)	Healthy (n=25)	Impingement (n=24)	Healthy (n=24)	Impingement (n=22)
Unloaded	Elevation	30°	-21.39°(6.7°)	-24.85° (5.2°)	23.26° (7.2°)	17.88° (6.1°)	-6.91° (14.4°)	-9.90° (17.0°)
		60°	-42.32° (7.6°)	-45.52° (7.1°)	36.27° (7.8°)	29.61° (7.5°)	-25.41° (15.4°)	-27.33° (17.5°)
		90°	-67.26° (7.8°)	-68.97° (7.9°)	36.78° (8.0°)	29.74° (8.9°)	-44.59° (16.3°)	-43.84° (17.9°)
		120°	-88.31° (6.6°)	-89.82° (8.2°)	25.63° (8.9°)	21.12° (9.8°)	-54.92° (16.1°)	-52.22° (17.1°)
	Lowering	30°	-17.51° (8.5°)	-23.36° (7.9°)	21.64° (7.9°)	17.63° (7.2°)	-2.82° (16.7°)	-6.01° (16.4°)
		60°	-35.36° (8.9°)	-42.01° (11.2°)	37.25° (9.0°)	31.55° (7.7°)	-19.61° (17.0°)	-22.65° (17.6°)
		90°	-60.43° (7.7°)	-65.68° (12.9°)	39.64° (9.9°)	33.62° (8.9°)	-40.28° (17.8°)	-39.07° (17.8°)
		120°	-85.49° (7.1°)	-88.39° (9.4°)	27.71° (10.8°)	24.86° (10.5°)	-53.93° (16.6°)	-50.16° (16.4°)
Loaded	Elevation	30°	-22.91° (6.0°)	-25.72° (6.6°)	19.70° (8.4°)	15.75° (7.5°)	-16.77° (15.4°)	-19.87° (17.7°)
		60°	-41.54° (6.8°)	-45.48° (7.5°)	28.76° (9.4°)	23.78° (8.7°)	-30.96° (5.5°)	-34.89° (16.8°)
		90°	-62.99° (6.0°)	-65.44° (7.8°)	28.04° (9.3°)	22.98° (9.7°)	-44.98° (16.2°)	-46.69° (16.9°)
		120°	-85.00° (5.9°)	-85.30° (8.2°)	19.62° (9.5°)	15.68° (10.5°)	-55.41° (16.3°)	-54.31° (16.9°)
	Lowering	30°	-23.84° (7.5°)	-26.15° (7.8°)	16.51° (8.1°)	12.30° (9.2°)	-13.51° (14.9°)	-16.75° (16.4°)
		60°	-40.41° (8.0°)	-43.93° (9.2°)	27.29° (9.2°)	22.45° (8.2°)	-26.80° (15.7°)	-29.19° (17.7°)
		90°	-61.39° (6.7°)	-63.64° (9.6°)	29.04° (10.0°)	23.99° (9.5°)	-43.19° (17.7°)	-42.00° (17.2°)
		120°	-85.11° (7.3°)	-84.76° (9.1°)	21.28° (10.3°)	17.57° (10.8°)	-56.24° (17.0°)	-53.27° (16.6°)
After repetitive Motion	Elevation	30°	-23.41° (6.3°)	-24.40° (7.9°)	20.57° (7.9°)	15.70° (8.6°)	-16.48° (14.9°)	-19.00° (16.9°)
		60°	-41.67° (6.5°)	-43.91° (9.4°)	28.79° (9.4°)	23.19° (10.0°)	-29.62° (15.1°)	-33.51° (16.2°)
		90°	-62.42° (5.8°)	-63.64° (10.0°)	27.25° (9.7°)	21.80° (10.6°)	-42.30° (15.7°)	-45.10° (17.3°)
		120°	-83.89° (6.0°)	-83.82° (10.8°)	18.66° (9.9°)	13.68° (11.5°)	-53.02° (15.8°)	-52.29° (18.3°)
	Lowering	30°	-23.47° (8.8°)	-25.42° (8.1°)	16.42° (8.5°)	12.42° (9.4°)	-11.75° (15.2°)	-15.44° (16.4°)
		60°	-39.88° (9.1°)	-43.01° (9.9°)	27.40° (9.7°)	22.25° (10.0°)	-25.14° (16.3°)	-28.34° (16.8°)
		90°	-60.70° (7.4°)	-63.24° (11.0°)	29.29° (10.4°)	22.91° (10.6°)	-41.67° (18.2°)	-41.37° (17.6°)
		120°	-84.05° (7.2°)	-84.74° (11.3°)	21.36° (10.8°)	15.90° (11.3°)	-54.70° (17.8°)	-51.59° (17.6°)

Table 4.4.6 Mean and standard deviation for muscle activities during elevation and lowering phases

Condition	**Phase**	**Motion Increment**	**Upper Trapezius (%)**		**Lower Trapezius (%)**		**Serratus Anterior (%)**	
			Healthy (n=24)	Impingement (n=24)	Healthy (n=25)	Impingement (n=24)	Healthy (n=25)	Impingement (n=24)
Unloaded	Elevation	30°-60°	15.34 (8.7)	19.97 (8.3)	22.04 (16.3)	24.72 (16.0)	14.66 (10.0)	17.73 (10.4)
		60°-90°	18.8 (10.2)	22.70 (9.5)	29.38 (29.0)	29.04 (18.0)	21.81 (12.0)	26.09 (14.2)
		90°-120°	19.16 (10.2)	21.36 (9.5)	38.07 (33.9)	34.21 (19.4)	32.73 (13.7)	37.49 (21.9)
	Lowering	120°-90°	10.16 (6.0)	11.96 (6.3)	18.05 (12.4)	19.48 (13.5)	16.64 (8.2)	16.77 (9.1)
		90°-60°	7.25 (4.2)	9.80 (6.1)	12.43 (8.0)	13.67 (10.6)	10.85 (6.7)	10.34 (6.3)
		60°-30°	4.07 (3.1)	6.13 (4.6)	5.51 (4.5)	6.94 (6.4)	6.18 (4.4)	5.67 (3.4)
Loaded	Elevation	30°-60°	38.03 (17.2)	45.60 (24.1)	57.90 (33.9)	49.85 (25.1)	35.04 (17.1)	38.95 (16.4)
		60°-90°	45.80 (14.8)	53.55 (24.3)	71.05 (63.3)	53.47 (23.0)	50.22 (18.7)	57.83 (22.1)
		90°-120°	40.06 (15.3)	46.46 (23.0)	59.82 (45.1)	50.50 (21.4)	56.97 (20.9)	67.53 (30.6)
	Lowering	120°-90°	22.91 (9.4)	26.51 (9.7)	39.18 (20.0)	38.96 (21.7)	40.09 (17.5)	38.87 (19.5)
		90°-60°	22.64 (8.6)	25.45 (10.0)	34.42 (17.2)	34.51 (22.1)	31.17 (16.2)	29.13 (14.6)
		60°-30°	15.84 (7.6)	17.44 (7.7)	20.56 (12.1)	19.45 (14.0)	16.70 (9.3)	14.39 (8.6)
After Repetitive Motion	Elevation	30°-60°	45.46 (17.5)	52.80 (26.9)	65.33 (53.7)	50.52 (23.9)	38.49 (20.9)	41.65 (15.5)
		60°-90°	52.00 (16.9)	59.35 (26.0)	74.02 (66.2)	55.46 (24.6)	51.96 (16.4)	59.72 (20.4)
		90°-120°	43.10 (15.8)	48.78 (22.7)	65.41 (55.3)	50.96 (20.7)	60.36 (19.8)	66.75 (26.8)
	Lowering	120°-90°	23.68 (10.0)	26.94 (10.2)	37.67 (19.7)	37.18 (20.6)	40.43 (19.6)	36.51 (17.7)
		90°-60°	22.23 (9.0)	25.26 (10.2)	31.80 (16.3)	32.28 (19.7)	30.72 (16.1)	25.03 (10.1)
		60°-30°	15.00 (6.8)	17.27 (8.1)	19.45 (13.0)	17.67 (12.0)	15.40 (8.1)	12.34 (6.3)

Table 4.5 Number of trials lost for each muscle across conditions

Condition	Upper Trapezius		Lower Trapezius		Serratus Anterior	
	Healthy (n=120)	Imp (n=115)	Healthy (n=120)	Imp (n=115)	Healthy (n=115)	Imp (n=120)
Unloaded	7	6	10	5	16	17
Loaded	10	12	12	11	13	17
After repetitive motions	5	6	14	8	14	13

Where, Imp = Subjects with impingement;
n = total number of available trials for the condition

Table 4.6.1 Mixed model ANOVA results: Absolute muscle latency

Dependent Variable	Factor	DF	F-ratio	P value
Upper Trapezius (Absolute latency)	Group	1/47	0.42	0.518
	Condition	2/94	4.04	0.021
	Group x Condition	2/94	1.51	0.226
Lower Trapezius (Absolute latency)	Group	1/47	0.65	0.426
	Condition	2/94	19.47	<0.001
	Group x Condition	2/94	3.11	0.049
Serratus Anterior (Absolute latency)	Group	1/47	2.4	0.128
	Condition	2/94	7.61	<0.001
	Group x Condition	2/94	0.31	0.736
Anterior Deltoid (Absolute Latency)	Group	1/47	1.87	0.178
	Condition	2/94	2.11	0.126
	Group x Condition	2/94	0.23	0.798

Table 4.6.2 Tukey-Kramer multiple pair-wise comparison results for 2 way mixed model ANOVA results: Absolute Latency

Dependent Variable	Factors	Levels	Geometric Mean (in msec.)	Significantly different from
Upper Trapezius	Condition	1	282.87	3
		2	308.27	
		3	321.59	1
Lower Trapezius	Condition	1	332.24	2,3
		2	253.33	1
		3	271.71	1
	Group x Condition	H, 1	339.22	(H,2), (H,3)
		H, 2	242.10	H, 1
		H, 3	248.06	H, 1
		Imp, 1	325.40	Imp, 2
		Imp, 2	265.08	Imp, 1
		Imp, 3	297.62	-
Serratus Anterior	Condition	1	288.44	2,3
		2	248.38	1
		3	257.53	1

Where, H = Healthy subjects
Imp = Subjects with impingement
1 = Unloaded condition
2 = Loaded condition
3 = After repetitive motion condition

Table 4.7.1 Mixed model ANOVA result: 3 way ANOVA for relative muscle latency

Dependent Variable	Factor	DF	F-ratio	P value
Relative Muscle latency	Group	1/41	0.13	0.723
	Muscle	2/82	7.24	0.001
	Condition	2/82	0.84	0.435
	Group x Condition	2/82	3.23	0.044
	Group x Muscle	2/82	1.79	0.173
	Condition x Muscle	4/164	14.57	<0.001
	Group x Condition x Muscle	4/164	1.45	0.221

Table 4.7.2 Tukey-Kramer multiple pair-wise comparison results for 3 way mixed model ANOVA results: Relative Latency of muscles

Dependent Variable	Factors	Levels	Mean (in msec.) †	Significantly different from
Relative Muscle latency	Muscle x Condition	UT, 1	17.65	(UT,2), (UT, 3), (LT, 1)
		UT, 2	60.32	(UT, 1), (LT, 2), (SA, 2)
		UT, 3	54.79	(UT, 1), (LT, 3), (SA, 3)
		LT, 1	64.56	(LT, 2), (LT, 3), (UT, 1)
		LT, 2	8.80	(LT, 1), (UT, 2)
		LT, 3	22.22	(LT, 1), (UT, 3)
		SA, 1	18.17	LT, 1
		SA, 2	2.41	UT, 2
		SA, 3	-3.68	UT, 3
	Group x Condition	H, 1	47.33	
		H, 2	20.69	
		H, 3	18.99	
		Imp, 1	19.59	
		Imp, 2	26.99	
		Imp, 3	29.89	

(**†**Negative sign signifies contraction before anterior deltoid)

Where, H = Healthy subjects
Imp = Subjects with impingement
UT = Upper trapezius
LT = Lower trapezius
SA = Serratus anterior
1 = Unloaded condition
2 = Loaded condition
3 = After repetitive motion condition

Table 4.8.1 Mixed model ANOVA results: Relative muscle latency

Dependent Variable	Factor	DF	F-ratio	P value
Upper Trapezius (Relative latency)	Group	1/45	3.28	0.077
	Condition	2/90	9.63	<0.001
	Group x Condition	2/90	1.22	0.301
Lower Trapezius (Relative latency)	Group	1/45	0.13	0.720
	Condition	2/90	10.39	<0.001
	Group x Condition	2/90	2.55	0.083
Serratus Anterior (Relative latency)	Group	1/45	1.35	0.251
	Condition	2/90	3.04	0.053
	Group x Condition	2/90	0.04	0.960

Table 4.8.2 Tukey-Kramer multiple pair-wise comparison results for individual 2 way mixed model ANOVA results: Relative Latency of muscles

Dependent Variable	Factors	Levels	Mean (in msec.)	Significantly different from
Upper Trapezius	Condition	1	19.23	2,3
		2	71.74	1
		3	64.81	1
Lower Trapezius	Condition	1	65.07	2,3
		2	6.78	1
		3	17.35	1

Where, 1 = Unloaded condition
2 = Loaded condition
3 = After repetitive motion condition

Table 4.9.1 Mixed model ANOVA results: Humeral angle corresponding to muscle deactivation time

Dependent Variable	Factor	DF	F-ratio	P value
Upper Trapezius (Deactivation)	Group Condition Group x Condition	1/47 2/94 2/94	0.1 20.4 0.59	0.749 <0.001 0.559
Lower Trapezius (Deactivation)	Group Condition Group x Condition	1/47 2/94 2/94	0.56 55.73 0.66	0.459 <0.001 0.519
Serratus Anterior (Deactivation)	Group Condition Group x Condition	1/47 2/94 2/94	6.69 26.0 2.25	0.013 <0.001 0.111
Anterior Deltoid (Deactivation)	Group Condition Group x Condition	1/47 2/94 2/94	3.88 20.48 0.92	0.054 <0.001 0.402

Table 4.9.2 Tukey-Kramer multiple pair-wise comparison results for individual 2 way mixed model ANOVA for the humeral angle corresponding to muscle deactivation time

Dependent Variable	Factors	Levels	Mean	Significantly different from
Upper Trapezius	Condition	1	38.22°	2,3
		2	23.09°	1
		3	22.71°	1
Lower Trapezius	Condition	1	46.37°	2,3
		2	25.99°	1
		3	28.60°	1
Serratus Anterior	Group	H	27.72°	Imp
		Imp	36.40°	H
	Condition	1	39.61°	2,3
		2	27.56°	1
		3	29.00°	1
Anterior Deltoid	Condition	1	28.33°	2,3
		2	20.90°	1
		3	20.62°	1

Where, H = Healthy subjects
Imp = Subjects with impingement
1 = Unloaded condition
2 = Loaded condition
3 = After repetitive motion condition

Table 4.10.1 Mixed model ANOVA results: Scapular kinematic variables during the elevation phase

Dependent Variable	Factor	DF	F-ratio	P value
Scapular Internal/External Rotation	Group	1/47	0.11	0.737
	Condition	2/94	9.26	<0.001
	Angle of elevation	3/141	15.18	<0.001
	Group x Condition	2/94	2.19	0.118
	Group x Angle of elevation	3/141	1.62	0.187
	Condition x Angle of elevation	6/282	20.02	<0.001
	Group x Condition x Angle of elevation	6/282	1.65	0.133
Scapular Upward Rotation	Group	1/47	1.31	0.257
	Condition	2/94	11.13	<0.001
	Angle of elevation	3/141	1557.92	<0.001
	Group x Condition	2/94	0.59	0.558
	Group x Angle of elevation	3/141	0.74	0.533
	Condition x Angle of elevation	6/282	21.03	<0.001
	Group x Condition x Angle of elevation	6/282	2.47	0.024
Scapular Tilt	Group	1/44	0.06	0.802
	Condition	2/88	3.39	0.038
	Angle of elevation	3/132	78.14	<0.001
	Group x Condition	2/88	0.13	0.882
	Group x Angle of elevation	3/132	0.07	0.977
	Condition x Angle of elevation	6/264	4.52	<0.001
	Group x Condition x Angle of elevation	6/264	0.47	0.827

Table 4.10.2 Tukey-Kramer multiple pair-wise comparison results for scapular kinematics during the elevation phase

Dependent Variable	Factors	Levels	Mean (in degrees)†	Significantly different from
Scapular Internal/External Rotation	Condition x Angle of elevation	30°, 1	32.56	(60,1),(90,1),(120,1),(30,2),(30,3)
		60°, 1	35.47	(30,1),(60,1),(60,2),(60,3)
		90°, 1	37.21	(30,1),(60,1),(120,1),(90,2),(90,3)
		120°, 1	34.99	(30,1),(90,1)
		30°, 2	35.75	(60,2),(90,2),(30,1)
		60°, 2	38.28	(30,2),(120,2),(60,1)
		90°, 2	38.71	(30,2),(120,2),(90,1)
		120°, 2	35.07	(60,2),(90,2)
		30°, 3	36.56	(60,3),(90,3),(120,3),(30,1)
		60°, 3	38.91	(30,3),(120,3),(60,1)
		90°, 3	39.12	(30,3),(120,3),(90,1)
		120°, 3	35.31	(30,3),(60,3),(90,3)
Scapular Tilt	Condition x Angle of elevation	30°, 1	-9.01	(60,1),(90,1),(120,1)
		60°, 1	-5.67	(30,1),(90,1),(120,1)
		90°, 1	-2.63	(30,1),(60,1),(120,1)
		120°, 1	2.88	(30,1),(60,1),(90,1)
		30°, 2	-8.89	(60,2),(90,2),(120,2)
		60°, 2	-4.84	(30,2),(90,2),(120,2)
		90°, 2	-1.64	(30,2),(60,2),(120,2),(90,3)
		120°, 2	2.99	(30,2),(60,2),(90,2),(120,3)
		30°, 3	-9.38	(60,3),(90,3),(120,3)
		60°, 3	-6.13	(30,3),(90,3),(120,3),
		90°, 3	-3.83	(30,3),(60,3),(120,3),(90,2)
		120°, 3	0.50	(30,3),(60,3),(90,3),(120,2)

(† Positive number for Scapular Internal/External Rotation signify internal rotation; positive number for scapular tilt signify posterior tilt)

Table 4.10.2 continued: Pair-wise comparison results using contrasts for scapular kinematics during the elevation phase.

Dependent Variable	Factors	Levels	Mean (in degrees) †	Significantly different from
Scapular Upward Rotation (Healthy subjects)	Condition x Angle of elevation	30, 1	-5.84	(60,1),(90,1),(120,1)
		60, 1	-14.68	(30,1),(90,1),(120,1),(60,2),(60,3)
		90, 1	-25.72	(30,1),(60,1),(120,1),(90,2),(90,3)
		120, 1	-35.28	(30,1),(60,1),(90,1),(120,3)
		30, 2	-5.38	(60,2),(90,2),(120,2)
		60, 2	-16.62	(30,2),(90,2),(120,2),(60,1)
		90, 2	-28.22	(30,2),(60,2),(120,2),(90,1)
		120, 2	-36.65	(30,2),(60,2),(90,2)
		30, 3	-5.14	(60,3),(90,3),(120,3)
		60, 3	-17.29	(30,3),(90,3),(120,3) ,(60,1)
		90, 3	-29.67	(30,3),(60,3),(120,3) ,(90,1)
		120, 3	-38.04	(30,3),(60,3),(90,3),(120,1)
Scapular Upward Rotation (Impingement subjects)	Condition x Angle of elevation	30, 1	-4.24	(60,1),(90,1),(120,1)
		60, 1	-13.04	(30,1),(90,1),(120,1),(60,3)
		90, 1	-22.82	(30,1),(60,1),(120,1),(90,2),(90,3)
		120, 1	-31.93	(30,1),(60,1),(90,1),(120,2),(120,3)
		30, 2	-3.68	(60,2),(90,2),(120,2)
		60, 2	-13.95	(30,2),(90,2),(120,2),(60,3)
		90, 2	-25.72	(30,2),(60,2),(120,2),(90,1),(90,3)
		120, 2	-35.24	(30,2),(60,2),(90,2),(120,1)
		30, 3	-5.23	(60,3),(90,3),(120,3)
		60, 3	-15.97	(30,3),(90,3),(120,3) ,(60,1),(60,2)
		90, 3	-27.73	(30,3),(60,3),(120,3) ,(90,1),(90,3)
		120, 3	-36.60	(30,3),(60,3),(90,3),(120,1)

(†Negative number for Scapular Upward Rotation signifies upward rotation)
Where, 1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition
30, 60, 90 and 120 = Angle of humerothoracic elevation

Table 4.11.1: Mixed model ANOVA results: Scapular kinematic variables during the lowering phase

Dependent Variable	Factor	DF	F-ratio	P value
Scapular Internal/External Rotation	Group	1/47	0.04	0.838
	Condition	2/94	11.24	<0.001
	Angle of elevation	3/141	17.10	<0.001
	Group x Condition	2/94	1.12	0.329
	Group x Angle of elevation	3/141	1	0.395
	Condition x Angle of elevation	6/282	1.56	0.159
	Group x Condition x Angle of elevation	6/282	0.53	0.788
Scapular Upward Rotation	Group	1/47	2.43	0.125
	Condition	2/94	1.42	0.247
	Angle of elevation	3/141	1369.61	<0.001
	Group x Condition	2/94	1.83	0.166
	Group x Angle of elevation	3/141	0.62	0.605
	Condition x Angle of elevation	6/282	12.01	<0.001
	Group x Condition x Angle of elevation	6/282	1.05	0.394
Scapular Tilt	Group	1/44	0.25	0.619
	Condition	2/88	3.95	0.023
	Angle of elevation	3/132	112.36	<0.001
	Group x Condition	2/88	0.14	0.871
	Group x Angle of elevation	3/132	0.17	0.919
	Condition x Angle of elevation	6/264	4.14	<0.001
	Group x Condition x Angle of elevation	6/264	0.75	0.610

Table 4.11.2 Tukey-Kramer multiple pair-wise comparison results for scapular kinematics during lowering phase

Dependent Variable	Factors	Levels	Mean (in degrees) †	Significantly different from
Scapular Internal/External Rotation	Condition	1	34.29	2,3
		2	36.79	1
		3	37.11	1
	Angle of elevation	30	35.96	30,60,90
		60	37.84	60,120
		90	37.17	120
		120	33.29	30,120
Scapular Upward Rotation	Condition x Angle of elevation	30, 1	-7.08	(60,1),(90,1),(120,1),(30,2),(30,3)
		60, 1	-17.60	(30,1),(90,1),(120,1)
		90, 1	-28.27	(30,1),(60,1),(120,1)
		120, 1	-35.59	(30,1),(60,1),(90,1),(120,2),(120,3)
		30, 2	-4.03	(60,2),(90,2),(120,2),(30,1)
		60, 2	-16.52	(30,2),(90,2),(120,2),(60,3)
		90, 2	-28.09	(30,2),(60,2),(120,2),(90,3)
		120, 2	-36.04	(30,2),(60,2),(90,2)
		30, 3	-4.85	(60,3),(90,3),(120,3),(30,1)
		60, 3	-17.77	(30,3),(90,3),(120,3),(60,1)
		90, 3	-29.44	(30,3),(60,3),(120,3),(90,1),(90,2)
		120, 3	-37.01	(30,3),(60,3),(90,3),(120,1)
Scapular Tilt	Condition x Angle of elevation	30, 1	-7.96	(60,1),(90,1),(120,1),(30,3)
		60, 1	-4.31	(30,1),(90,1),(120,1),(60,3)
		90, 1	-0.26	(30,1),(60,1),(120,1),(90,3)
		120, 1	4.96	(30,1),(60,1),(90,1),(120,3)
		30, 2	-9.39	(60,2),(90,2),(120,2)
		60, 2	-4.33	(30,2),(90,2),(120,2),(60,2),(60,3)
		90, 2	0.13	(30,2),(60,2),(120,2),(90,3)
		120, 2	4.68	(30,2),(60,2),(90,2),(120,3)
		30, 3	-10.11	(60,3),(90,3),(120,3),(30,1)
		60, 3	-5.48	(30,3),(90,3),(120,3),(60,1)
		90, 3	-1.72	(30,3),(60,3),(120,3),(90,1)
		120, 3	2.57	(30,3),(60,3),(90,3),(120,1)

(† Positive number for scapular internal/external rotation signify internal rotation; negative number for **s**capular upward rotation signify upward rotation; negative number for scapular tilt signify anterior tilt)
Where, 1 = Unloaded condition; 2 = Loaded condition; 3 = After repetitive motion condition and 30, 60, 90 and 120 = Angles of Humerothoracic elevation

Table 4.12.1 Mixed model ANOVA results: Scapulohumeral rhythm (slope of regression line) for elevation phase

Dependent Variable	Factor	DF	F-ratio	P value
Slope for scapulohumeral rhythm for 30 degree increments	Group	1/45	0.22	0.640
	Condition	2/90	48.62	<0.001
	Motion increment	2/90	1.98	0.143
	Group x Condition	2/90	0.19	0.830
	Group x Motion increment	2/90	0.20	0.820
	Condition x Motion increment	4/180	11.75	<0.001
	Group x Condition x Motion increment	4/180	2.67	0.34
Slope for scapulohumeral rhythm from 30 to 120 degree increment	Group	1/47	0.07	0.797
	Condition	2/94	41.68	<0.001
	Group x Condition	2/94	1.16	0.318

Table 4.12.2a Tukey Kramer pair wise comparisons results: Scapulohumeral rhythm (slope of regression line) for elevation phase

Dependent Variable	Factors	Levels	Mean	Significantly different from
Healthy (n=24)	Condition x Motion increment	30°-60°, 1	2.63	(30°-60°,2),(30°-60°,3)
		60°-90°, 1	2.47	(60°-90°,2) (60°-90°,3)
		90°-120°, 1	2.51	
		30°-60°, 2	1.85	(30°-60°,1),(90°-120°,2)
		60°-90°, 2	1.98	(60°-90°,1)
		90°-120°, 2	2.64	(30°-60°,2), (90°-120°,1)
		30°-60°, 3	1.66	(90°-120°,3),(30°-60°,1)
		60°-90°, 3	1.77	(90°-120°,3),(60°-90°,1)
		90°-120°, 3	2.67	(30°-60°,3),(60°-90°,3)
Impingement (n=23)	Condition x Motion increment	30°-60°, 1	1.75	(30°-60°,2),(30°-60°,3)
		60°-90°, 1	2.57	(60°-90°,2) (60°-90°,3)
		90°-120°, 1	2.60	
		30°-60°, 2	2.60	(30°-60°,1)
		60°-90°, 2	2.84	(60°-90°,1)
		90°-120°, 2	2.11	(30°-60°,2)
		30°-60°, 3	1.98	(30°-60°,1)
		60°-90°, 3	2.64	(60°-90°,1)
		90°-120°, 3	1.92	

Where, 1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition

Table 4.12.2b Tukey Kramer pair wise comparisons results: Scapulohumeral rhythm (slope of regression line) for elevation phase by 2 way ANOVA

Dependent Variable	Factors	Levels	Mean	Significantly different from
	Condition	1	2.40	2,3
		2	1.89	1
		3	1.81	1

Where, 1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition
30°-60°,60°-90°,90°-120° are motion increments.

Table 4.13.1 Mixed model ANOVA results: Muscle activity during the elevation phase

Dependent Variable	**Factor**	**DF**	**F-ratio**	**P value**
Upper Trapezius	Group	1/46	1.73	0.195
	Condition	2/92	318.63	<0.001
	Motion increment	2/92	12.29	<0.001
	Group x Condition	2/92	1.81	0.169
	Group x Motion Increment	2/92	0.29	0.747
	Condition x Motion Increment	4/184	8.19	<0.001
	Group x Condition x Motion Increment	4/184	1.51	0.201
Lower Trapezius	Group	1/47	0.33	0.568
	Condition	2/94	167.16	<0.001
	Motion increment	2/94	13.62	<0.001
	Group x Condition	2/94	2.5	0.088
	Group x Motion Increment	2/94	0.25	0.778
	Condition x Motion Increment	4/188	72.02	<0.001
	Group x Condition x Motion Increment	4/188	4.86	<0.001
Serratus Anterior	Group	1/47	1.77	0.190
	Condition	2/94	431.12	<0.001
	Motion increment	2/94	141.13	<0.001
	Group x Condition	2/94	0.69	0.512
	Group x Motion Increment	2/94	0.14	0.866
	Condition x Motion Increment	4/188	30.13	<0.001
	Group x Condition x Motion Increment	4/188	0.85	0.495

Table 4.13.2 Tukey-Kramer multiple pair-wise comparison results for muscle activity during the elevation phase

Dependent Variable	Factors	Levels	Geometric Mean (in %)	Significantly different from
Upper Trapezius	Condition x Motion Increment	30-60, 1	15.56	(60-90,1),(90-120,1),(30-60,2),(30-60,3)
		60-90, 1	18.51	(30-60,1),(60-90,2),(60-90,3)
		90-120, 1	17.92	(30-60,1),(90-120,2),(90-120,3)
		30-60, 2	37.25	(60-90,2),(30-60,1), (30-60,3)
		60-90, 2	46.11	(30-60,2),(90-120,2),(60-90,1),(60-90,2)
		90-120, 2	39.69	(60-90,2),(90-120,1)
		30-60, 3	44.41	(60-90,3),(30-60,1),(30-60,2)
		60-90, 3	51.68	(30-60,3),(90-120,3),(60-90,1),(60-90,2)
		90-120, 3	42.43	(60-90,3),(90-120,1)
Serratus Anterior	Condition x Motion Increment	30-60, 1	14.13	(60-90,1),(90-120,1),(30-60,2),(30-60,3)
		60-90, 1	21.52	(30-60,1),(90-120,1),(60-90,2),(60-90,3)
		90-120, 1	31.97	(30-60,1),(60-90,1),(90-120,2),(90-120,3)
		30-60, 2	33.46	(60-90,2),(90-120,2),(30-60,1),(30-60,3)
		60-90, 2	50.36	(30-60,2),(90-120,2),(60-90,1)
		90-120, 2	57.33	(30-60,2),(60-90,2),(90-120,1)
		30-60, 3	37.02	(60-90,3),(90-120,3),(30-60,1)
		60-90, 3	52.31	(30-60,3),(90-120,3),(60-90,1)
		90-120, 3	59.66	(30-60,3),(60-90,3),(90-120,1)

Table 4.13.2 contd. Multiple pair-wise comparison results for Lower Trapezius muscle activity using contrasts during the elevation phase

Dependent Variable	Factors	Levels	Geometric Mean (in %)	Significantly different from
Healthy	Condition x Motion Increment	30-60, 1	17.88	(60-90,1),(90-120,1), (30-60,2), (30-60,3)
		60-90, 1	23.02	(30-60,1),(90-120,1),(60-90,2), (60-90,3)
		90-120, 1	30.12	(30-60,1),(60-90,1),(90-120,2), (90-120,3)
		30-60, 2	50.46	(60-90,2),(30-60,1)
		60-90, 2	58.90	(30-60,2),(90-120,2)
		90-120, 2	50.72	(60-90,2),(90-120,1)
		30-60, 3	54.31	(60-90,3),(30-60,1)
		60-90, 3	60.63	(30-60,3),(90-120,3),(60-90,1)
		90-120, 3	53.91	(60-90,3),(90-120,1)
Impingement	Condition x Motion Increment	30-60, 1	20.22	(60-90,1),(90-120,1), (30-60,2), (30-60,3)
		60-90, 1	24.07	(30-60,1),(90-120,1),(60-90,2), (60-90,3)
		90-120, 1	28.81	(30-60,1),(60-90,1),(90-120,2), (90-120,3)
		30-60, 2	44.72	(30-60,1)
		60-90, 2	49.17	(60-90,1)
		90-120, 2	46.86	(90-120,1)
		30-60, 3	45.64	(30-60,1)
		60-90, 3	50.65	(60-90,1)
		90-120, 3	47.30	(90-120,1)

Where, 1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition
30-60, 60-90, 90-120 = Motion Increments

Table 4.14.1 Mixed model ANOVA results: Muscle activity during the lowering phase

Dependent Variable	Factor	DF	F-ratio	P value
Upper Trapezius	Group	1/46	2.71	0.106
	Condition	2/92	196.33	<0.001
	Motion increment	2/92	132.84	<0.001
	Group x Condition	2/92	1.07	0.348
	Group x Motion Increment	2/92	0.25	0.782
	Condition x Motion Increment	4/184	19.77	<0.001
	Group x Condition x Motion Increment	4/184	0.91	0.460
Lower Trapezius	Group	1/47	0	0.95
	Condition	2/94	129.85	<0.001
	Motion increment	2/94	175.12	<0.001
	Group x Condition	2/94	0.84	0.436
	Group x Motion Increment	2/94	0.17	0.844
	Condition x Motion Increment	4/188	13.34	<0.001
	Group x Condition x Motion Increment	4/188	2.17	0.074
Serratus Anterior	Group	1/47	0.55	0.464
	Condition	2/94	596.35	<0.001
	Motion increment	2/94	391.01	<0.001
	Group x Condition	2/94	1.18	0.313
	Group x Motion Increment	2/94	1.29	0.279
	Condition x Motion Increment	4/188	9.18	<0.001
	Group x Condition x Motion Increment	4/188	0.30	0.878

Table 4.14.2 Tukey-Kramer multiple pair-wise comparison results for muscle activity during the lowering phase

Dependent Variable	Factors	Levels	Mean (in %)	Significantly different from
Upper Trapezius	Condition x Motion Increment	60-30, 1	3.74	(90-60,1),(120-90,1),(60-30,2), (60-30,3)
		90-60, 1	7.01	(60-30,1),(120-90,1),90-60,2), (90-60,3)
		120-90, 1	9.30	(60-30,1),(90-60,1),(120-90,2), (120-90,3)
		60-30, 2	15.19	(90-60,2),(120-90,2),(60-30,1)
		90-60, 2	22.47	(60-30,2),(90-60,1)
		120-90, 2	23.04	(60-30,2),(120-90,1)
		60-30, 3	14.67	(90-60,3),(120-90,3),(60-30,1)
		90-60, 3	22.04	(60-30,3),(90-60,1)
		120-90, 3	23.42	(60-30,3),(120-90,1)
Lower Trapezius	Condition x Motion Increment	60-30, 1	4.61	(90-60,1),(120-90,1),(60-30,2), (60-30,3)
		90-60, 1	9.45	(60-30,1),(120-90,1),(90-60,2), (90-60,3)
		120-90, 1	14.49	(60-30,1),(90-60,1),(120-90,2), (120-90,3)
		60-30, 2	15.88	(90-60,2),(120-90,2),(60-30,1)
		90-60, 2	29.52	(60-30,2),(120-90,2),(90-60,1)
		120-90, 2	34.28	(60-30,2),(90-60,2),(120-90,1)
		60-30, 3	14.70	(90-60,3),(120-90,3),(60-30,1)
		90-60, 3	27.70	(60-30,3),(120-90,3),(90-60,1)
		120-90, 3	33.05	(60-30,3),(90-60,3),(120-90,1)

Table 4.14.2 continued: Tukey-Kramer multiple pair-wise comparison results for muscle activity during the lowering phase

Dependent Variable	Factors	Levels	Mean (in %)	Significantly different from
Serratus Anterior	Condition x Motion Increment	60-30, 1	5.03	(90-60,1),(120-90,1),(60-30,2),(60-30,3)
		90-60, 1	9.31	(60-30,1),(120-90,1),(90-60,2),(90-60,3)
		120-90, 1	15.15	(60-30,1),(90-60,1),(120-90,2),(120-90,3)
		60-30, 2	13.29	(90-60,2),(120-90,2),(60-30,1),(60-30,3)
		90-60, 2	26.83	(60-30,2),(120-90,2),(90-60,1)
		120-90, 2	35.89	(60-30,2),(90-60,2),(120-90,1)
		60-30, 3	12.06	(90-60,3),(120-90,3),(60-30,1),(60-30,2)
		90-60, 3	25.07	(60-30,3),(120-90,3),(90-60,1)
		120-90, 3	34.77	(60-30,3),(90-60,3),(120-90,1)

Where, 1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition
60-30, 90-60 and 120- 90 = Motion Increment

Table 4.15 Mean and standard deviation for kinematic variables at initial rest position

Dependent variable	Healthy (n = 25)	Impingement (n = 24)	P-value
Scapular internal/external rotation	28.65° (5.5)	28.48° (6.1)	0.92
Scapular upward rotation	2.20° (6.2)	3.00° (6)	0.65
Scapular tilt	-12.64° (5.6)	-13.29° (5.1)	0.67
Glenohumeral elevation	-5.43° (6.4)	-10.07° (7.2)	0.02
Glenohumeral plane of elevation	2.51° (4.4)	2.49° (6.4)	0.99
Glenohumeral axial rotation	3.96° (11.6)	2.50° (15.7)	0.71

Table 4.16.1 Mixed model ANOVA results: Kinematics at peak humeral elevation

Dependent Variable	Factor	DF	F-ratio	P value
Scapular Internal/External Rotation	Group	1/47	3.00	0.089
	Condition	2/94	0.59	0.558
	Group x Condition	2/94	0.64	0.532
Scapular Upward Rotation	Group	1/47	0.69	0.409
	Condition	2/94	1.10	0.338
	Group x Condition	2/94	0.50	0.605
Scapular Tilt	Group	1/47	0.15	0.704
	Condition	2/94	9.88	<0.001
	Group x Condition	2/94	1.12	0.332
Glenohumeral elevation	Group	1/47	0.39	0.534
	Condition	2/94	5.45	0.006
	Group x Condition	2/94	0.58	0.560
Glenohumeral plane of elevation	Group	1/47	1.41	0.241
	Condition	2/94	9.01	<0.001
	Group x Condition	2/94	4.86	0.009
Glenohumeral axial rotation	Group	1/47	0.86	0.360
	Condition	2/94	8.28	<0.001
	Group x Condition	2/94	1.16	0.317

Table 4.16.2 Tukey-Kramer multiple pair-wise comparison results for the kinematics at peak humeral elevation

Dependent Variable	Factors	Levels	Mean (in degrees) †	Significantly different from
Scapular Tilt	Condition	1	6.92	3
		2	6.09	3
		3	3.11	1,2
Glenohumeral elevation	Condition	1	-100.27	2,3
		2	-97.29	1
		3	-97.02	1
Glenohumeral plane of elevation	Group x Condition	H,1	12.26	-
		H,2	12.05	Imp,2
		H,3	11.40	Imp,3
		Imp,1	11.62	Imp,3
		Imp,2	8.73	H,2
		Imp,3	6.07	(Imp,1),(H,3)
Glenohumeral axial rotation	Condition	1	-56.82	2,3
		2	-60.07	1
		3	-59.16	1

(† Negative number for glenohumeral elevation signifies elevation; positive number for glenohumeral plane of elevation signifies anterior to scapular plane; negative number for glenohumeral axial rotation signifies external rotation; positive number for scapular internal/external rotation signifies internal rotation; negative number for scapular upward rotation signifies upward rotation; negative number for scapular tilt signifies anterior tilt)

Where, H = Healthy subjects
Imp = Subjects with impingement
1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition

Table 4.17.1 Mixed model ANOVA results: Glenohumeral kinematic variables during the elevation phase

Dependent Variable	Factor	DF	F-ratio	P value
Glenohumeral Elevation Angle	Group	1/47	1.3	0.260
	Condition	2/94	7.11	<0.001
	Angle of elevation	3/141	3696.82	<0.001
	Group x Condition	2/94	0.57	0.568
	Group x Angle of elevation	3/141	1.49	0.221
	Condition x Angle of elevation	6/282	33.26	<0.001
	Group x Condition x Angle of elevation	6/282	1.16	0.328
Glenohumeral Plane of elevation	Group	1/47	6.14	0.017
	Condition	2/94	39.04	<0.001
	Angle of elevation	3/141	65.80	<0.001
	Group x Condition	2/94	0.40	0.673
	Group x Angle of elevation	3/141	0.29	0.834
	Condition x Angle of elevation	6/282	28.31	<0.001
	Group x Condition x Angle of elevation	6/282	0.7	0.654
Glenohumeral axial rotation	Group	1/47	0.12	0.735
	Condition	2/94	20.21	<0.001
	Angle of elevation	3/141	384.96	<0.001
	Group x Condition	2/94	0.76	0.471
	Group x Angle of elevation	3/141	1.56	0.202
	Condition x Angle of elevation	6/282	44.4	<0.001
	Group x Condition x Angle of elevation	6/282	1.13	0.343

Table 4.17.2 Tukey-Kramer multiple pair-wise comparison results for glenohumeral kinematics during elevation phase

Dependent Variable	Factors	Levels	Mean (in degrees) †	Significantly different from
Glenohumeral elevation	Condition x Angle of elevation	30, 1	-23.12	(60,1),(90,1),(120,1)
		60, 1	-43.92	(30,1),(90,1),(120,1)
		90, 1	-68.11	(30,1),(60,1),(120,1),(90,2),(90,3)
		120, 1	-89.07	(30,1),(60,1),(90,1),(120,2),(120,3)
		30, 2	-24.31	(60,2),(90,2),(120,2)
		60, 2	-43.51	(30,2),(90,2),(120,2)
		90, 2	-64.21	(30,2),(60,2),(120,2), (90,1)
		120, 2	-85.15	(30,2),(60,2),(90,2),(120,1)
		30, 3	-23.91	(60,3),(90,3),(120,3)
		60, 3	-42.79	(30,3),(90,3),(120,3)
		90, 3	-63.03	(30,3),(60,3),(120,3),(90,1)
		120, 3	-83.85	(30,3),(60,3),(90,3),(120,1)
Glenohumeral plane of elevation	Group	H	26.11	Imp
		Imp	20.91	H
	Condition x Angle of elevation	30, 1	20.57	(60,1),(90,1),(120,1),(30,2),(30,3)
		60, 1	32.94	(30,1),(120,1),(60,2),(60,3)
		90, 1	33.26	(30,1),(120,1),(90,2),(90,3)
		120, 1	23.38	(30,1),(60,1),(90,1),(120,2),(120,3)
		30, 2	17.72	(60,2),(90,2),(30,1)
		60, 2	26.27	(30,2),(120,2),(60,1)
		90, 2	25.51	(30,2),(120,2),(90,1)
		120, 2	17.65	(60,2),(90,2),(120,1),(120,3)
		30, 3	18.14	(60,3),(90,3),(120,3),(30,1)
		60, 3	25.99	(30,3),(90,3),(120,3),(60,1)
		90, 3	24.52	(30,3),(60,3),(120,3),(90,1)
		120, 3	16.17	(30,3),(60,3),(90,3),(120,1),(120,2)

Table 4.17.2 continued: Tukey-Kramer multiple pair-wise comparison results for glenohumeral kinematics during elevation phase

Dependent Variable	Factors	Levels	Mean (in degrees) †	Significantly different from
Glenohumeral axial Rotation	Condition x Angle of elevation	30, 1	-8.41	(60,1),(90,1),(120,1),(30,2),(30,3)
		60, 1	-26.37	(30,1),(90,1),(120,1),(60,2),(60,3)
		90, 1	-44.21	(30,1),(60,1),(120,1)
		120, 1	-53.57	(30,1),(60,1),(90,1)
		30, 2	-18.32	(60,2),(90,2),(120,2),(30,1)
		60, 2	-32.93	(30,2),(90,2),(120,2),(60,1)
		90, 2	-45.83	(30,2),(60,2)(120,2),(90,3)
		120, 2	-54.86	(30,2),(60,2),(90,2),(120,3)
		30, 3	-17.74	(60,3),(90,3),(120,3),(30,1)
		60, 3	-31.57	(30,3),(90,3),(120,3),(60,1)
		90, 3	-43.70	(30,3),(60,3),(120,3),(90,2)
		120, 3	-52.66	(30,3),(60,3),(90,3),(120,2)

(† Negative number for glenohumeral elevation signifies elevation; for axial rotation signifies external rotation; Positive number for glenohumeral plane of elevation signifies horizontal adduction)

Where, H = Healthy subjects
Imp = Subjects with impingement
1 = Unloaded condition
2 = Loaded condition
3 = after repetitive motion condition
30, 60, 90 and 120 = Angles of Humerothoracic elevation

Table 4.18.1 Mixed model ANOVA results: Glenohumeral kinematic variables during the lowering phase

Dependent Variable	Factor	DF	F-ratio	P value
Glenohumeral Elevation Angle	Group	1/47	2.14	0.151
	Condition	2/94	1.28	0.282
	Angle of elevation	3/141	2418.7	<0.001
	Group x Condition	2/94	2.24	0.117
	Group x Angle of elevation	3/141	1.66	0.179
	Condition x Angle of elevation	6/282	26.74	<0.001
	Group x Condition x Angle of elevation	6/282	0.37	0.899
Glenohumeral plane of elevation	Group	1/47	4.62	0.037
	Condition	2/94	71.73	<0.001
	Angle of elevation	3/141	78.42	<0.001
	Group x Condition	2/94	0.14	0.868
	Group x Angle of elevation	3/141	0.40	0.756
	Condition x Angle of elevation	6/282	18.30	<0.001
	Group x Condition x Angle of elevation	6/282	1.47	0.187
Glenohumeral axial rotation	Group	1/47	0.01	0.907
	Condition	2/94	31.23	<0.001
	Angle of elevation	3/141	364.0	<0.001
	Group x Condition	2/94	0.08	0.922
	Group x Angle of elevation	3/141	2.68	0.049
	Condition x Angle of elevation	6/282	24.15	<0.001
	Group x Condition x Angle of elevation	6/282	0.14	0.990

Table 4.18.2 Tukey-Kramer multiple pair-wise comparison results for glenohumeral kinematics during lowering phase

Dependent Variable	Factors	Levels	Mean (in degrees) †	Significantly different from
Glenohumeral elevation	Condition x Angle of elevation	30, 1	-20.44	(60,1),(90,1),(120,1),(30,2),(30,3)
		60, 1	-38.68	(30,1),(90,1),(120,1),(60,2),(60,3)
		90, 1	-63.06	(30,1),(60,1),(120,1)
		120, 1	-86.94	(30,1),(60,1),(90,1),(120,2),(120,3)
		30, 2	-25.00	(60,2),(90,2),(120,2),(30,1)
		60, 2	-42.17	(30,2),(90,2),(120,2),(60,1)
		90, 2	-62.52	(30,2),(60,2),(120,2)
		120, 2	-84.93	(30,2),(60,2),(90,2),(120,1)
		30, 3	-24.44	(60,3),(90,3),(120,3),(30,1)
		60, 3	-41.45	(30,3),(90,3),(120,3),(60,1)
		90, 3	-61.97	(30,3),(60,3),(120,3)
		120, 3	-84.40	(30,3),(60,3),(90,3),(120,1)
Glenohumeral plane of elevation	Group	H	26.24	Imp
		Imp	21.45	H
	Condition x Angle of elevation	30, 1	19.63	(60,1),(90,1),(120,1),(30,2),(30,3)
		60, 1	34.40	(30,1),(90,1),(120,1),(60,2),(60,3)
		90, 1	36.63	(30,1),(60,1),(120,1),(90,2),(90,3)
		120, 1	26.29	(30,1),(60,1),(90,1),(120,2),(120,3)
		30, 2	14.40	(60,2),(90,2),(120,2),(30,1)
		60, 2	24.87	(30,2),(90,2),(120,2),(60,1)
		90, 2	26.51	(30,2),(60,2),(120,2),(90,1)
		120, 2	19.43	(30,2),(60,2),(90,2),(120,1)
		30, 3	14.42	(60,3),(90,3),(120,3),(30,1)
		60, 3	24.82	(30,3),(120,3),(60,1)
		90, 3	26.10	(30,3),(120,3),(90,1)
		120, 3	18.63	(30,3),(60,3),(90,3),(120,1)

(† Negative number for glenohumeral elevation signifies elevation; positive number for glenohumeral plane of elevation signifies horizontal adduction)

Where, H = Healthy subjects
Imp = Subjects with impingement
1 = Unloaded condition
2 = Loaded condition
3 = After repetitive motion condition

4.19a Logistic regression results: Group prediction by kinematic variables at 60 degrees of humerothoracic elevation

Parameter	DF	Estimate	Wald's Chi-square	P value	Odds-ratio	95% Walds CI
Intercept	1	84.07	5.46	0.020		
Scapular IRER	1	-0.13	3.31	0.069	0.88	0.76-1.01
Scapular UR	1	1.29	5.65	0.017	3.62	1.25-10.46
Scapular tilt	1	-1.08	6.79	0.009	0.34	0.15-0.77
GH plane of elevation	1	-0.71	8.27	0.004	0.49	0.30-0.80
GH angle of elevation	1	1.20	5.25	0.022	3.32	1.18-9.25
GH axial rotation	1	0.02	0.31	0.579	1.02	0.96-1.08
Normalized Pectoralis Minor length	1	0.87	1.55	0.213	2.40	0.61-9.42

4.19 b Logistic regression results: Group prediction by kinematic variables at 90 degrees of humerothoracic elevation

Parameter	DF	Estimate	Wald's Chi-square	P value	Odds-ratio	95% Walds CI
Intercept	1	53.58	3.27	0.071		
Scapular IRER	1	-0.09	2.51	0.113	0.91	0.81-1.02
Scapular UR	1	0.65	3.62	0.057	1.92	0.98-3.77
Scapular tilt	1	-0.41	3.87	0.049	0.66	0.44-0.99
GH plane of elevation	1	-0.16	6.83	0.009	0.85	0.76-0.96
GH angle of elevation	1	0.58	3.14	0.076	1.78	0.94-3.4
GH axial rotation	1	0.00	0.02	0.886	1.00	0.94-1.07
Normalized Pectoralis Minor length	1	1.02	2.47	0.116	2.77	0.78-9.9

Where, CI = confidence intervals

4.20 Equality of variance test results

	Standard Deviation (Absolute latency)		**P value**	**Standard Deviation (Relative latency)**		**P value**
	Healthy (n=25)	**Impingement** (n=24)		**Healthy**	**Impingement**	
Upper Trapezius	73.61	103.07	0.09	61.81 (n=24)	45.53 (n=24)	0.156
Lower Trapezius	129.43	72.44	0.007*	92.82 (n=24)	39.37 (n=24)	<0.001*
Serratus Anterior	94.85	77.53	0.33	44.18 (n=23)	77.53 (n=24)	0.010*

* indicates group difference ($p<0.05$)

FIGURES

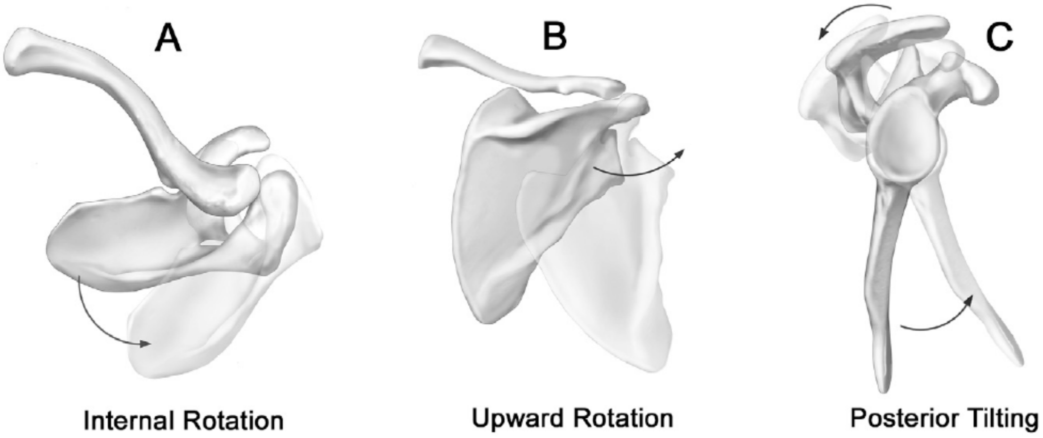


Figure 1 Scapular motion in 3 dimensions: (A) Scapular internal/external rotation; (B) Scapular upward/downward rotation and (C) Scapular anterior/posterior tilt**.**
(Ludewig et al. 2009)

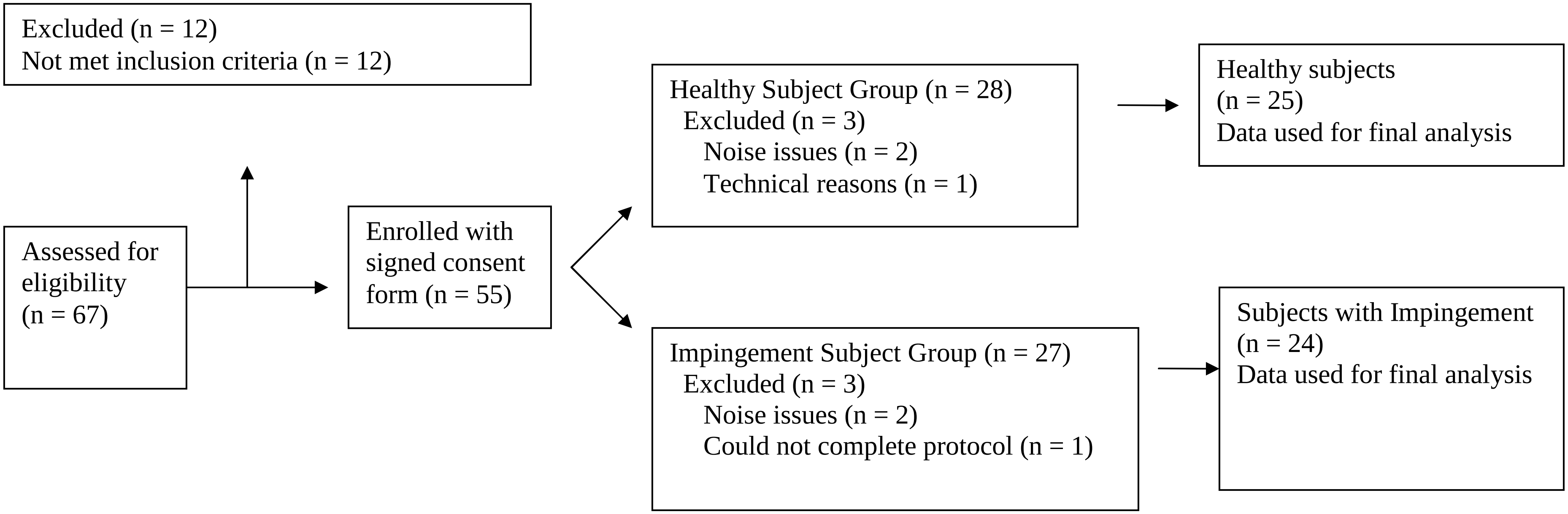
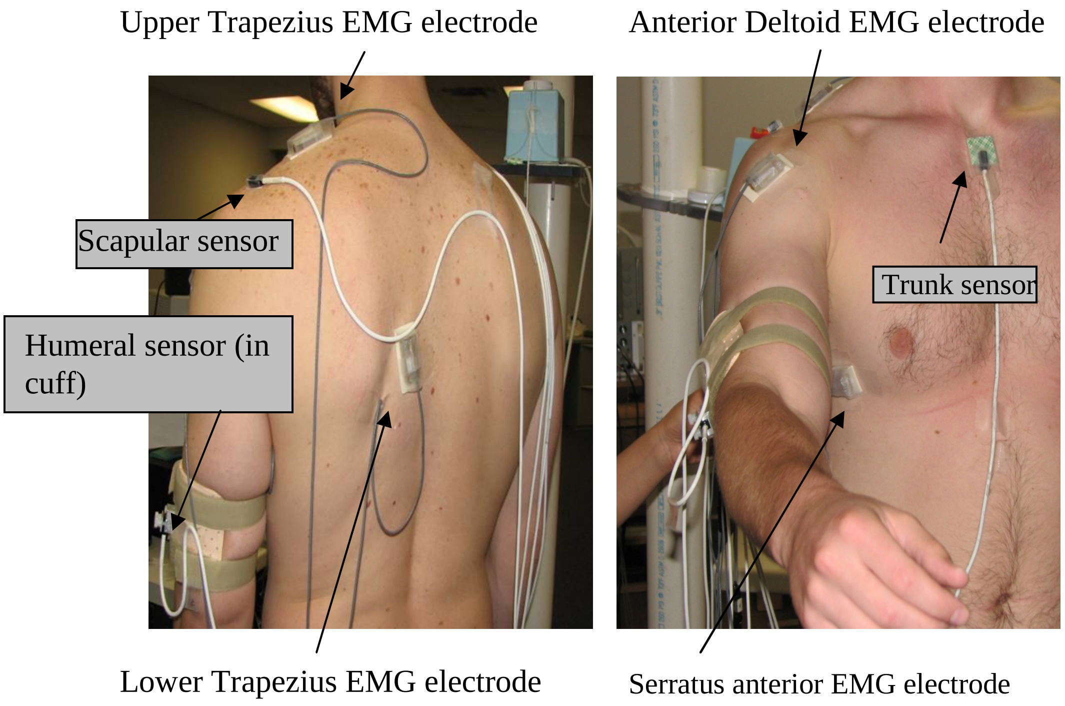


Figure 3.1 Flow chart showing number of subjects recruited and included in the study



(a) (b)

Figure 3.2 a and 3.2 b: Kinematic and EMG electrode set up for data collection**.**

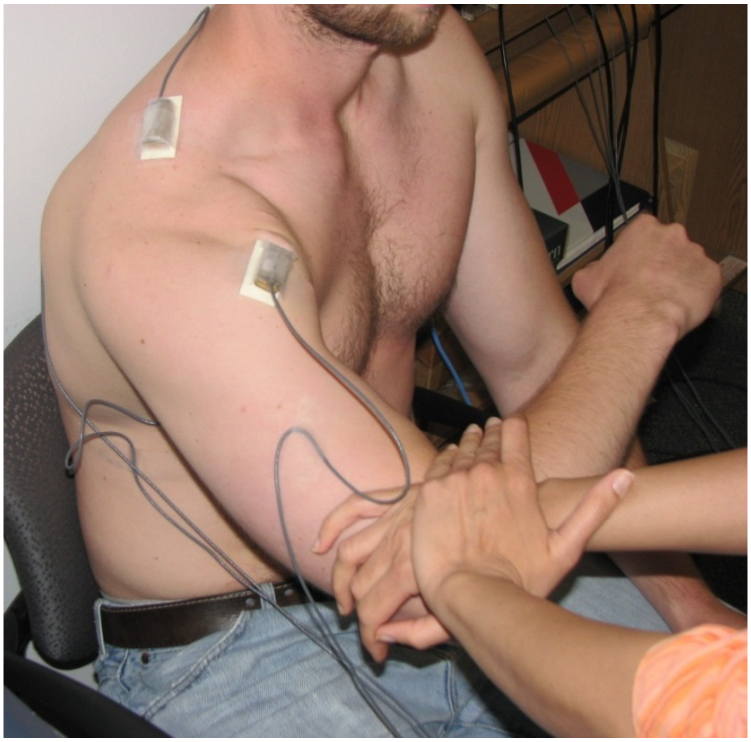


Figure 3.3 a: Position for recording maximum voluntary contraction (MVC) for upper trapezius and anterior deltoid.

Figure 3.3 b: Position for recording maximum voluntary contraction (MVC) for lower trapezius.

Figure 3.3 c: Position for recording maximum voluntary contraction (MVC) for serratus anterior.

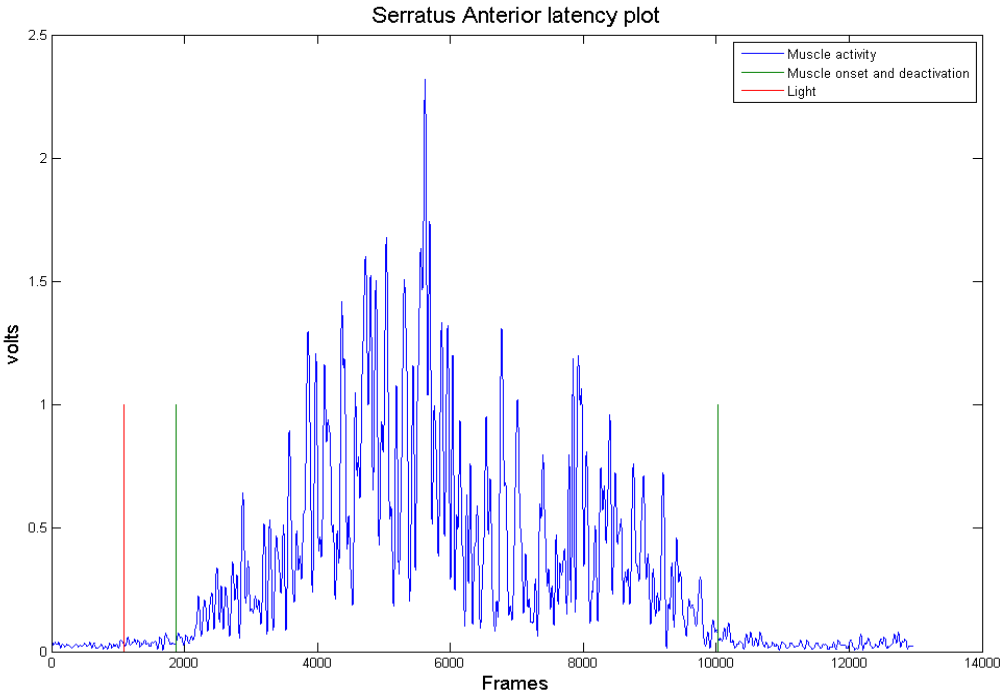


Figure 3.4 Muscle latency estimation using computer algorithm (Appendix 10) (Hodges and Bui, 1996). The blue line shows rectified and smoothed serratus anterior muscle activity. The vertical red line indicates the light cue. The first vertical green line indicates muscle activation whereas the second vertical green line indicates muscle deactivation.

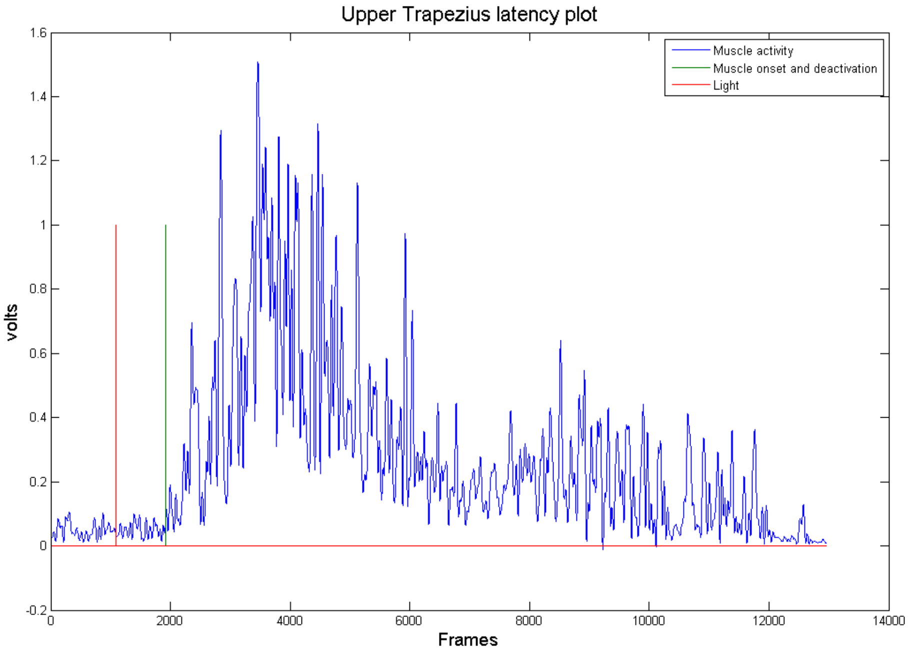


Figure 3.5 No deactivation of upper trapezius muscle detected for the trial.

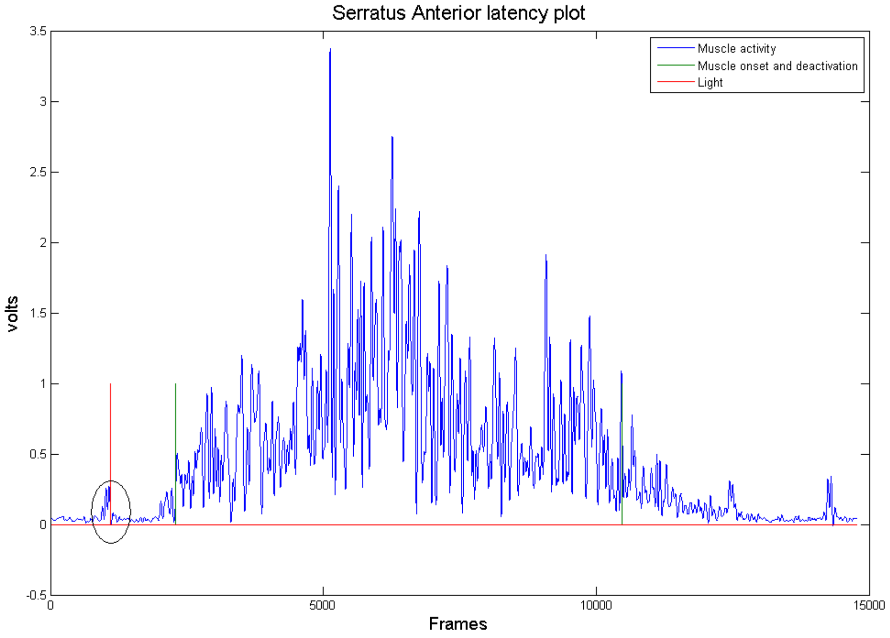


Figure 3.6a Possible cardiac artifact (in circle) affecting result interpretation of muscle onset. The blue line shows rectified and smoothed serratus anterior muscle activity for the motion. The vertical red line indicates the light cue. The first vertical green line indicates muscle activation whereas the second vertical green line indicates muscle deactivation.

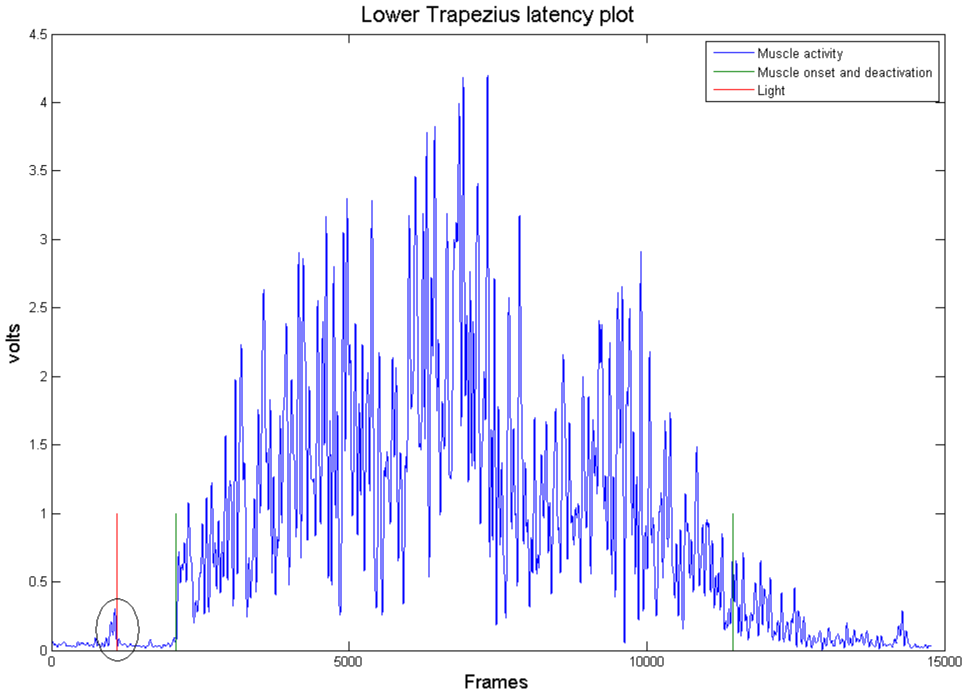


Figure 3.6b Possible cardiac artifact (in circle) affecting result interpretation of muscle deactivation. The blue line shows rectified and smoothed lower trapezius muscle activity for the motion. The vertical red line indicates the light cue. The first vertical green line indicates muscle activation whereas the second vertical green line indicates muscle deactivation.

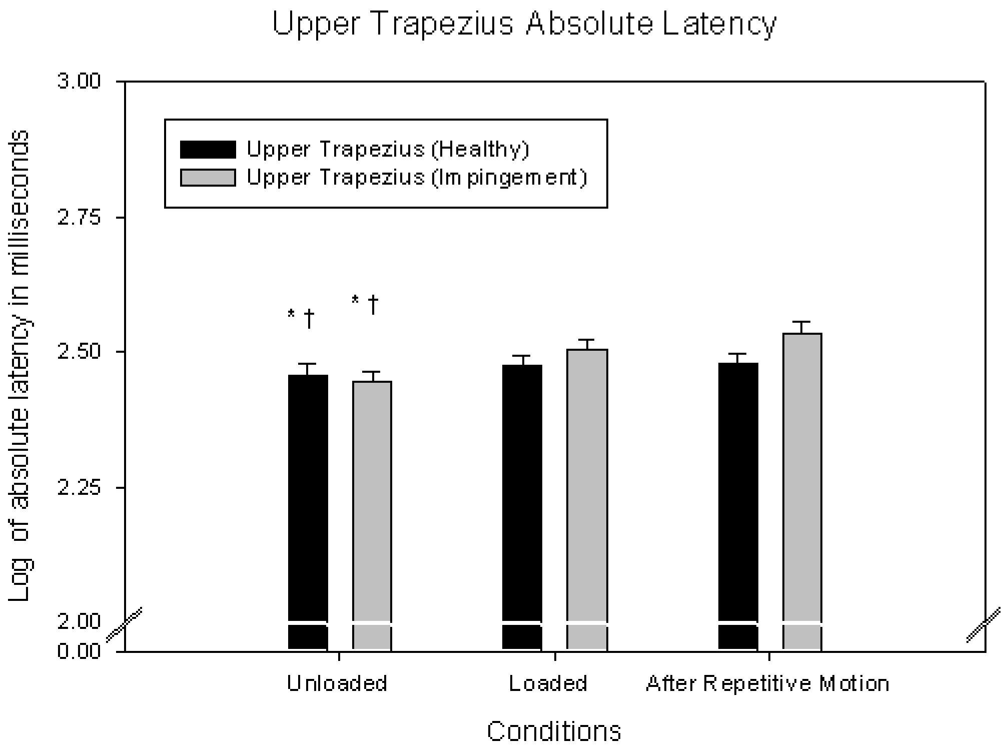


Figure 4.1a Absolute latency of upper trapezius**.**

* signifies difference between the unloaded condition and the loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

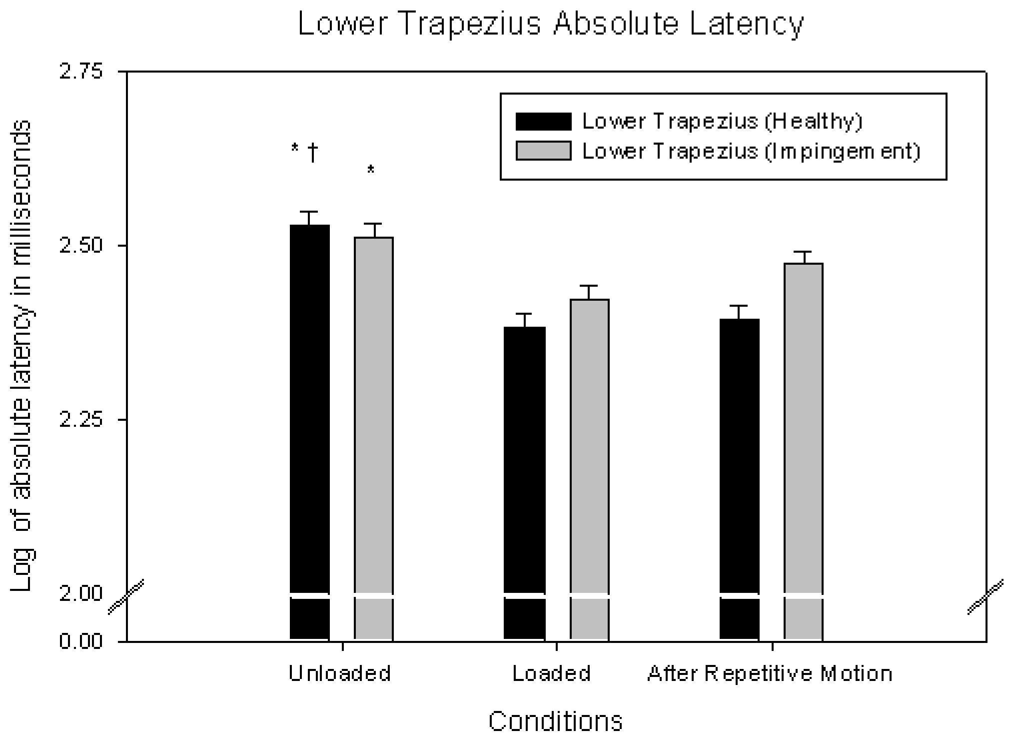


Figure 4.1b Absolute latency of lower trapezius.

* signifies difference between the unloaded condition and the loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

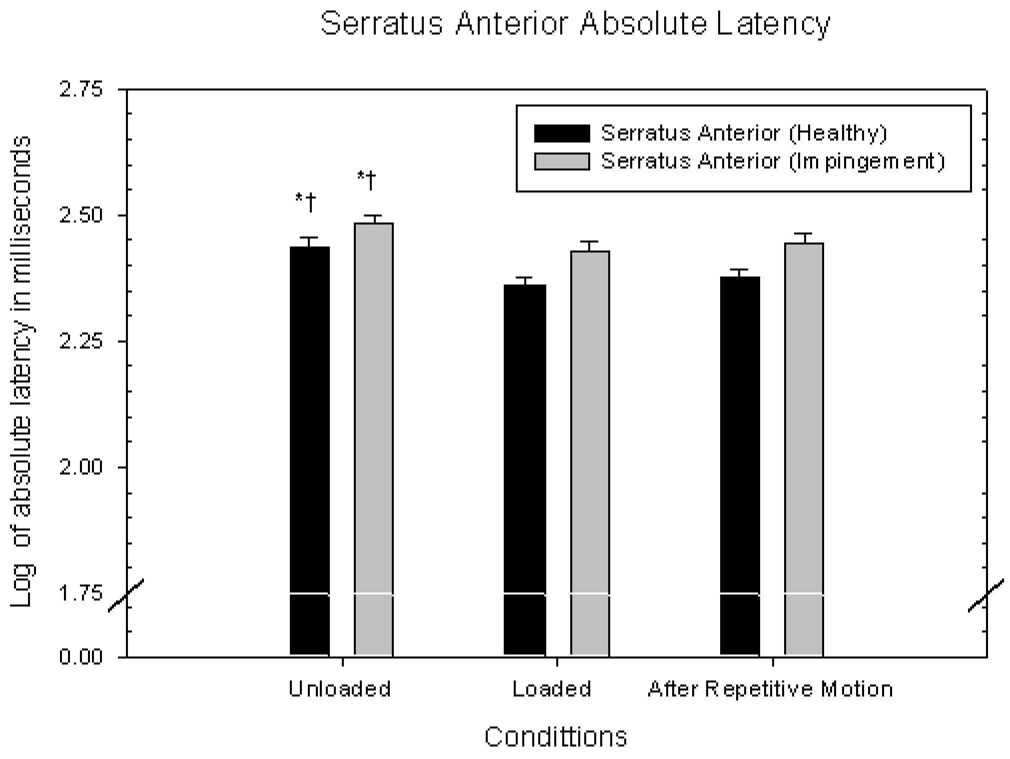


Figure 4.1c Absolute latency of serratus anterior.

* signifies difference between the unloaded condition and the loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

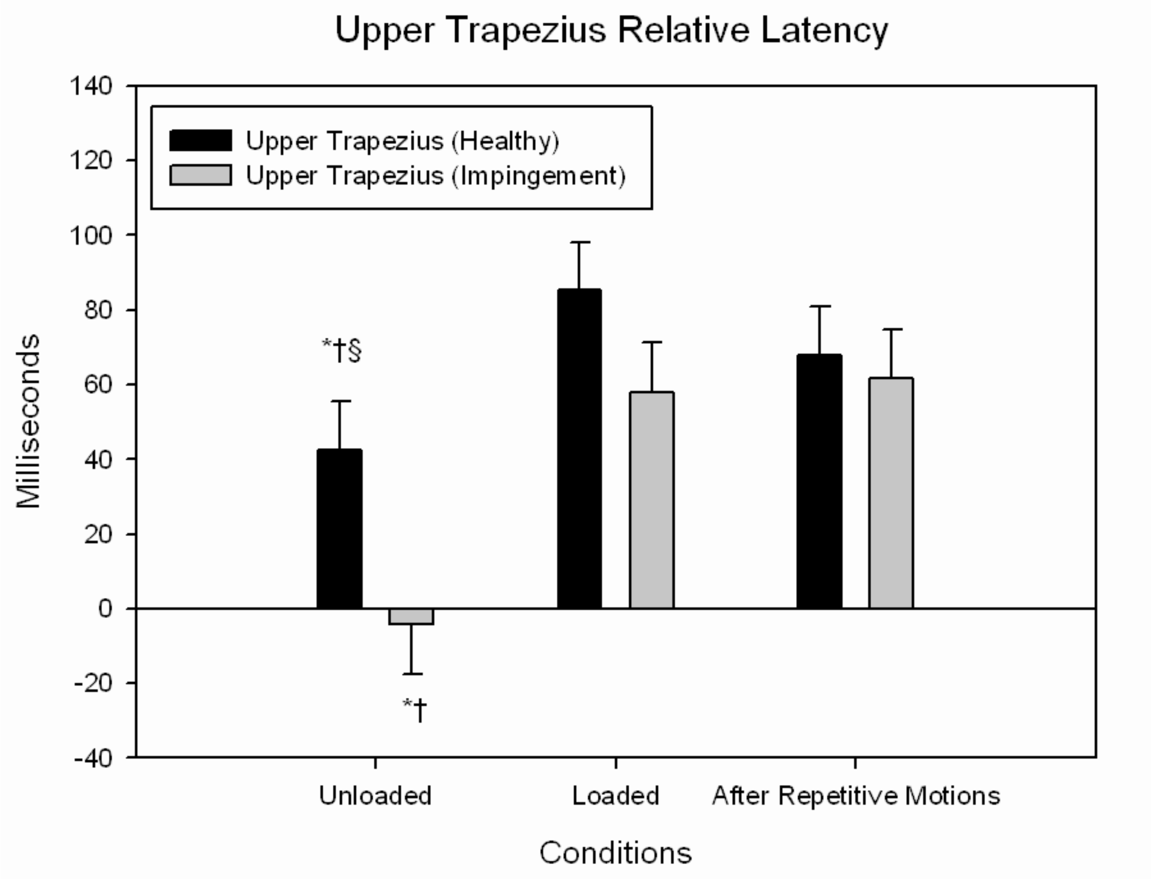


Figure 4.2a Relative latency of upper trapezius.

* signifies difference between the unloaded condition and loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)
§ signifies group difference at a condition ($p<0.05$)

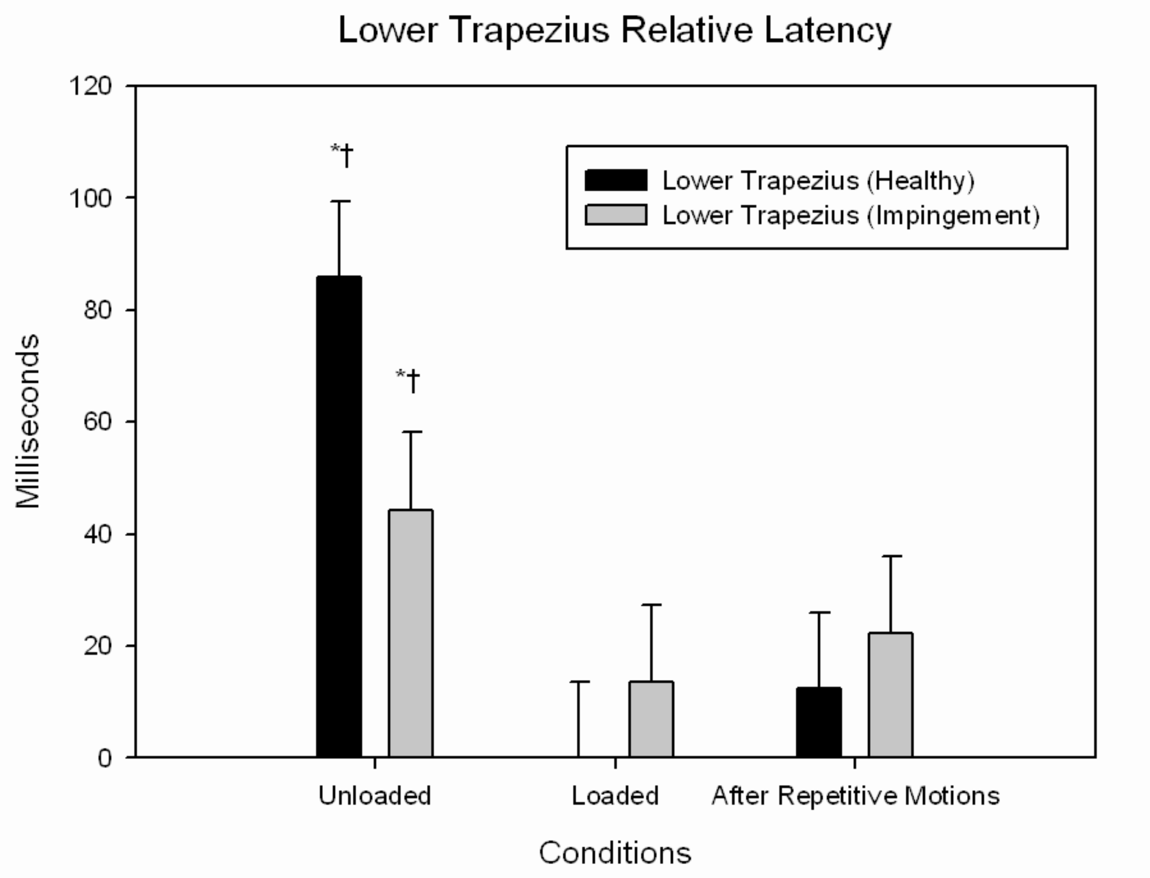


Figure 4.2b Relative latency of lower trapezius.

* signifies difference between the unloaded condition and loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

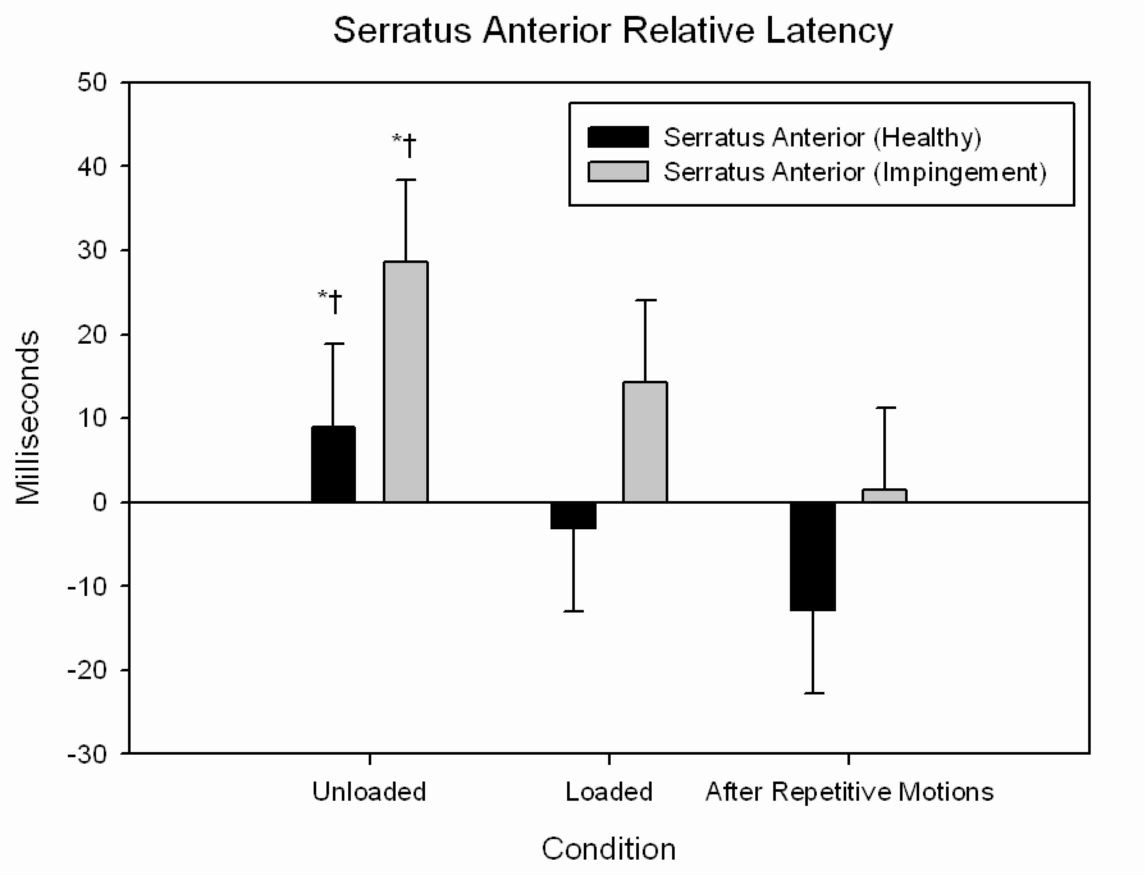


Figure 4.3c Relative latency of serratus anterior.

* signifies difference between the unloaded condition and loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

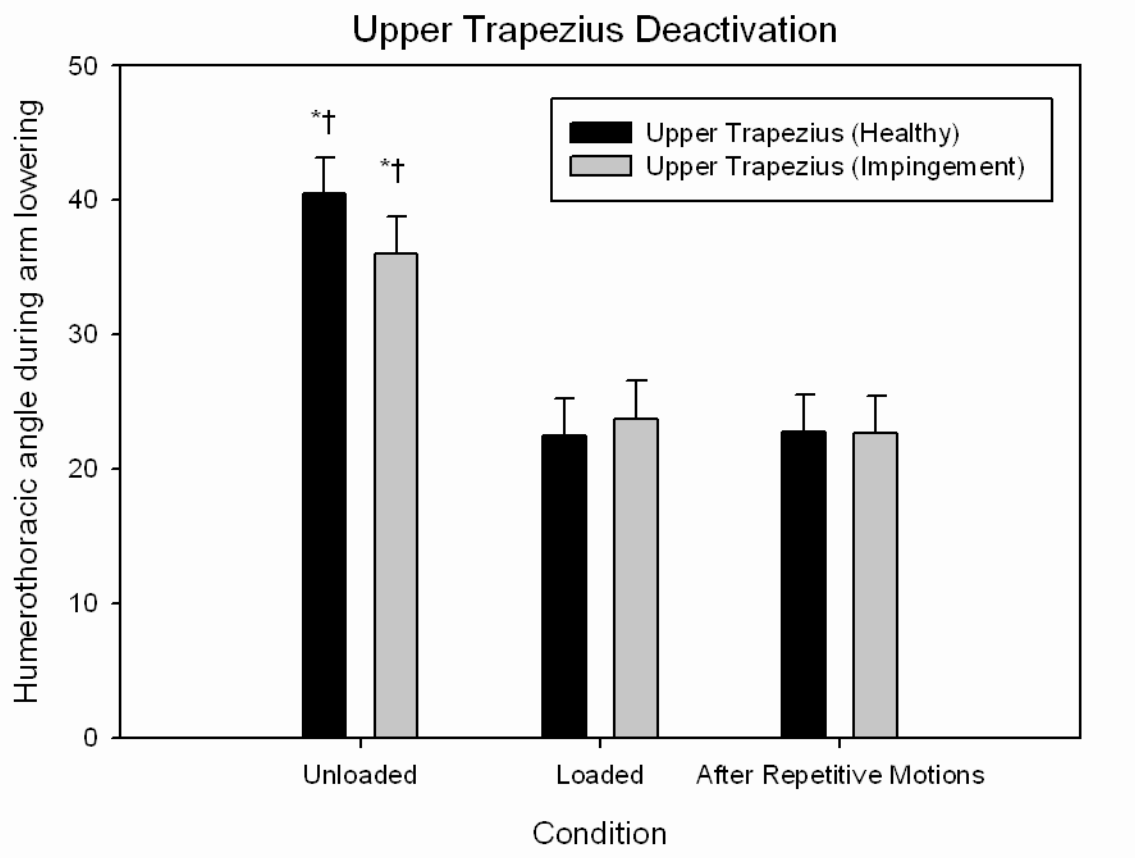


Figure 4.3a Humerothoracic angle corresponding to deactivation of upper trapezius.

* signifies difference between the unloaded condition and loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

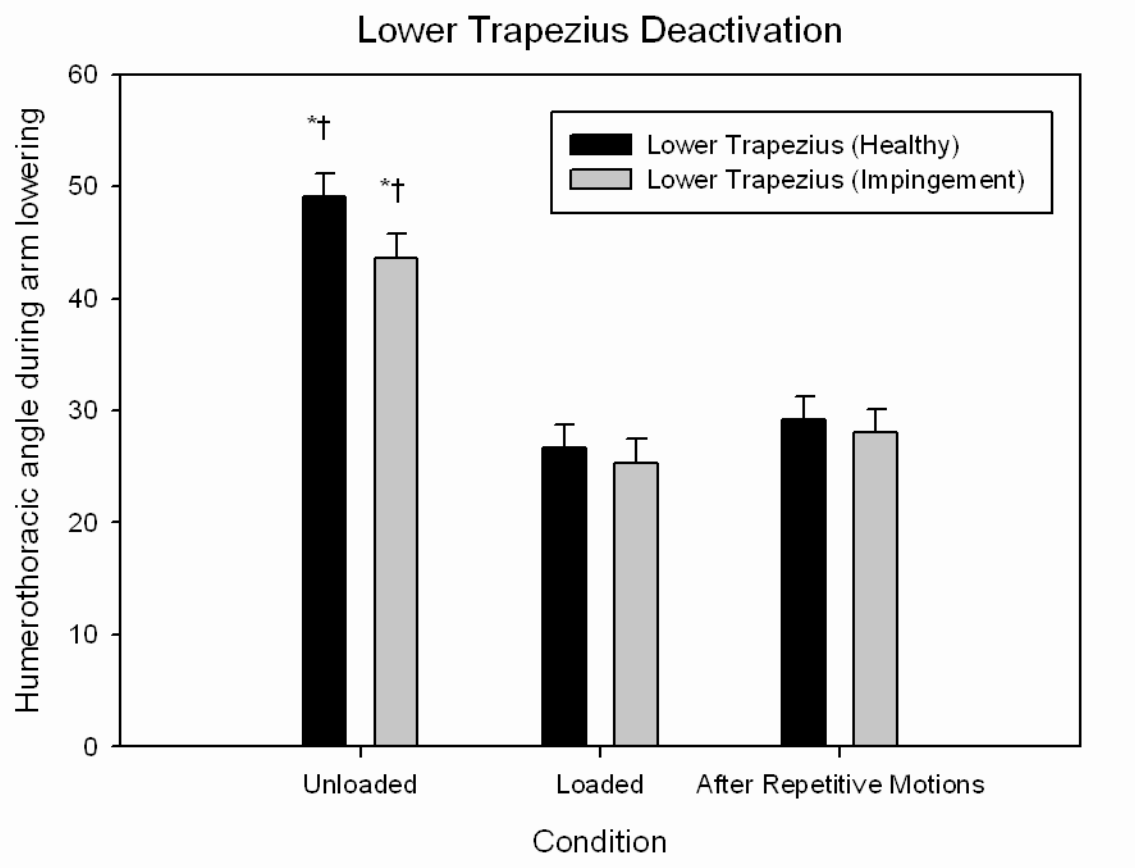


Figure 4.3b Humerothoracic angle corresponding to deactivation of lower trapezius.

* signifies difference between the unloaded condition and loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)

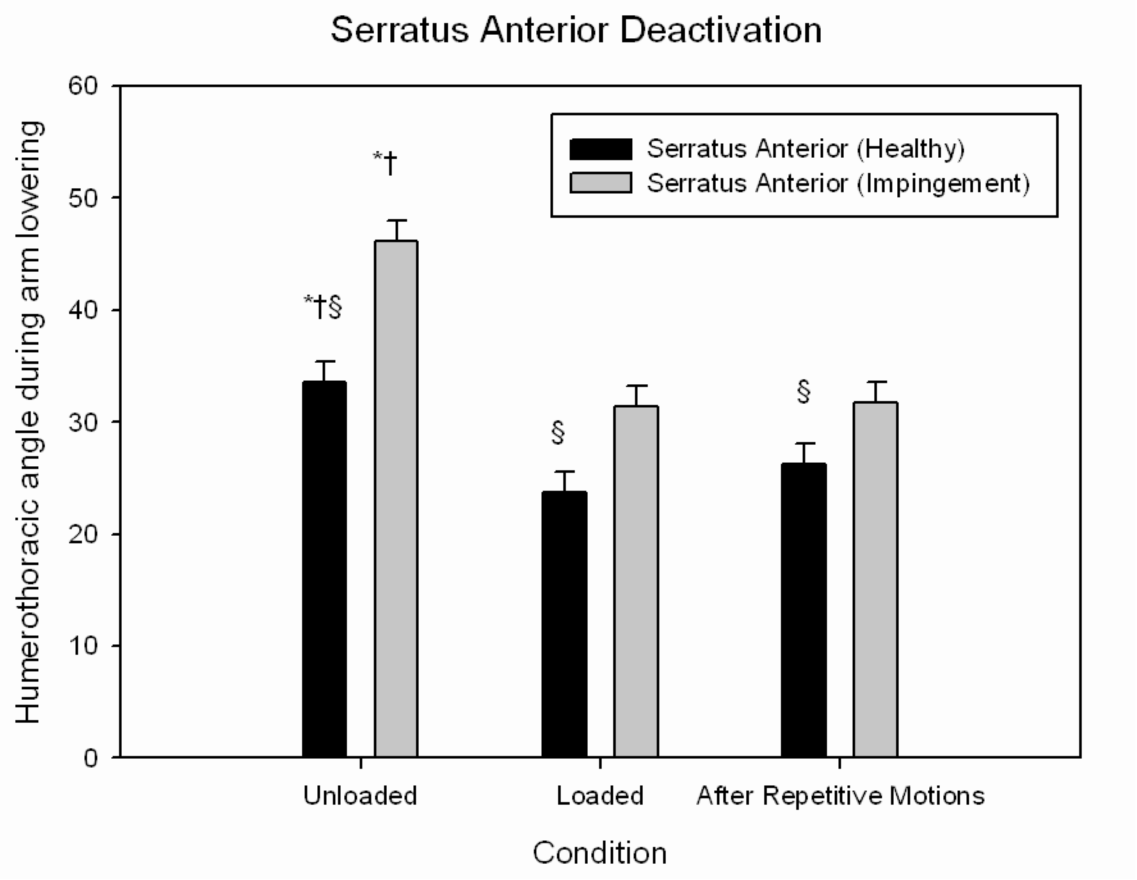


Figure 4.3c Humerothoracic angle corresponding to deactivation of serratus anterior.

* signifies difference between the unloaded condition and loaded condition ($p<0.05$)
† signifies difference between the unloaded condition and the after repetitive motion condition ($p<0.05$)
§ signifies group difference at a condition ($p<0.05$)

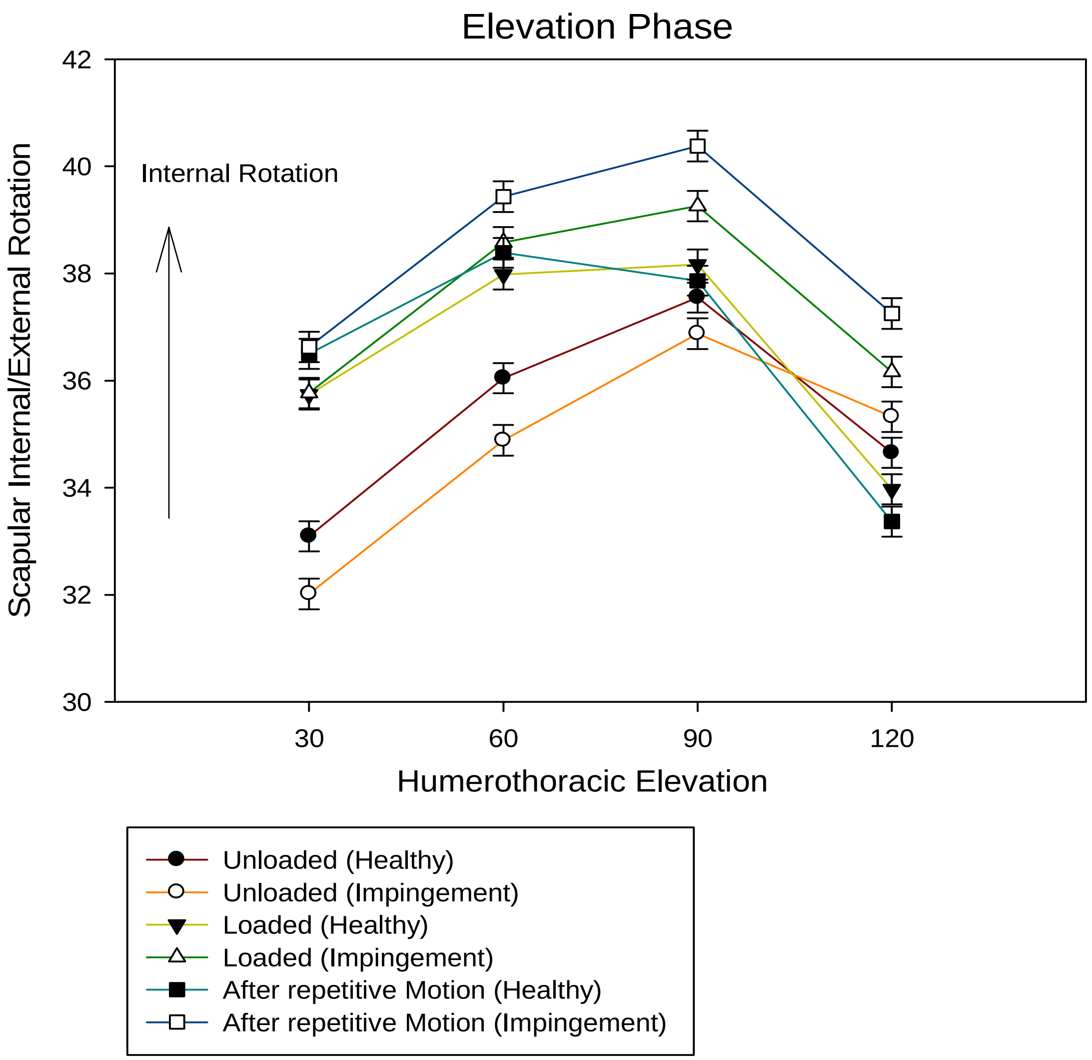


Figure 4.4a Scapular internal/external rotation during the elevation phase.

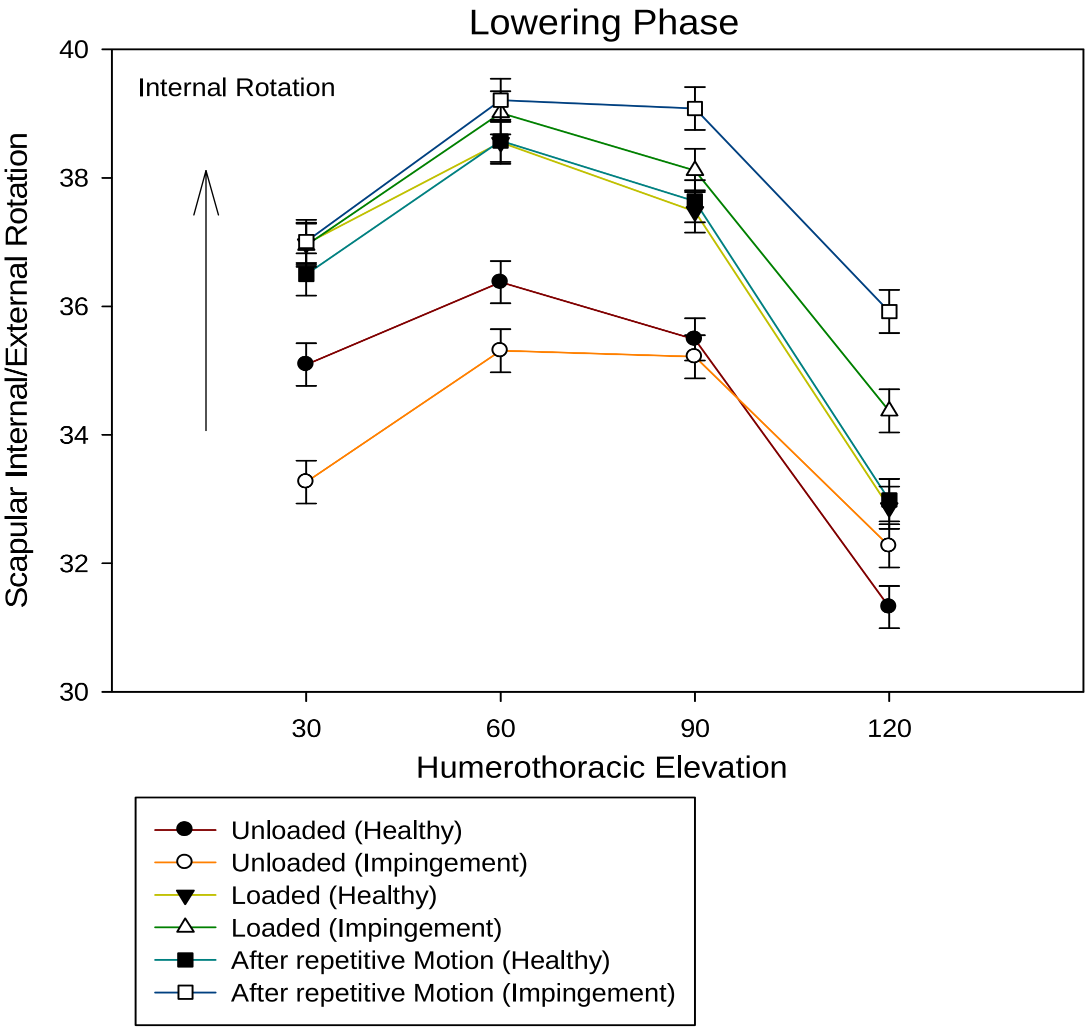


Figure 4.4b Scapular internal/external rotation during the lowering phase.

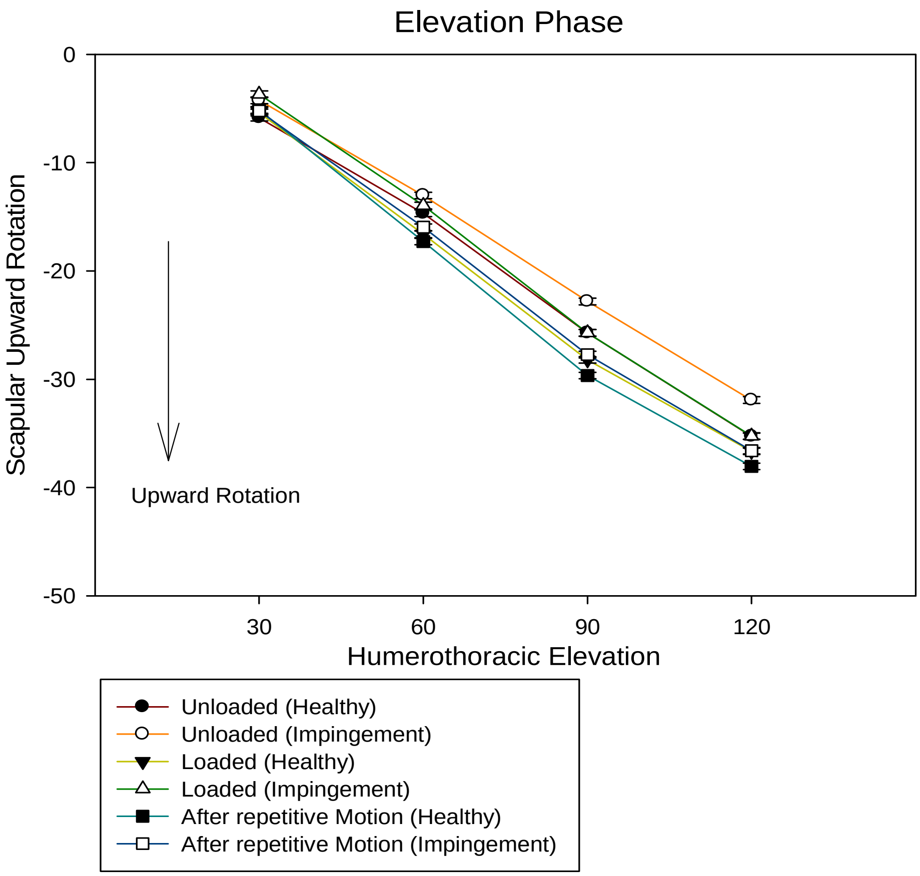


Figure 4.4c Scapular upward rotation during the elevation phase.

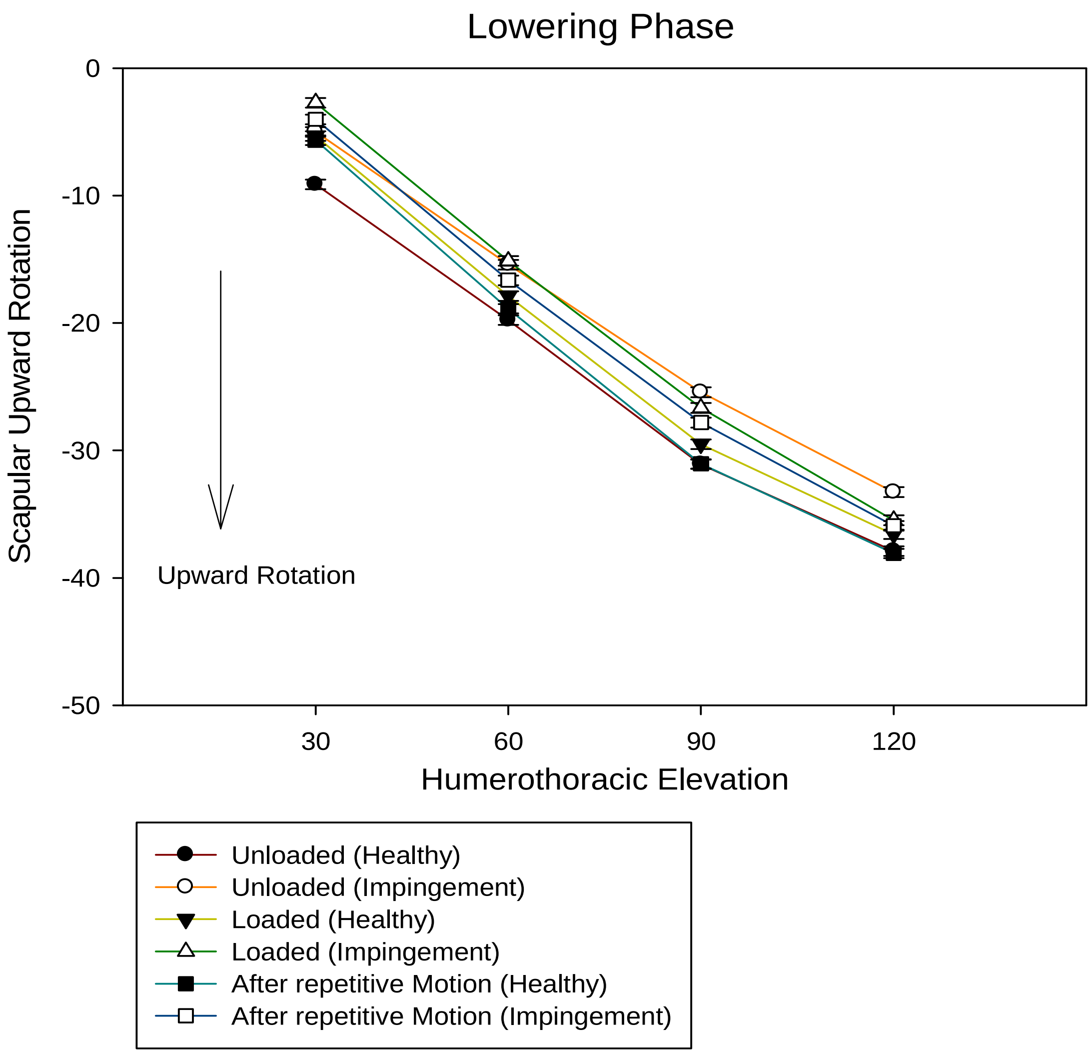


Figure 4.4d Scapular upward rotation during the lowering phase.

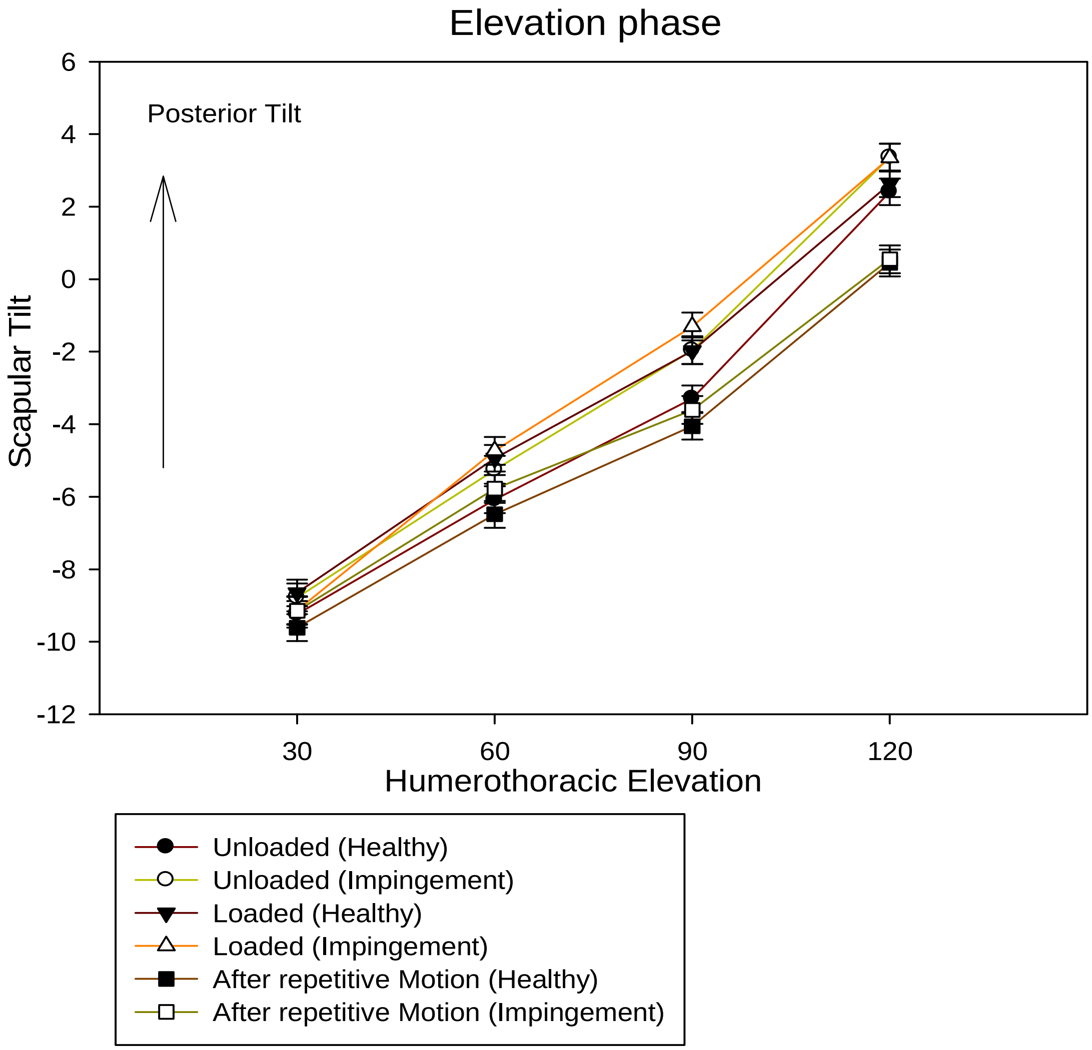


Figure 4.4e Scapular tilt during the elevation phase.

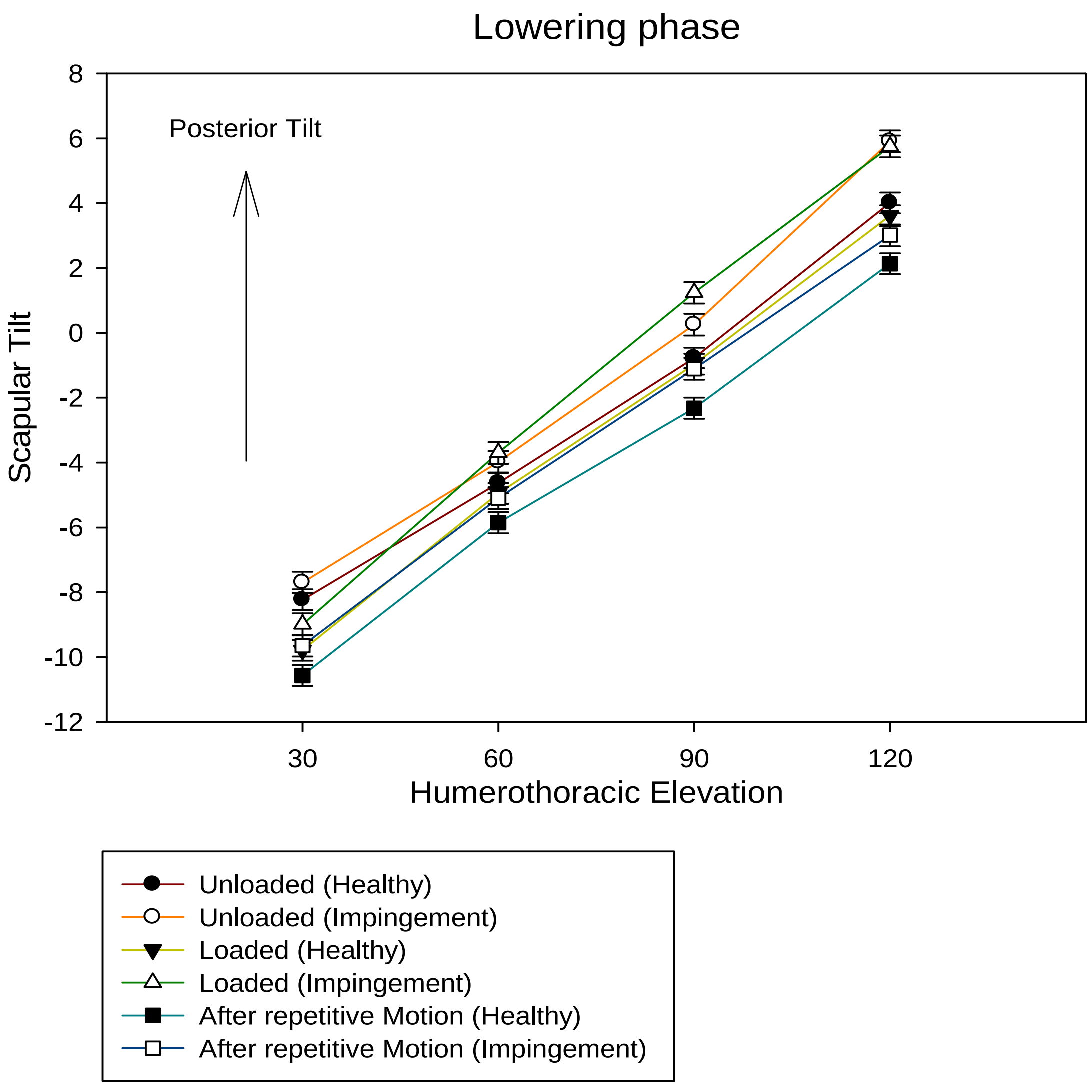


Figure 4.4f Scapular tilt during the lowering phase.

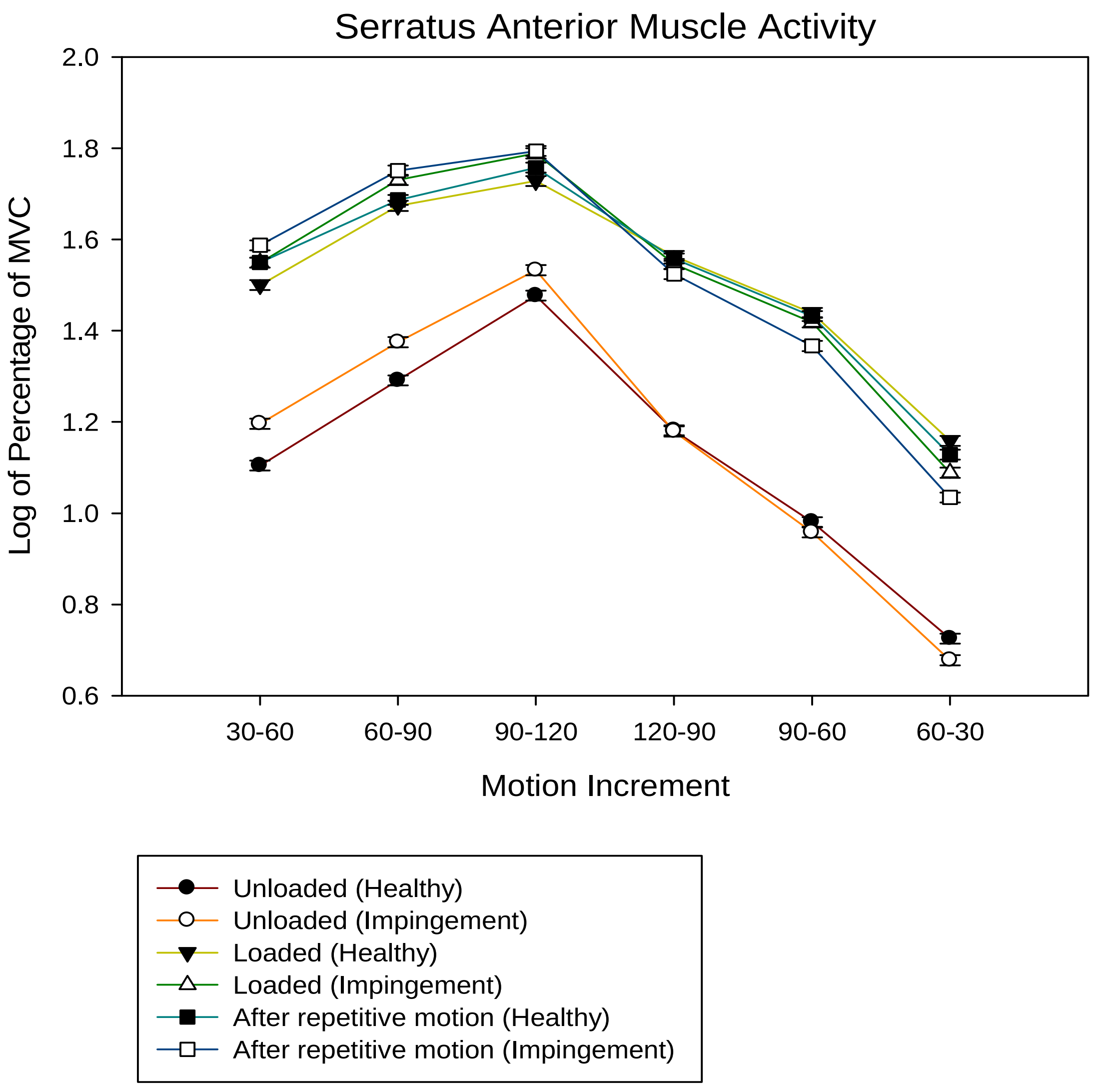


Figure 4.5a Serratus Anterior muscle activity.

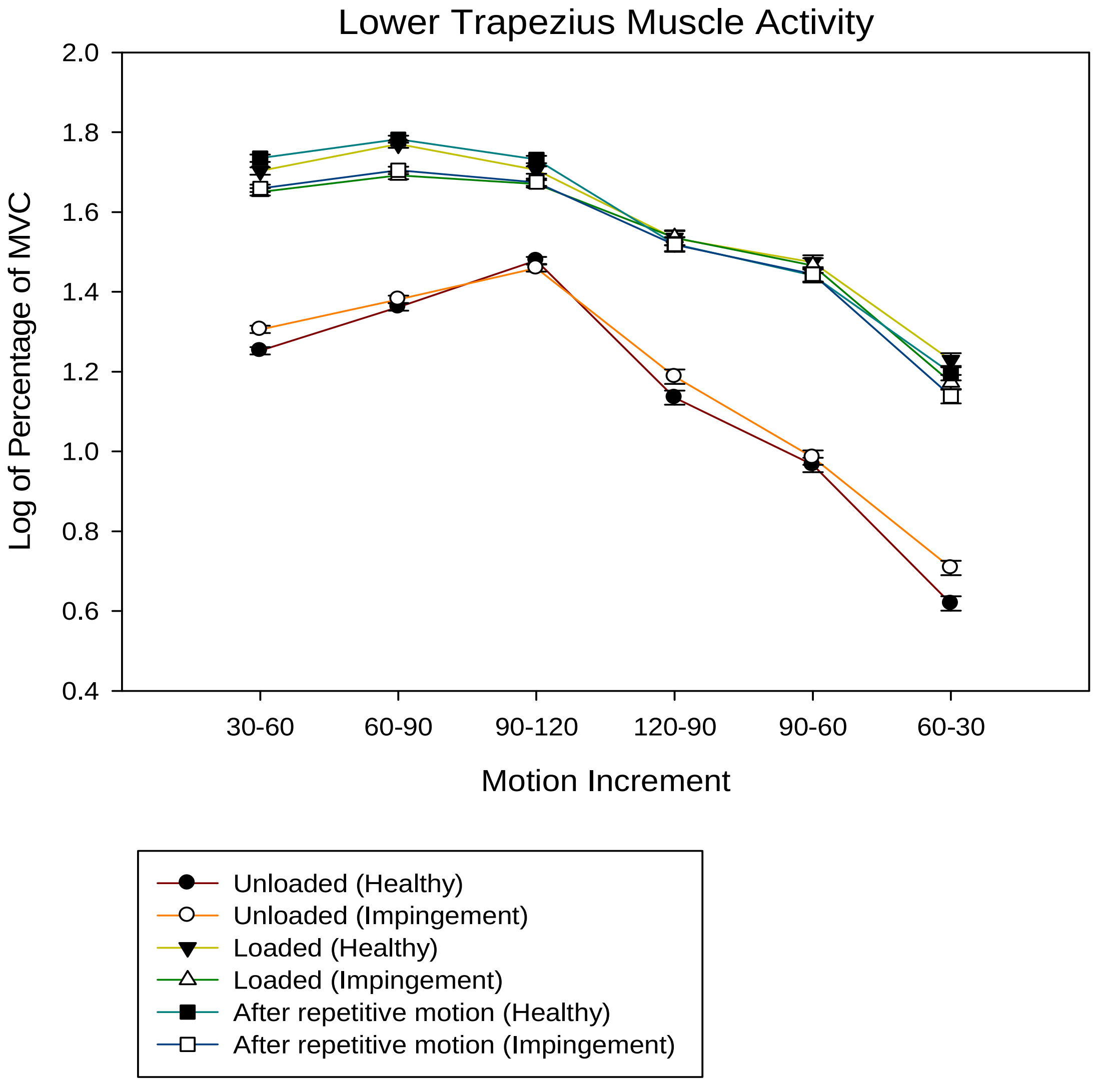


Figure 4.5b Lower Trapezius muscle activity.

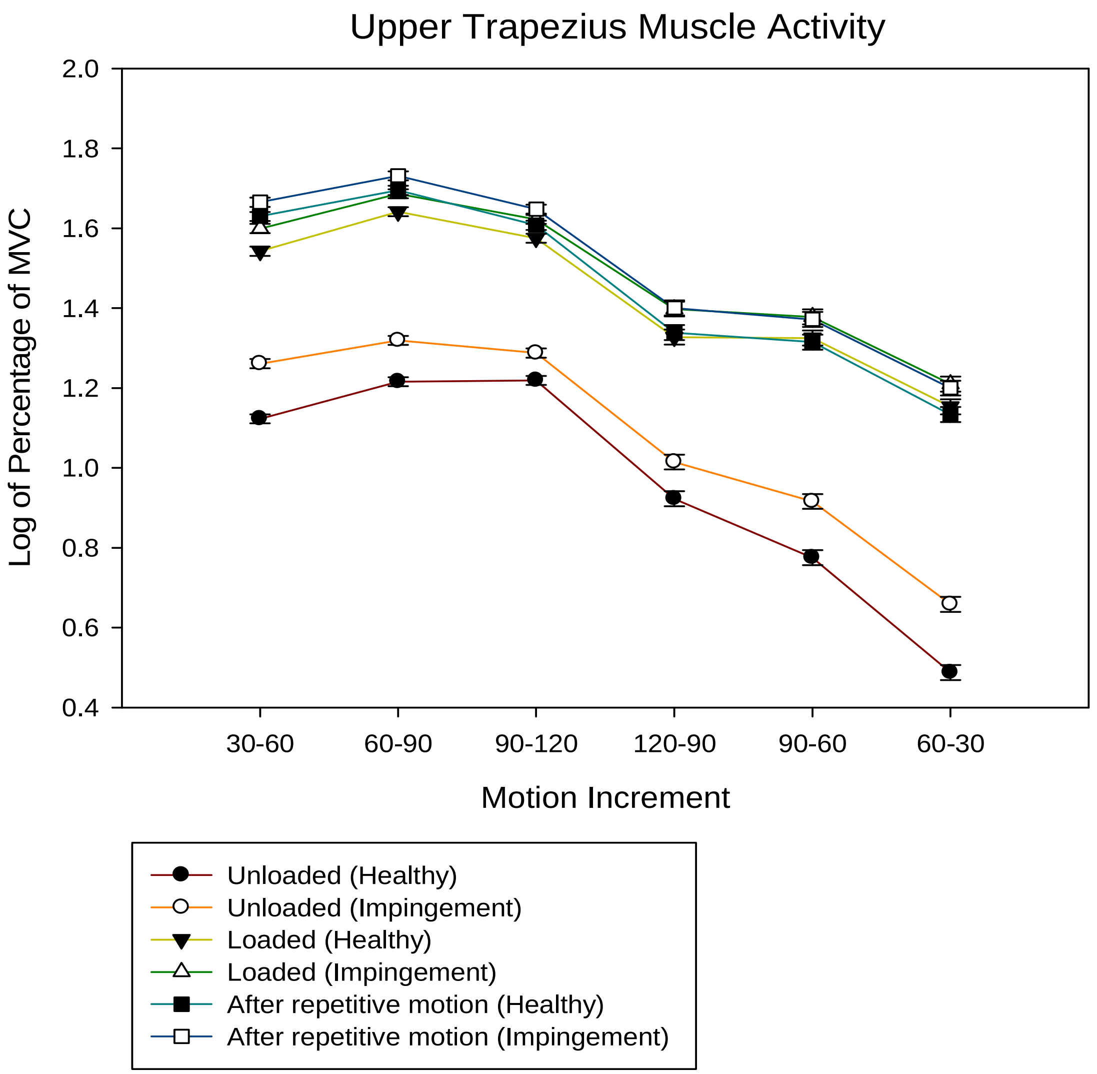


Figure 4.5c Upper Trapezius muscle activity.

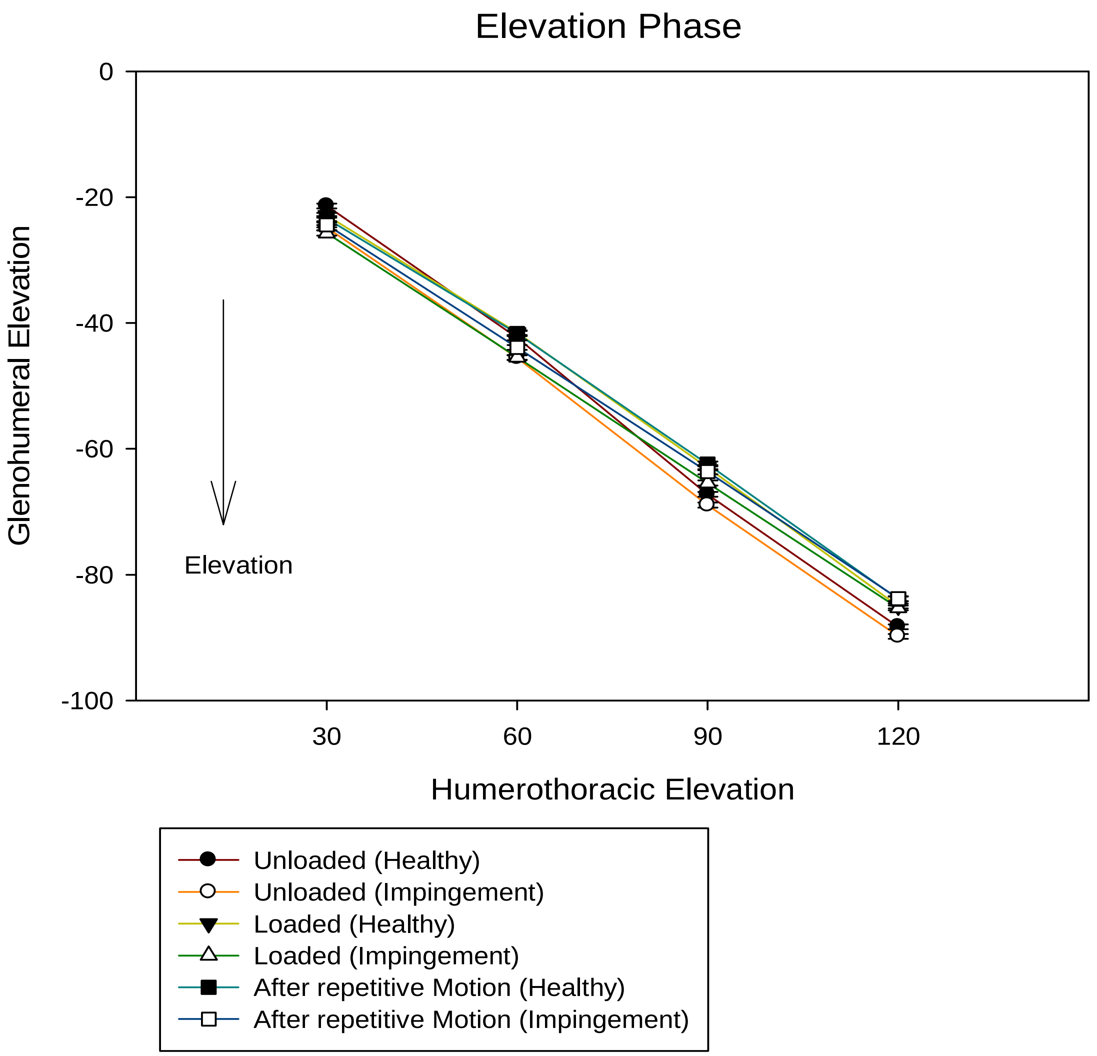


Figure 4.6a Glenohumeral angle of elevation during the elevation phase.

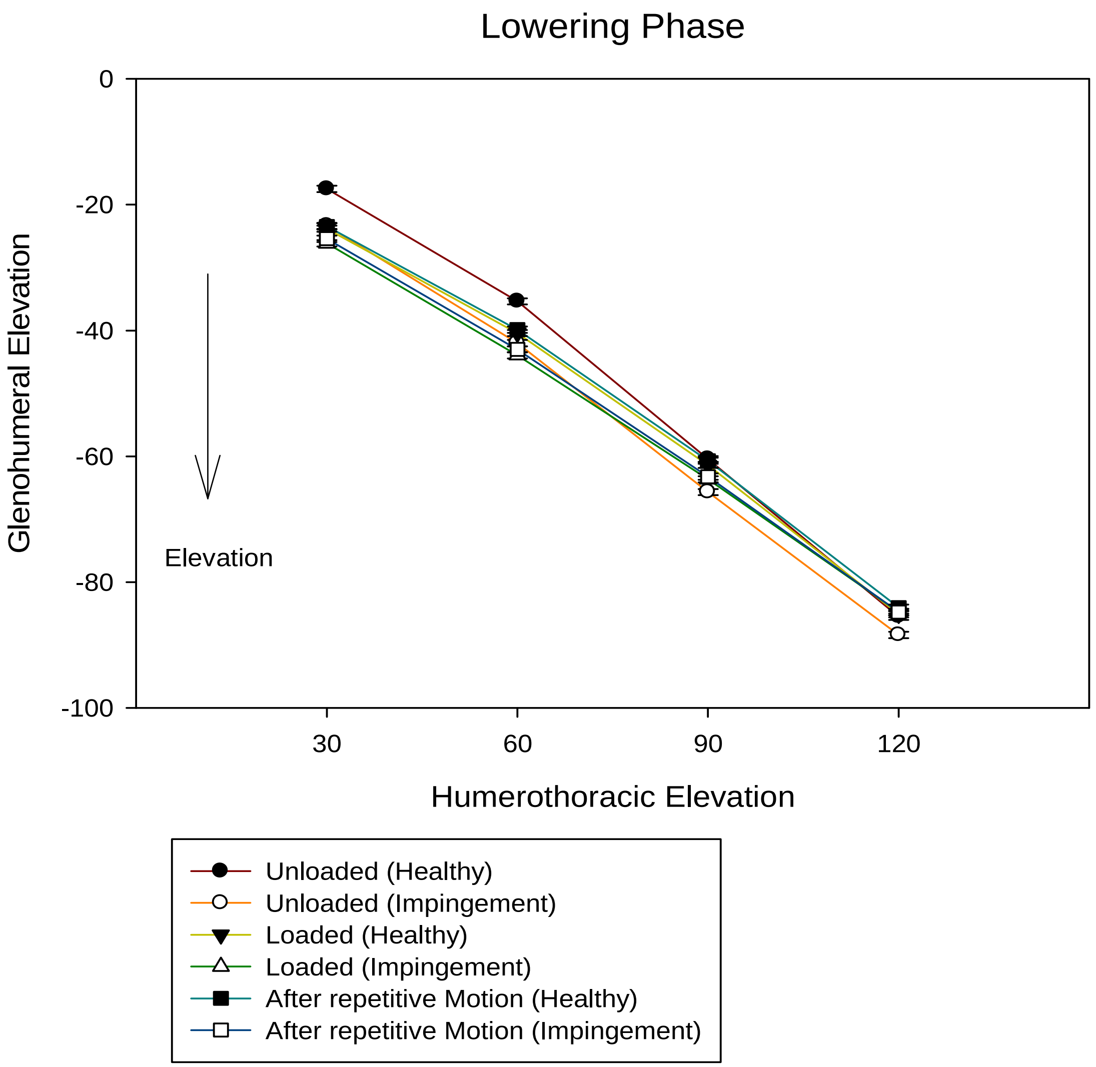


Figure 4.6 b Glenohumeral angle of elevation during the lowering phase.

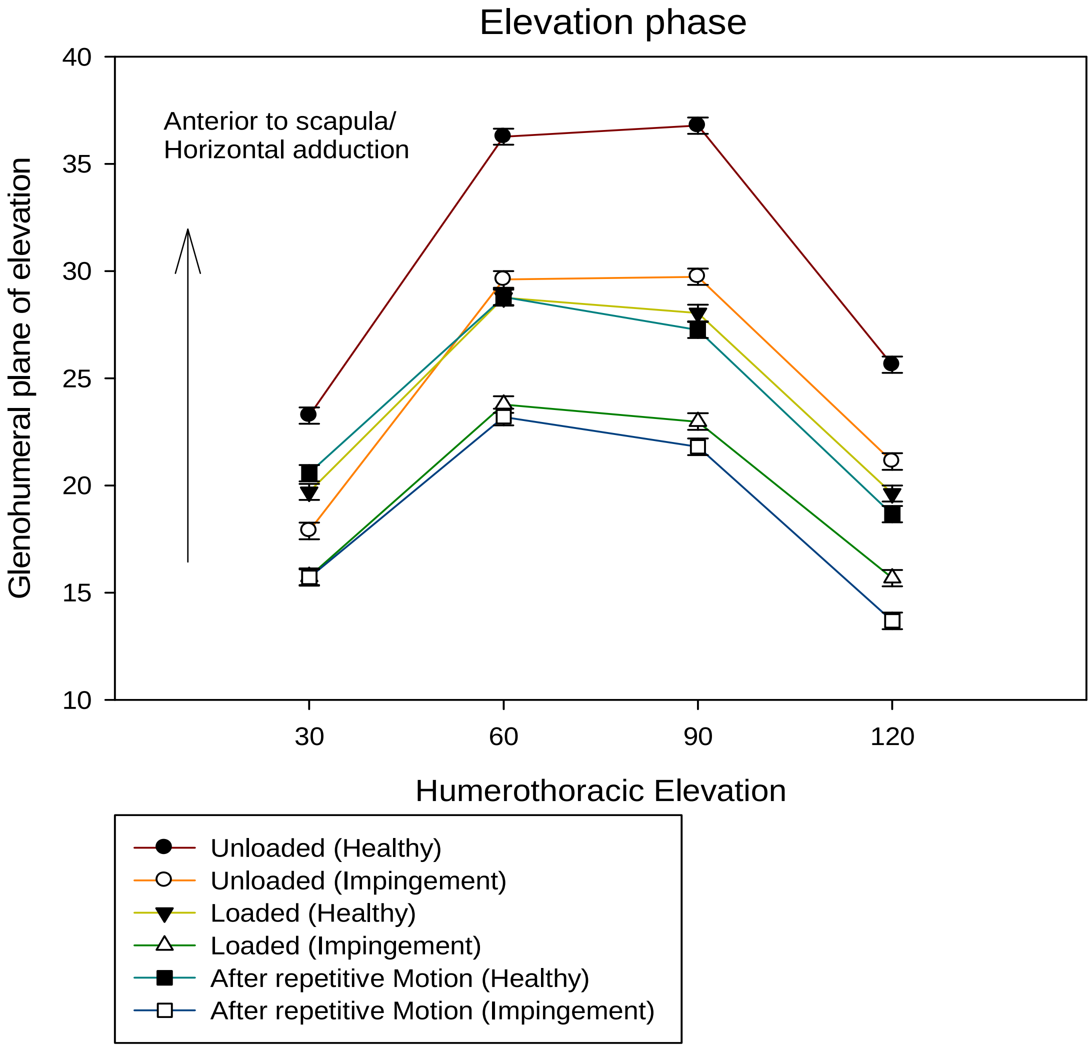


Figure 4.6 c Glenohumeral plane of elevation during the elevation phase.

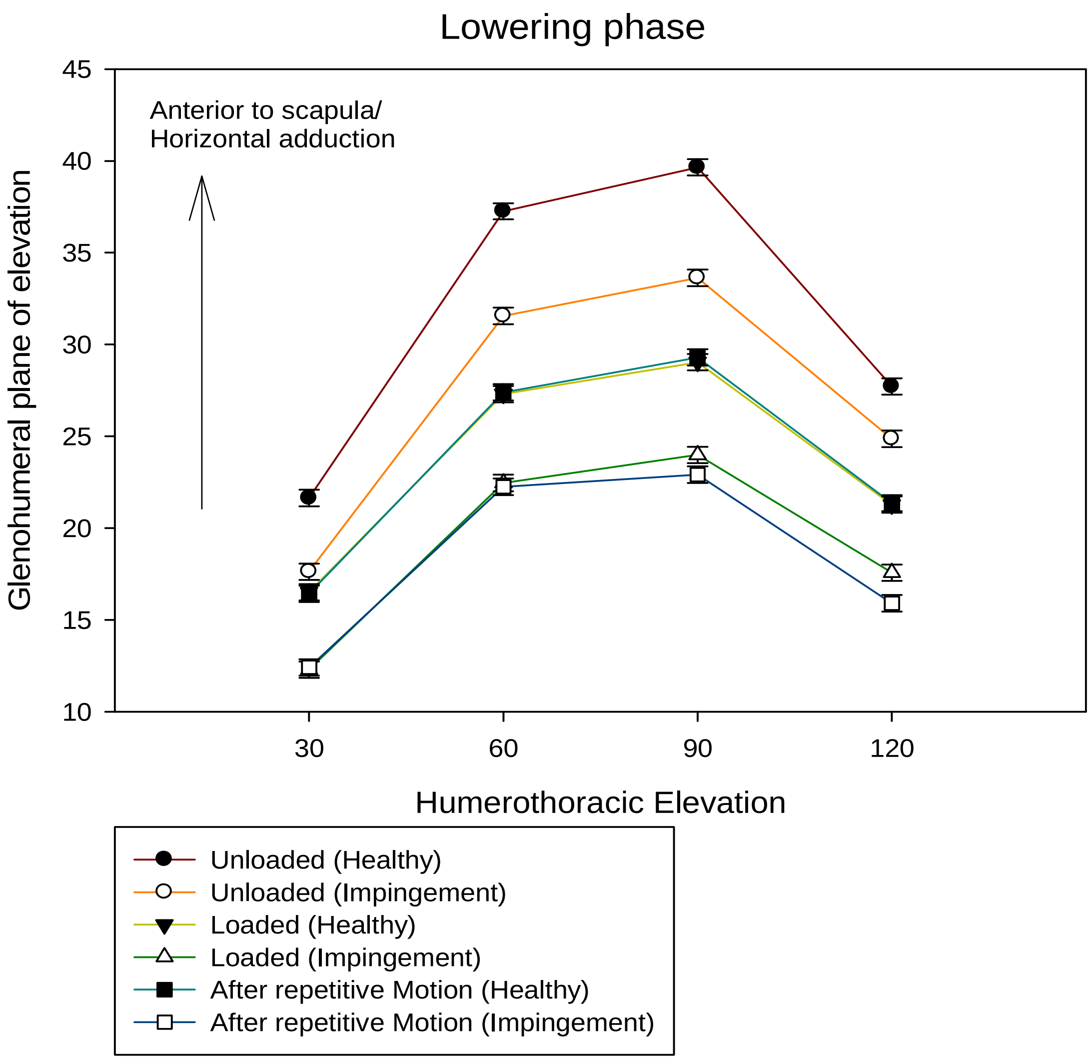


Figure 4.6d Glenohumeral plane of elevation during the lowering phase.

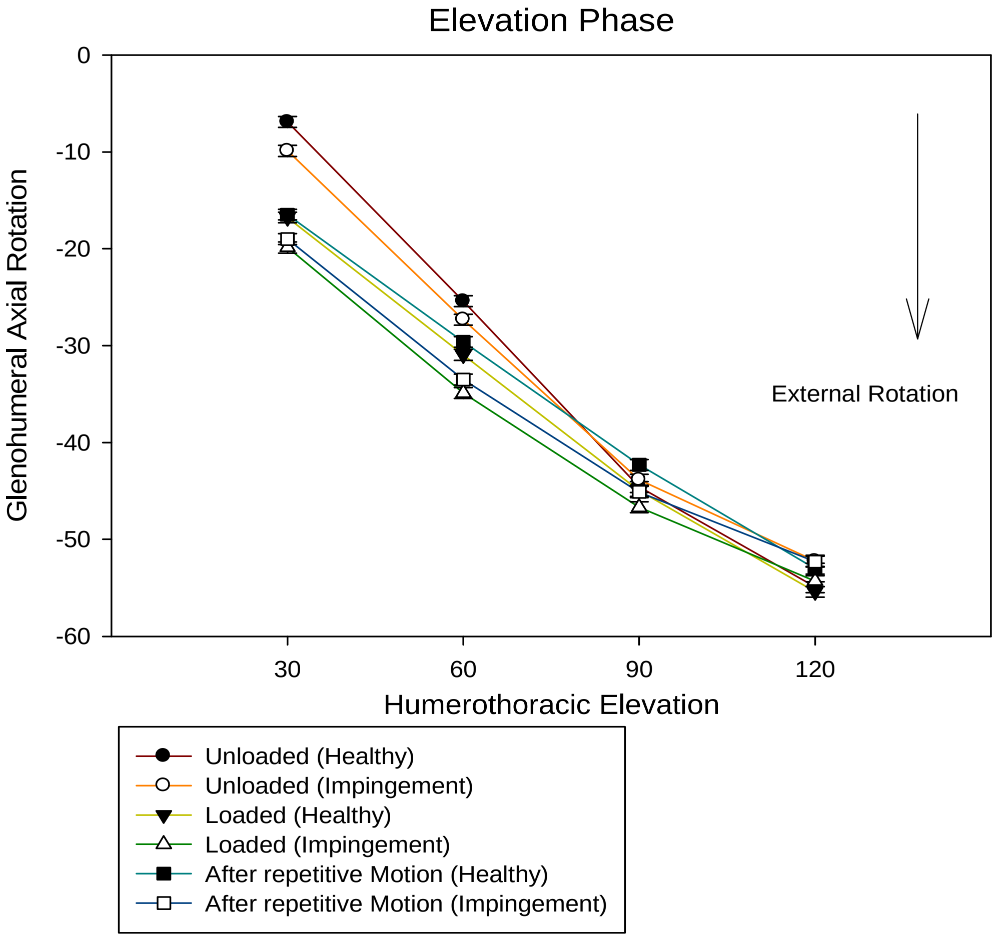


Figure 4.6e Glenohumeral axial rotation during the elevation phase.

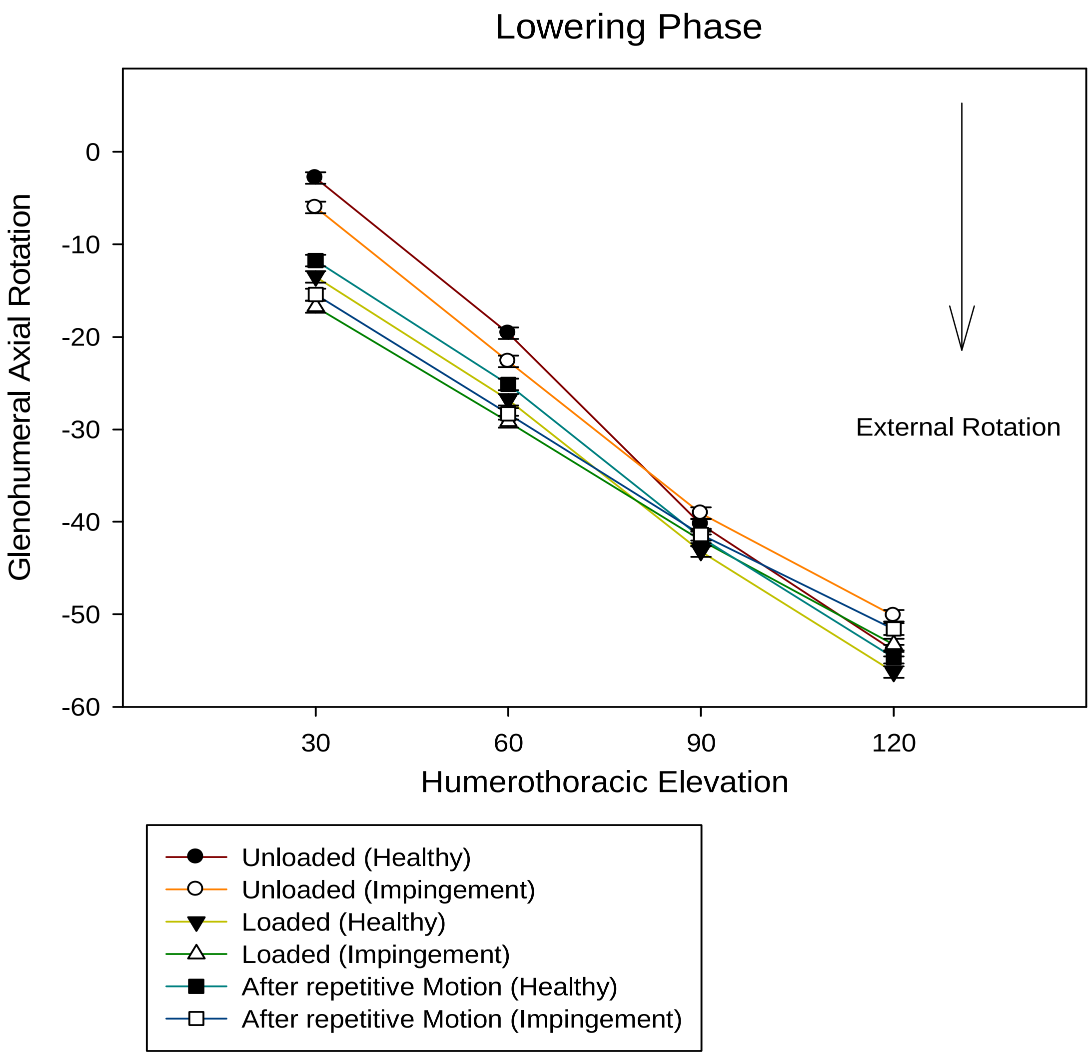


Figure 4.6 f Glenohumeral axial rotation during the lowering phase.

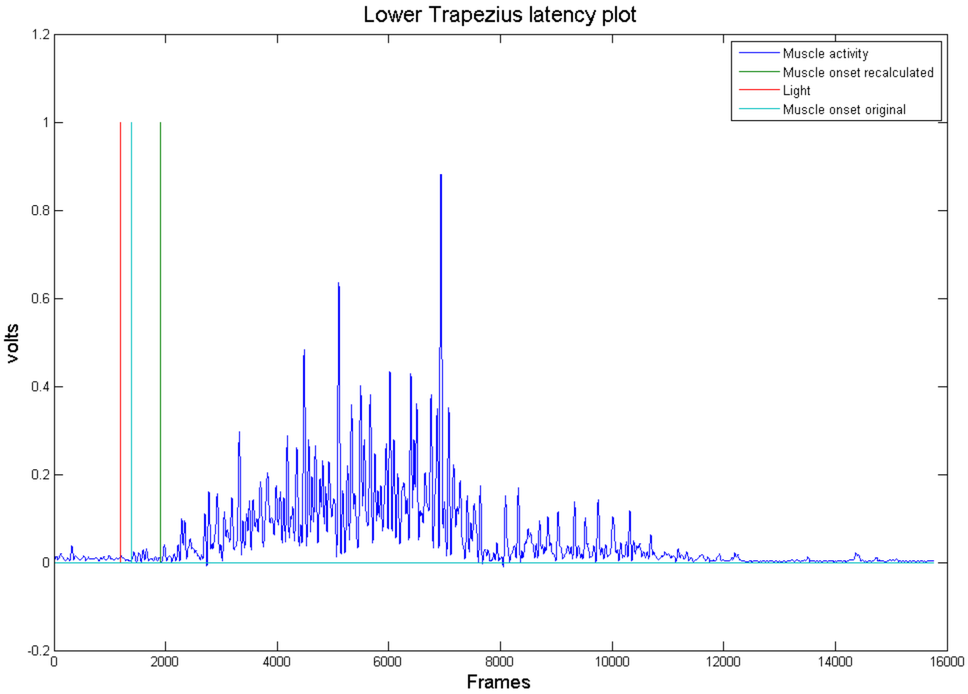


Figure 5.1 Spurt of lower trapezius muscle activity to cause inaccurate latency estimation.

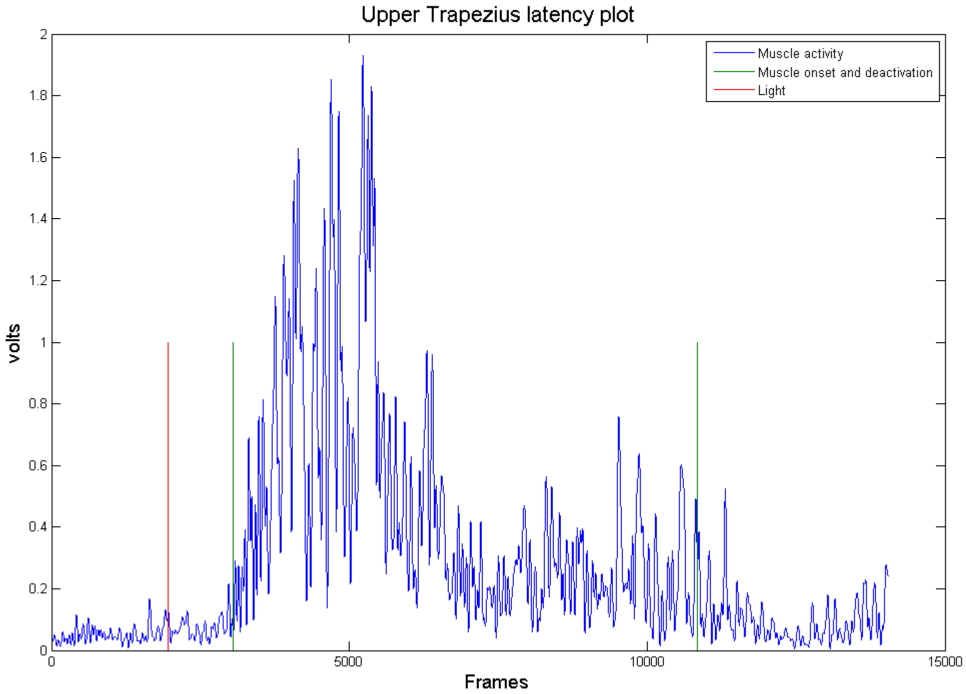


Figure 5.2 Delayed activation and early deactivation detected for upper trapezius due to high baseline activity.

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Appendix 1

Clinical Exam

(Tested Unilaterally)

STANDING

	Left	Right
ROM:		
Flexion (150° min)	WNL Y/N	WNL Y/N
Painful arc unloaded	+/-	+/-
Painful arc loaded (2-4kgs)	+/-	+/-

Scoliosis screen: ____

Visual observation:

Scapular resting position: ________________________________

Flexion:
Medial border: – immature or excessive prominence/ concentric or eccentric phase
Inferior angle: – immature or excessive prominence/ concentric or eccentric phase

Flexion ROM with 2-4 Kg wt:
Medial border: – immature or excessive prominence/ concentric or eccentric phase
Inferior angle: – immature or excessive prominence/ concentric or eccentric phase

Ability to perform repetitive motion (15 at least with weight)	Y/N	Y/N

SITTING

	Left	Right
Cervical (reproduce shoulder symptoms):		
AROM	+/-	+/-
Quadrant Tests	+/-	+/-
Compression/Distraction test	+/-	+/-
Resisted shoulder:		
ER – 90°	+/-	+/-
Abduction-90°	+/-	+/-
IR-90°	+/-	+/-
Impingement Signs (if >2):		
Neer	+/-	+/-

Modified Hawkins/Kennedy	+/-	+/-
Supraspinatus test (Jobe’s)	+/-	+/-
Speed’s Test	+/-	+/-
Yergason’s Test	+/-	+/-
Yocum Test	+/-	+/-

Laxity Tests:

Sulcus Test (if >1 cm)	+/-	+/-
A/P Translation	+/-	+/-

Special Tests:

Lift Off test	+/-	+/-
Drop Arm Test	+/-	+/-
Flip Test	+/-	+/-
External rotation lag test	+/-	+/-
O’Briens Test	+/-	+/-

Pain with Palpation:	+/-	+/-

Localization (circle if positive):

Supraspinatus Long head biceps Coracoid process

AC joint post shoulder

Crepitus during passive arm motion:	+/-	+/-

SUPINE

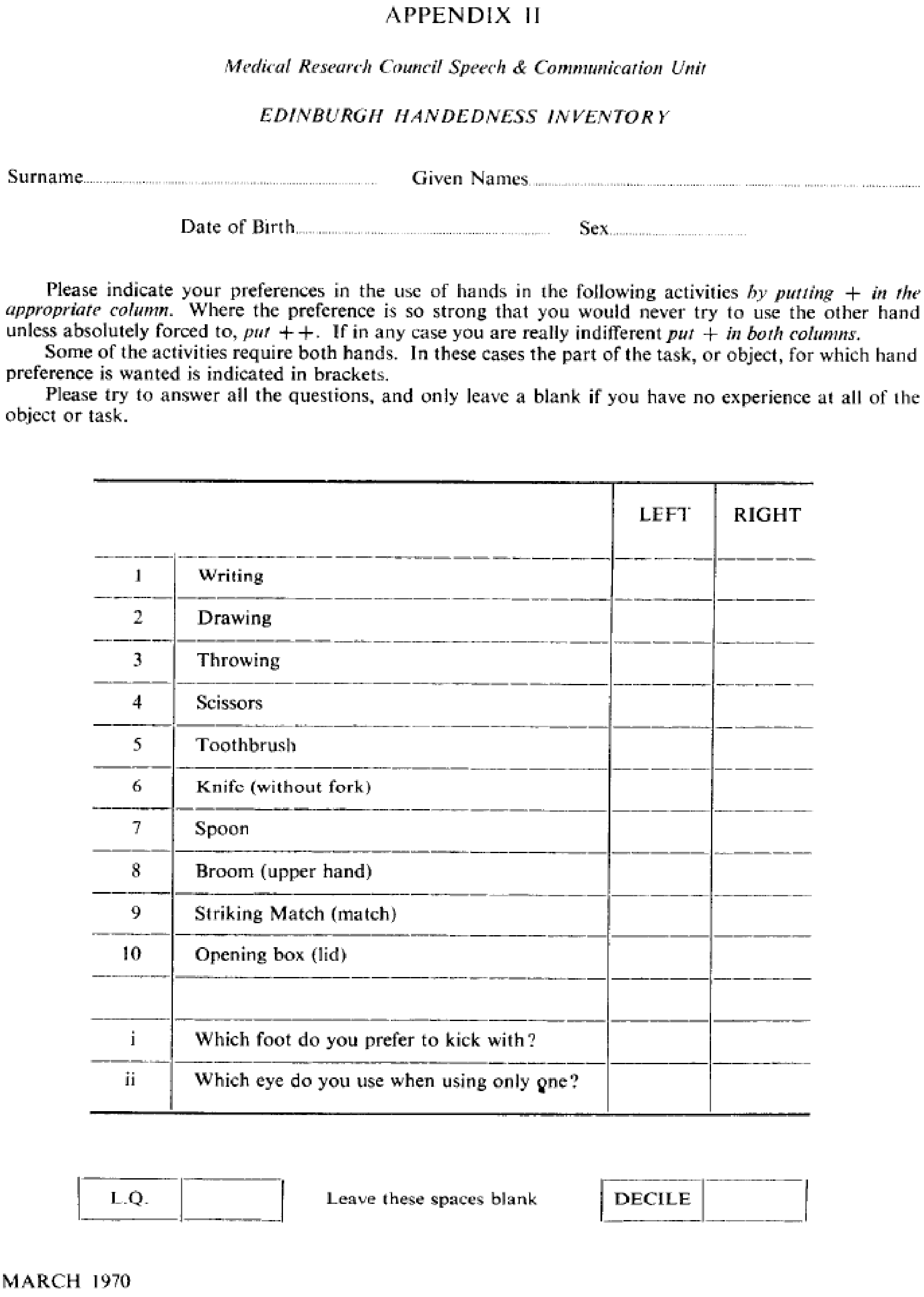
	Left	**Right**
Supine PROM:		
IR (@ 90° ABD	__________	___________
ER (@ 90° ABD)	__________	___________
Anterior Apprehension Test:	+/-	+/-

Fixed kyphosis screen:

Arm length (side to be tested)-

Other:

Appendix 2
Oldfield Hand Dominance Test



APPENDIX II

Medical Research Council Speech & Communication Unit

EDINBURGH HANDEDNESS INVENTORY

Surname.................................... Given Names....................................

Date of Birth.................................... Sex....................

Please indicate your preferences in the use of hands in the following activities *by putting + in the appropriate column.* Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, *put* + +. If in any case you are really indifferent *put + in both columns.*

Some of the activities require both hands. In these cases the part of the task, or object, for which hand preference is wanted is indicated in brackets.

Please try to answer all the questions, and only leave a blank if you have no experience at all of the object or task.

		LEFT	RIGHT
1	Writing		
2	Drawing		
3	Throwing		
4	Scissors		
5	Toothbrush		
6	Knife (without fork)		
7	Spoon		
8	Broom (upper hand)		
9	Striking Match (match)		
10	Opening box (lid)		
i	Which foot do you prefer to kick with?		
ii	Which eye do you use when using only one?		

L.Q.		Leave these spaces blank	DECILE	

MARCH 1970

Appendix 3
Subject Questionnaire Form

Name ____
E-mail address ______________________________
Phone____________________________
Age ________________
Height ______________
Weight ______________
Gender ___________

Optional information-
Ethnic category (check one): _________ Hispanic or Latino
_________ Non-Hispanic or Latino

Racial category (check one): _________ American Indian/Alaska Native
_________ Asian
_________ Native Hawaiian or Other Pacific Islander
_________ Black or African American
_________ White

Subject Questionnaire Form

Do you consider yourself left or right-handed? L/R
Side to test ______________
Do you have a history of skin sensitivity or skin allergies, especially with tape? Y/N

Do you have any other diagnosed medical condition? Y/N
(Lupus, RA, MS, brain injury, spinal cord injury, MND, myasthenia gravis, Stroke, Nerve injury)

Do you have a history of performing overhead work activities? Y/N
If yes: What occupation(s)? ____
What activities? ____
How often? (how much during workday?)______________________
For how long? (years & months) ______________________________

Have you played competitive or recreational sports within the last 5 years? Y/N
If yes: which sport(s)? ____
What level of competition? ___________________________________
How often? (per week)____
For how long? (years & months) _______________________________

Have you ever injured your shoulder(s)? Y/N
Has your injury occurred in the past 2 months? Y/N
When was the injury or onset of symptoms? ______________

If yes, what was the type of injury:

Labral tear		Y/N	L/R
Glenohumeral joint dislocation		Y/N	L/R
AC or SC joint instability		Y/N	L/R

What if any stabilization was performed?_________________________________
What if any displacement was noted?____________________________________

Fracture:	collarbone (clavicle)	Y/N	L/R
	Upper arm (humerus)	Y/N	L/R
	Shoulder blade (scapula)	Y/N	L/R
Shoulder tendonitis		Y/N	L/R
Shoulder impingement			Y/N
L/RRotator cuff tears		Y/N	L/R
Shoulder bursitis		Y/N	L/R
Scoliosis		Y/N	L/R
Shoulder strain		Y/N	L/R

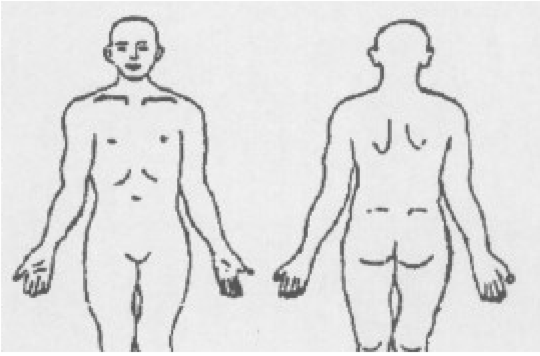
Other:
Describe injury (ies):

Have you ever had surgery on your shoulder(s)? Y/N L/R

If yes, describe: ____

Are you currently experiencing pain in your shoulder(s)? Y/N L/R

If yes, mark location ____



If yes, describe Type-

Duration-
Intensity-

Aggravating factors-

Relieving factors-

If yes, do you have difficulty in sleeping on affected side? Y/N
If yes, do you wake up in night due to pain in shoulder? Y/N

Are you currently experiencing any neck pain? Y/N L/R

If yes, describe: ____

Are you currently experiencing any arm pain, numbness or tingling? Y/N L/R

If yes, describe: ____

Are you currently receiving any treatment for your shoulder(s)? Y/N L/R

If yes, describe: ____

Are you currently receiving any treatment that includes narcotics or (anti-spasmodic) muscle relaxant drugs? Y/N

Appendix 4
CONSENT FORM

Scapular muscle latency, shoulder kinematics and muscle activity in people with and without shoulder impingement

You are invited to participate in a research study of human shoulder motion. You were selected as a possible participant because you contacted one of the investigators and either you had a normal shoulder without any past or current problems or you have a past shoulder problem. We ask that you read this form and ask any questions you may have before agreeing to be in the study.

This study is being conducted through the Department of Physical, Medicine and Rehabilitation Sciences by Vandana Phadke, BPT.

Study Purpose

One of the purposes of the study is to identify the differences in muscle activation and deactivation patterns between people with and without shoulder impingement. This would give us information about the differences, if any exist, for the motor control strategies between the groups. The conservative and exercise treatment for shoulder pain are based on the assumptions about how the shoulder actually moves and are usually targeted at strengthening and stretching soft tissue structures. This study will provide combined information about the way our shoulder bones move, muscle activation and deactivation patterns and muscle activity through out the range of motion. An increased understanding of these issues will allow for improvements in exercise programs designed to increase shoulder function for people with these shoulder problems.
The second purpose of the study is to find the effects of having a weight in hand and repetitive motion on the shoulder joint motion and related muscle activity parameters.

Study Procedures

If you agree to participate in this study, we would ask you to do the following:

1. Provide background information to the investigator (age, height, weight; history of shoulder surgery, pain, or injury; and functional limitations).
2. Complete a questionnaire about routine activities using your arm, any history of shoulder problems, regular athletic activities (for example, throwing), and a basic health questionnaire.
3. Receive a clinical screening of your shoulder motion, tenderness, and muscle function.

4. Have motion sensors (about one inch square each) taped to the skin over your shoulder blade and the top of your breast bone, a plastic arm brace strapped to your arm just above the elbow, and up to five muscle sensors taped to the skin over your shoulder muscles.
5. Contract certain muscles for brief periods (30-60 seconds) in specific positions.
6. Perform active motions lifting your arm from your side to up overhead, within a comfortable amount of motion for you after you receive a visual light cue to move.
7. Perform active motions lifting your arm from your side to up overhead with a weight (up to 4 Kgs) in hand within a comfortable amount of motion for you after you receive a visual light cue to move.
8. Photographs and videos will be taken during the experimental procedure. The use of such photographic data is for research purposes only. They will not be published without your consent. Photos will not include your face without your consent.

All sensors will be removed at the end of the data collection session. The testing does not involve any invasive procedures. You will be asked to participate for one session lasting up to two hours.

Risks of Study Participation

The study has the following risks. First, a foreseeable risk or discomfort to you as a subject is minor skin irritation (possible redness from tape removal) due to the application and removal of the motion or muscle activity sensors. This discomfort would be comparable to the removal of a bandage taped to the skin. Second, this testing may result in mild muscle soreness from contracting muscles. Third, you will be asked to stand for up to 45 minutes during experiment which may cause tiredness or fainting due to pooling of blood in the legs. However, if required, you will be allowed to move or sit on chair even after when the sensors are fixed to avoid these risks. Finally, this testing may involve risks to you that are currently unforeseeable.

Benefits of Study Participation

There are no immediate benefits to you for your participation in this study. Information gathered from the study may assist in designing exercise programs for persons with a history of shoulder problems.

Study Costs/Compensation

All procedures for the research study will be performed at no cost to you.
Each subject will receive $15 at the end of data collection to compensate for your time in participating in the study.

Research Related Injury

In the event that this research activity results in an injury, treatment will be available, including first aid, emergency treatment and follow-up care as needed. Care for such injuries will be billed in the ordinary manner to you or your insurance company. If you think that you have suffered a research related injury, let the study physicians know right away.

Confidentiality

The records of this study will be kept private. In any publications or presentations, we will not include any information that will make it possible to identify you as a subject.

Voluntary Nature of the Study

Participation in this study is voluntary. Your decision whether or not to participate in this study will not affect your current or future relations with the University, University of Minnesota Medical Center, Fairview or the physicians. If you decide to participate, you are free to withdraw at any time without affecting those relationships.

Contacts and Questions

The researchers conducting this study are Vandana Phadke BPT, and Dr. Paula M Ludewig PhD. You may ask any questions you have now, or if you have questions later, **you are encouraged to** contact Vandana Phadke at 612.625.7930 or 724.549.1732 or contact Dr. Paula Ludewig at 612.626.0420.

If you have any questions or concerns regarding the study and would like to talk to someone other than the researcher(s), you are encouraged to contact the Fairview Research Helpline at telephone number 612-672-7692 or toll free at 866-508-6961. You may also contact this office in writing or in person at University of Minnesota Medical Center, Fairview-Riverside Campus, 2200 Riverside Avenue, Minneapolis, MN 55454.

You will be given a copy of this form to keep for your records.

Statement of Consent

I have read the above information. I have asked questions and have received answers. I consent to participate in the study.

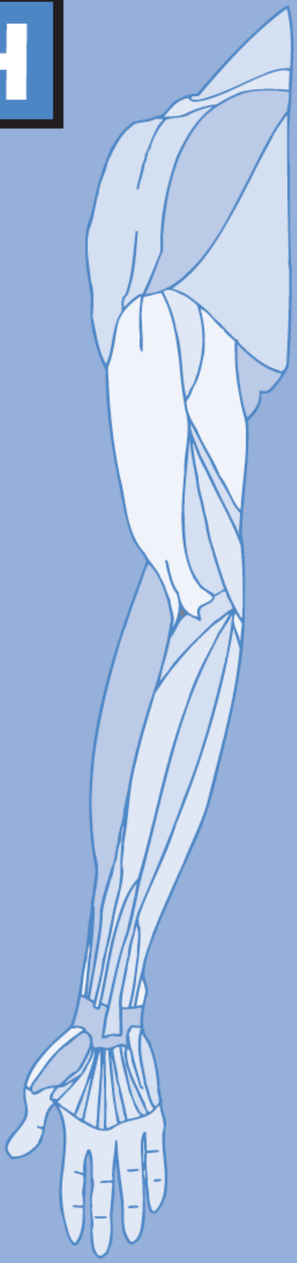
Signature of Subject____
Date_________________

Signature of Investigator____
Date_________________

Appendix 5
Disabilities of the Arm. Shoulder and Hand (DASH)

DISABILITIES OF THE ARM, SHOULDER AND HAND

THE DASH



INSTRUCTIONS

This questionnaire asks about your symptoms as well as your ability to perform certain activities.

Please answer *every question*, based on your condition in the last week, by circling the appropriate number.

If you did not have the opportunity to perform an activity in the past week, please make your *best estimate* on which response would be the most accurate.

It doesn't matter which hand or arm you use to perform the activity; please answer based on your ability regardless of how you perform the task.

DISABILITIES OF THE ARM, SHOULDER AND HAND

Please rate your ability to do the following activities in the last week by circling the number below the appropriate response.

	NO DIFFICULTY	MILD DIFFICULTY	MODERATE DIFFICULTY	SEVERE DIFFICULTY	UNABLE
1. Open a tight or new jar.	1	2	3	4	5
2. Write.	1	2	3	4	5
3. Turn a key.	1	2	3	4	5
4. Prepare a meal.	1	2	3	4	5
5. Push open a heavy door.	1	2	3	4	5
6. Place an object on a shelf above your head.	1	2	3	4	5
7. Do heavy household chores (e.g., wash walls, wash floors).	1	2	3	4	5
8. Garden or do yard work.	1	2	3	4	5
9. Make a bed.	1	2	3	4	5
10. Carry a shopping bag or briefcase.	1	2	3	4	5
11. Carry a heavy object (over 10 lbs).	1	2	3	4	5
12. Change a lightbulb overhead.	1	2	3	4	5
13. Wash or blow dry your hair.	1	2	3	4	5
14. Wash your back.	1	2	3	4	5
15. Put on a pullover sweater.	1	2	3	4	5
16. Use a knife to cut food.	1	2	3	4	5
17. Recreational activities which require little effort (e.g., cardplaying, knitting, etc.).	1	2	3	4	5
18. Recreational activities in which you take some force or impact through your arm, shoulder or hand (e.g., golf, hammering, tennis, etc.).	1	2	3	4	5
19. Recreational activities in which you move your arm freely (e.g., playing frisbee, badminton, etc.).	1	2	3	4	5
20. Manage transportation needs (getting from one place to another).	1	2	3	4	5
21. Sexual activities.	1	2	3	4	5

DISABILITIES OF THE ARM, SHOULDER AND HAND

	NOT AT ALL	SLIGHTLY	MODERATELY	QUITE A BIT	EXTREMELY
22. During the past week, *to what extent* has your arm, shoulder or hand problem interfered with your normal social activities with family, friends, neighbours or groups? *(circle number)*	1	2	3	4	5

	NOT LIMITED AT ALL	SLIGHTLY LIMITED	MODERATELY LIMITED	VERY LIMITED	UNABLE
23. During the past week, were you limited in your work or other regular daily activities as a result of your arm, shoulder or hand problem? *(circle number)*	1	2	3	4	5

Please rate the severity of the following symptoms in the last week. *(circle number)*

	NONE	MILD	MODERATE	SEVERE	EXTREME
24. Arm, shoulder or hand pain.	1	2	3	4	5
25. Arm, shoulder or hand pain when you performed any specific activity.	1	2	3	4	5
26. Tingling (pins and needles) in your arm, shoulder or hand.	1	2	3	4	5
27. Weakness in your arm, shoulder or hand.	1	2	3	4	5
28. Stiffness in your arm, shoulder or hand.	1	2	3	4	5

	NO DIFFICULTY	MILD DIFFICULTY	MODERATE DIFFICULTY	SEVERE DIFFICULTY	SO MUCH DIFFICULTY THAT I CAN'T SLEEP
29. During the past week, how much difficulty have you had sleeping because of the pain in your arm, shoulder or hand? *(circle number)*	1	2	3	4	5

	STRONGLY DISAGREE	DISAGREE	NEITHER AGREE NOR DISAGREE	AGREE	STRONGLY AGREE
30. I feel less capable, less confident or less useful because of my arm, shoulder or hand problem. *(circle number)*	1	2	3	4	5

DASH DISABILITY/SYMPTOM SCORE = $\left[\left(\frac{\text{sum of n responses}}{n}\right) - 1\right] \times 25$, where n is equal to the number of completed responses.

A DASH score may <u>not</u> be calculated if there are greater than 3 missing items.

DISABILITIES OF THE ARM, SHOULDER AND HAND

WORK MODULE (OPTIONAL)

The following questions ask about the impact of your arm, shoulder or hand problem on your ability to work (including homemaking if that is your main work role).

Please indicate what your job/work is:_______________________________

p I do not work. (You may skip this section.)

Please circle the number that best describes your physical ability in the past week. Did you have any difficulty:

	NO DIFFICULTY	MILD DIFFICULTY	MODERATE DIFFICULTY	SEVERE DIFFICULTY	UNABLE
1. using your usual technique for your work?	1	2	3	4	5
2. doing your usual work because of arm, shoulder or hand pain?	1	2	3	4	5
3. doing your work as well as you would like?	1	2	3	4	5
4. spending your usual amount of time doing your work?	1	2	3	4	5

SPORTS/PERFORMING ARTS MODULE (OPTIONAL)

The following questions relate to the impact of your arm, shoulder or hand problem on playing *your musical instrument or sport or both*.
If you play more than one sport or instrument (or play both), please answer with respect to that activity which is most important to you.

Please indicate the sport or instrument which is most important to you:_

○ I do not play a sport or an instrument. (You may skip this section.)

Please circle the number that best describes your physical ability in the past week. Did you have any difficulty:

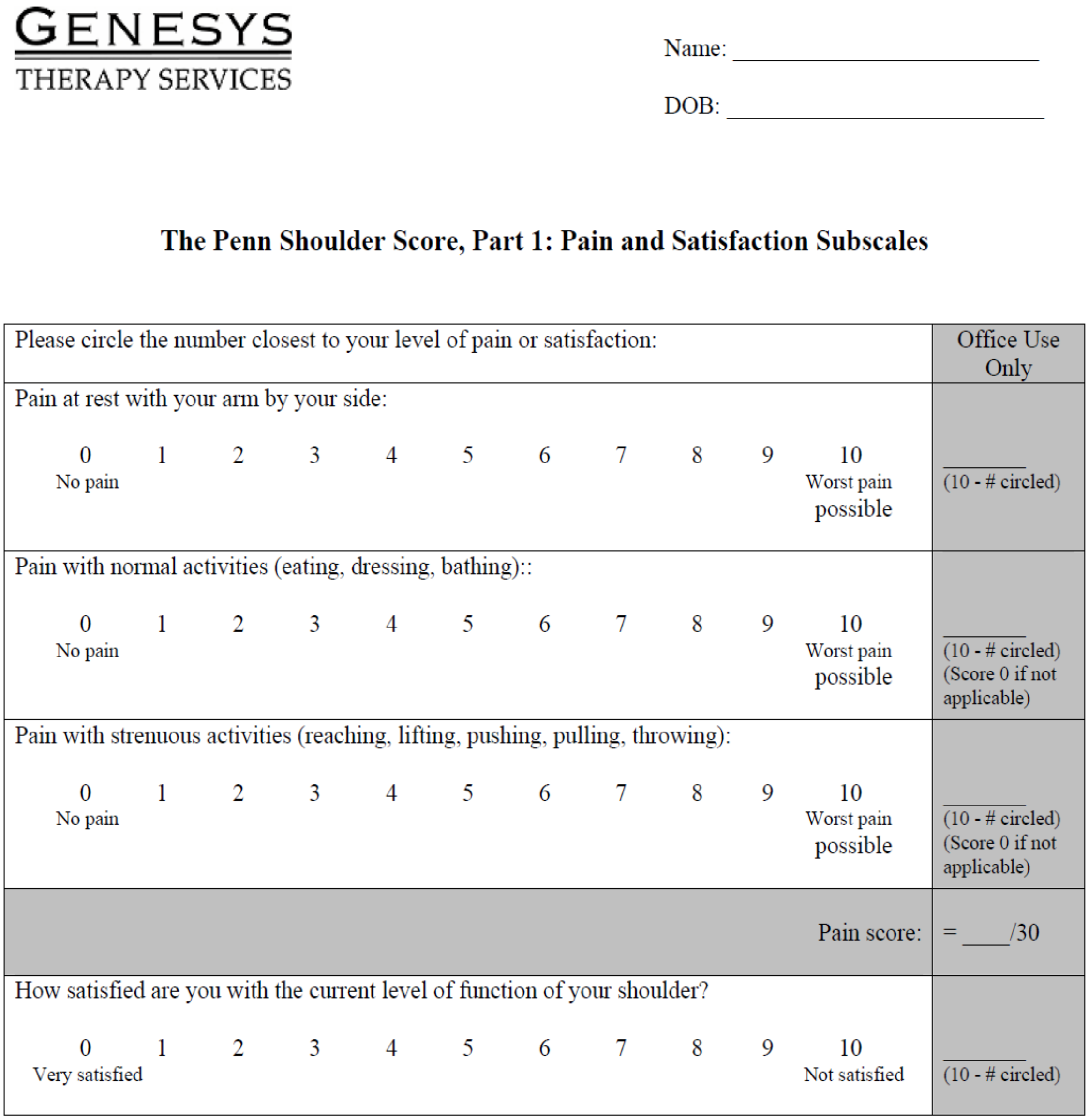
	NO DIFFICULTY	MILD DIFFICULTY	MODERATE DIFFICULTY	SEVERE DIFFICULTY	UNABLE
1. using your usual technique for playing your instrument or sport?	1	2	3	4	5
2. playing your musical instrument or sport because of arm, shoulder or hand pain?	1	2	3	4	5
3. playing your musical instrument or sport as well as you would like?	1	2	3	4	5
4. spending your usual amount of time practising or playing your instrument or sport?	1	2	3	4	5

SCORING THE OPTIONAL MODULES: Add up assigned values for each response; divide by 4 (number of items); subtract 1; multiply by 25.
An optional module score may <u>not</u> be calculated if there are any missing items.

INSTITUTE FOR WORK & HEALTH
INSTITUT DE RECHERCHE SUR LE TRAVAIL ET LA SANTÉ

Appendix 6

Penn Shoulder Score



GENESYS
THERAPY SERVICES

Name: ________________________

DOB: _________________________

The Penn Shoulder Score, Part 1: Pain and Satisfaction Subscales

Please circle the number closest to your level of pain or satisfaction:	Office Use Only
Pain at rest with your arm by your side: 0 1 2 3 4 5 6 7 8 9 10 0 = No pain; 10 = Worst pain possible	________ (10 - # circled)
Pain with normal activities (eating, dressing, bathing):: 0 1 2 3 4 5 6 7 8 9 10 0 = No pain; 10 = Worst pain possible	________ (10 - # circled) (Score 0 if not applicable)
Pain with strenuous activities (reaching, lifting, pushing, pulling, throwing): 0 1 2 3 4 5 6 7 8 9 10 0 = No pain; 10 = Worst pain possible	________ (10 - # circled) (Score 0 if not applicable)
Pain score:	= ____/30
How satisfied are you with the current level of function of your shoulder? 0 1 2 3 4 5 6 7 8 9 10 0 = Very satisfied; 10 = Not satisfied	________ (10 - # circled)

GENESYS
THERAPY SERVICES

Name: ______________________

DOB: ______________________

The Penn Shoulder Score: Function Subscale

	Please circle the number that best describes the level of difficulty you might have performing each activity	No difficulty	Some difficulty	Much difficulty	Can't do at all	Did not do before injury
1.	Reach the small of your back to tuck in your shirt with your hand	3	2	1	0	X
2.	Wash the middle of your back/hook bra	3	2	1	0	X
3.	Perform necessary toileting activities	3	2	1	0	X
4.	Wash the back of opposite shoulder	3	2	1	0	X
5.	Comb hair	3	2	1	0	X
6.	Place hand behind head with elbow held straight out to the side	3	2	1	0	X
7.	Dress self (including put on coat and pull shirt off overhead)	3	2	1	0	X
8.	Sleep on affected side	3	2	1	0	X
9.	Open a door with affected arm	3	2	1	0	X
10.	Carry a bag of groceries with affected arm	3	2	1	0	X
11.	Carry a briefcase/small suitcase with affected arm	3	2	1	0	X
12.	Place a soup can (1-2 lb) on a shelf at shoulder level without bending elbow	3	2	1	0	X
13.	Place a one gallon container (8-10 lb) on a shelf at shoulder level without bending elbow	3	2	1	0	X
14.	Reach a shelf above your head without bending your elbow	3	2	1	0	X
15.	Place a soup can (1-2 lb) on a shelf overhead without bending your elbow	3	2	1	0	X
16.	Place a one gallon container (8-10 lb) on a shelf overhead without bending your elbow	3	2	1	0	X
17.	Perform usual sport/hobby	3	2	1	0	X
18.	Perform household chores (cleaning, laundry, cooking)	3	2	1	0	X
19.	Throw overhand/swim/overhead racquet sports (circle all that apply to you)	3	2	1	0	X
20.	Work full-time at your regular job	3	2	1	0	X

SCORING
Total of columns = _____ (a)

Number of Xs x 3 = _____(b), 60 - _____(b) = _____c)   (if no Xs are circled, function score = total of columns)

Function Score = _____(a) ÷ _____(c) = _____ × 60 _____/60

Appendix 7

Borg's Scale of perceived exertion

Exertion	**RPE**
no exertion at all	6
extremely light	7
	8
very light	9
	10
light	11
	12
somewhat hard	13
	14
hard (heavy)	15
	16
very hard	17
	18
extremely hard	19
maximal exertion	20

Numerical pain scale

0	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
No Pain		Mild			Moderate		Severe			Worst Possible Pain

Appendix 8

Kinematic Calculations

Formations of the anatomical joint coordinate systems of individual segments:

SCAPULA

The following points will be digitized:

1) Angulus Inferior (inferior angle of the scapula) (AI)
2) Angulus Acromialis (acromion process) (AA)
3) Trigonum Spinae (root of the spine)(TS)

Following the ISB recommendations on definitions of JCS, the scapular coordinate system can be established in the following way –

1) Establish the origin at AA.
2) Draw the line connecting TS and AA, pointing towards AA .This is done by subtracting the coordinates of TS from AA (AA- TS). It forms the Z axis.
3) Form the intermediate vector (I / M) using the coordinates of AA and AI, pointing towards AI by subtracting the coordinates of AA from AI (AI-AA).
4) These two vectors form a plane .Calculate a vector perpendicular to this plane pointing forwards by using the cross product as Z x I / M. This forms the X axis pointing forwards.
5) Calculate the Y axis as a common perpendicular to both X and Z axes, pointing upwards by calculating Z x X.

TRUNK

The points needed to build a reference frame for the trunk can not be palpated directly; hence four points are used to build the frame.

Anatomical landmarks:

1) Spine of C 7 vertebra(C7)
2) Spine of T 7 vertebra(T8)
3) Deepest point of suprasternal notch(SN)
4) Most caudal point of xiphoid process(XP)

Coordinate system

Origin (O): Its coincident with the deepest point of suprasternal notch.

Y axis: join the midpoint of the line joining C7 and SN with the midpoint of the line joining T8 and XP, pointing upwards.

Intermediate axis(I/M): Join the points of SN and C7, pointing backwards.

Z axis: Join the line perpendicular to the plane formed by C7, SN and the midpoint of T8 and XP, pointing to the right, i.e. take a cross product of the Intermediate axis and the Y axis. (Z=Y x I/M).

X axis: Take the cross product of X and Z, pointing forwards. (X=Z x Y).

HUMERUS

Anatomical landmarks:

1) Center of rotation for the Humeral head(GH)
2) Lateral Epicondyle(LE)
3) Medial Epicondyle(ME)

Coordinate system

Origin: It coincides with GH.

Y axis: Join the midpoint of the line joining LE and ME with the GH pointing upwards.

Intermediate axis (I/M): The line joining ME and LE pointing to the right.

X axis: It is the common perpendicular to the Y and Intermediate axes. Calculate the cross product of Y and Intermediate vector, pointing forwards(X = Y x I/M).

Z axis: It is the common perpendicular to the X and Y axes. Calculate the cross product of X axis and Y axis, pointing to the right. (Z=X x Y).

B Euler angle estimations from direction cosine matrix

The information obtained for the position and orientation of the segment is in the form of a transformation matrix. It consists of the location of the origin of the coordinate system and a rotation matrix. The rotation matrix consists of direction cosines which define the cosine of the angular value between each axis of a local coordinate system and each axis of the global coordinate system..

The following mathematical equations are used to calculate euler/cardan angles (α β γ) from the rotation matrix.

The rotation matrix for the YX'Z"' (α β γ) sequence used for scapular motion analysis is

$$\begin{pmatrix} \cos\alpha\cos\beta & \cos\alpha\sin\beta\sin\gamma-\sin\alpha\cos\gamma & \cos\alpha\sin\beta\cos\gamma+\sin\alpha\sin \\ \sin\alpha\cos\beta & \sin\alpha\sin\beta\sin\gamma+\cos\alpha\cos\gamma & \sin\alpha\sin\beta\cos\gamma-\cos\alpha\sin\gamma \\ -\sin\beta & \cos\beta\sin\gamma & \cos\beta\cos\gamma \end{pmatrix}$$

The angles are calculated as follows where r denotes an element and the subscript defines its position in the row and column of the matrix. Example, r_{21} defines the first element of the second row.

$\alpha = \text{arctan2}\ (r_{21} / r_{11})$

$\beta = \text{arctan2}\ (-r_{31/}\ [r_{32}^2 + r_{33}^2]^{1/2}\ )$

$\gamma = \text{arctan2}\ (r_{32\,/}\ r_{33}\ )$

The rotation matrix for the YX'Y" sequence used for estimation of humerothoracic motion is:

$$\begin{pmatrix} \cos\alpha\cos\beta\cos\gamma-\sin\alpha\sin\gamma & -\cos\alpha\cos\beta\sin\gamma-\sin\alpha\cos\gamma & \cos\alpha\sin\beta \\ \sin\alpha\cos\beta\cos\gamma+\cos\alpha\sin\gamma & -\sin\alpha\cos\beta\sin\gamma+\cos\alpha\cos\gamma & \sin\alpha\sin\beta \\ -\sin\beta\cos\gamma & \sin\beta\sin\gamma & \cos\beta \end{pmatrix}$$

The angles are extracted as follows:

$\alpha = \text{arctan2}\ (r_{23} / r_{13}\ )$

$\beta = \text{arctann2}\ ([r_{13}^{\ 2} + r_{23}^{\ 2}\ ]^{1/2} / r_{33}\ )$

$\gamma = \text{arctan2}\ (-r_{32}\ / r_{31}\ )$

The rotation matrix for the XZ'Y" sequence used for estimation of glenohuemral motion is:

$$\begin{pmatrix} \sin\alpha\sin\beta\sin\gamma + \cos\alpha\cos\gamma & \sin\alpha\sin\beta\cos\gamma - \cos\alpha\sin\gamma & \sin\alpha\cos\beta \\ \cos\beta\sin\gamma & \cos\beta\cos\gamma & -\sin\beta \\ \cos\alpha\sin\beta\sin\gamma - \sin\alpha\cos\gamma & \cos\alpha\sin\beta\cos\gamma + \sin\alpha\sin\gamma & \cos\alpha\cos\beta \end{pmatrix}$$

The angles are extracted as follows:

$\alpha = \arctan2\,(r_{13} / r_{33})$

$\beta = \arctan2\,(-r_{23} / [r_{13}{}^2 + r_{33}{}^2]^{1/2})$

$\gamma = \arctan2\,(r_{21} / r_{22})$

Appendix 9

Weight estimation according to BMI and arm length percent of total height

Arm length as percent of total height	BMI (less than 22)	BMI (22 to 25)	BMI (25 to 28)
Less than 45%	3 Kgs	3 Kgs	4 Kgs
45%-50%	3 Kgs	3 Kgs	3 Kgs
More than 50%	2 Kgs	3 Kgs	3 Kgs

Appendix 10

MATLAB custom code for calculation of muscle latency and deactivation (for upper trapezius only)

```
clear;
FN= input('file name: ');
importfile1(FN);%reading input file
A(:,:)=data(:,:);

marker=floor(A(:,13));%light
hum_elev=floor(A(:,6));%humerus elevation
hum_elev1=zeros;

for i=1:length(A(:,1));
    if marker(i) >3
        marker1(i)=0;
    elseif marker(i)==0
        marker1(i)=1;
    end
    if hum_elev(i)==-120
        hum_elev1(i)=1;
    end
end

index=find(marker1,1,'first');%finds the frame when light turns on
frame_ecc=find(hum_elev1,1,'last');%finds frame when humeral angle is
120 when lowering

%samp_freq=2500;
%%writing the filters;
[b,a] = butter(7,50/1250,'low');%coeffecients

EMG_UT(:,:)=abs(A(:,14));%rectify
EMG_UTsm(:,:) = filtfilt(b,a,EMG_UT);%filter

base_row=(index-125);%50 msec

M1_UT=mean(EMG_UTsm(base_row:index));%mean of baseline
SD_UT=std(EMG_UTsm(base_row:index));%sd of baseline

N=length(A(:,1));

i=index;
j=i+62;%25 msec window width
count=1;
while (1)
    Mean_UT=mean(EMG_UTsm(i:j));
    if Mean_UT>=(M1_UT+3*(SD_UT))
        lat_UT=i;
        if lat_UT>index+375;%%375 frames =150millisec response time
        break;
        end
    end
```

```
        i=i+1;
        j=i+62;
        count=count+1;
        if (i<N & j>N)%%for the last window being less than 50 frames
            disp 'latency UT can not be calculated';
            break;
        end;
    end;

    %UT switch off time
    i=frame_ecc;
    j=i+249;%100 msec window width
    count=1;
    cnt = 0;
    while (1)
        Mean_UT=mean(EMG_UTsm(i:j));
        if Mean_UT<=(M1_UT+3*(SD_UT));
            flg = 1;
        else
            flg = 0;
        end
        if flg == 1
            cnt = cnt+1;
            if cnt == 2000
                SO_UT = i-2000 + 1;
                break;
            end
        elseif cnt > 0 & flg == 0
            cnt = 0;
        end
        i=i+1;
        j=i+249;
        count=count+1;
        if (i<N & j>N)%%for the last window being less than 50 frames
            disp 'switch off UT can not be calculated';
            SO_UT=1;
            break;
        end;
    end;
    %%making plots
    x1 = 1:N;
    y3_index=zeros(N,1);
    y3_index(index,1)=1;%light
    y3=y3_index;

    figure(1);
    lat_UT_plot=zeros(N,1);
    lat_UT_plot(lat_UT,1)=1;%Muscle activation
    lat_UT_plot(SO_UT,1)=1;%muscle swutch off

    y1 = EMG_UTsm;
    y2=lat_UT_plot;
    plot(x1,y1,x1,y2,x1,y3)
    title('UT latency plot')
```

Appendix 11

Calculation of Intraclass Correlation Coefficients (ICC)

Model 3

ICC (3, 1) = (BMS-WMS) / (BMS + (k-1)*WMS))

Where, BMS is the variance between subjects; WMS is the within subject variance or trial to trial variance and k is the number of trials.

Calculation of the Standard Error of Measurement (SEM)

SEM = square root of within subject variance from a one-way repeated measures ANOVA with subjects as the factor (WMS) or,

SEM = square root (sum of $(X_{ij} - X_{jmean})^2 / n$)

Where, X_{ij} is the individual trial reading for subjects j; X_{jmean} is the mean of the values across trials for each subject and n is the total number of subjects.

Appendix 12

Correlations charts

Table A Correlation coefficients between muscle latency and NPR and RPE scores.

Variable	**Condition**	**Healthy**		**Impingement**	
		NPR	RPE	NPR	RPE
Upper Trapezius (Absolute latency)	Unloaded	-0.02	-0.13	-0.21	-0.07
	Loaded	-0.09	0.11	-0.28	-0.26
	ARM	0.24	0.19	-0.32	-0.41
Lower Trapezius (Absolute latency)	Unloaded	0.12	0.16	-0.04	0.00
	Loaded	-0.04	-0.08	-0.32	-0.39
	ARM	-0.07	0.09	-0.06	-0.34
Serratus Anterior (Absolute latency)	Unloaded	-0.06	0.14	0.12	0.28
	Loaded	0.02	-0.15	-0.08	-0.10
	ARM	0.04	0.30	-0.26	-0.24
Anterior Deltoid (Absolute latency)	Unloaded	-0.06	-0.07	-0.15	0.15
	Loaded	0.01	-0.01	-0.23	-0.30
	ARM	0.08	0.15	-0.44	-0.36
Upper Trapezius (Relative latency)	Unloaded	0.05	-0.06	-0.17	-0.27
	Loaded	-0.04	0.10	-0.23	-0.07
	ARM	0.30	0.18	0.25	-0.05
Lower Trapezius (Relative latency)	Unloaded	0.21	0.27	0.21	-0.25
	Loaded	-0.08	-0.18	-0.18	-0.25
	ARM	-0.09	-0.04	0.42	-0.08
Serratus Anterior (Relative latency)	Unloaded	-0.02	0.28	0.44	0.22
	Loaded	-0.03	-0.31	0.15	0.29
	ARM	0.06	0.28	0.23	0.15

Where,
ARM = after repetitive motion condition
NPR = numerical pain score
RPE = Borg's score of perceived exertion

Table B Correlation coefficients between muscle latency with average velocity of arm elevation

Variable	Condition	Healthy	Impingement
Upper Trapezius (Absolute latency)	Unloaded	-0.07	-0.26
	Loaded	-0.11	-0.13
	ARM	-0.28	-0.24
Lower Trapezius (Absolute latency)	Unloaded	-0.27	-0.23
	Loaded	-0.23	-0.20
	ARM	-0.30	-0.13
Serratus Anterior (Absolute latency)	Unloaded	-0.21	-0.21
	Loaded	-0.22	-0.09
	ARM	-0.39	-0.11
Anterior Deltoid (Absolute latency)	Unloaded	0.00	-0.31
	Loaded	-0.23	-0.17
	ARM	-0.25	-0.15
Upper Trapezius (Relative latency)	Unloaded	-0.09	-0.07
	Loaded	0.05	0.00
	ARM	-0.14	-0.16
Lower Trapezius (Relative latency)	Unloaded	-0.32	0.06
	Loaded	-0.10	-0.06
	ARM	-0.15	-0.02
Serratus Anterior (Relative latency)	Unloaded	-0.31	0.04
	Loaded	-0.03	0.05
	ARM	-0.29	0.02

Table C Correlation coefficients between muscle deactivation and average velocity of arm lowering

Variable	Condition	Healthy	Impingement
Upper Trapezius	Unloaded	-0.11	-0.03
	Loaded	-0.04	-0.13
	ARM	0.14	-0.21
Lower Trapezius	Unloaded	-0.11	-0.10
	Loaded	0.04	-0.03
	ARM	0.17	-0.19
Serratus Anterior	Unloaded	0.30	0.19
	Loaded	0.20	0.22
	ARM	0.15	0.22
Anterior Deltoid	Unloaded	0.09	-0.18
	Loaded	0.19	0.18
	ARM	0.19	0.12

Where ARM = after repetitive motion condition

Table D Correlation coefficients for muscle latency and deactivation with the demographic variables

Variable	**Age**		**Height (cms)**		**Weight (kgs)**		**BMI (Kg/m^2)**	
	Healthy	Imp	Health	Imp	Healthy	Imp	Healthy	Imp
Upper Trapezius (Absolute latency)	0.25	-0.40	0.02	-0.18	0.07	-0.20	0.06	-0.16
Lower Trapezius (Absolute latency)	-0.06	-0.27	-0.01	-0.18	-0.04	-0.20	-0.06	-0.12
Serratus Anterior (Absolute latency)	0.39	-0.25	0.46	0.00	0.60	0.03	0.30	0.04
Anterior Deltoid (Absolute latency)	-0.07	-0.26	0.02	0.03	-0.01	-0.08	-0.01	-0.19
Upper Trapezius (Relative latency)	0.42	-0.35	0.04	-0.36	0.13	-0.27	0.09	-0.06
Lower Trapezius (Relative latency)	0.03	-0.16	-0.02	-0.42	-0.03	-0.29	-0.07	0.02
Serratus Anterior (Relative latency)	0.37	-0.10	0.48	-0.11	0.69	0.10	0.36	0.31
Upper Trapezius (Deactivation)	-0.14	0.23	0.36	0.33	0.26	0.35	-0.02	0.23
Lower Trapezius (Deactivation)	0.09	0.00	-0.34	0.42	0.10	0.35	0.38	0.09
Serratus Anterior (Deactivation)	-0.40	0.07	-0.20	-0.17	-0.46	-0.23	-0.42	-0.21
Anterior Deltoid (Deactivation)	-0.23	0.06	-0.19	0.25	-0.07	0.24	0.12	0.17

Where,
Imp = subjects with impingement
BMI = body mass index

Table E Correlation coefficients between kinematic variables and average velocity for elevation and lowering phases

Group	Phase	Condition	Scapular IRER	Scapular UR	Scapular tilt	GH elevation	GH plane	GH axial rotation
Healthy	Elevation	Unloaded	-0.12 to -0.36	-0.09 to -0.17	0 to 0.13	0.04 to 0.32	0.03 to 0.27	0.02 to 0.14
		Loaded	-0.03 to -0.35	-0.01 to -0.44	0 to 0.11	-0.04 to 0.56	0.16 to 0.4	-0.07 to 0.02
		ARM	-0.41 to 0.01	-0.14 to -0.34	-0.14 to 0.13	-0.04 to 0.43	0.31 to 0.46	0.01 to 0.23
	Lowering	Unloaded	-0.12 to -0.01	0.06 to 0.18	0.07 to 0.16	-0.07 to 0.07	0.01 to 0.21	-0.06 to 0.09
		Loaded	-0.32 to -0.03	-0.26 to 0.16	0.06 to 0.20	-0.17 to 0.35	0.21 to 0.29	-0.12 to -0.06
		ARM	-0.30 to 0.01	-0.19 to -0.08	-0.09 to -0.06	-0.11 to 0.28	0.30 to 0.41	-0.05 to 0.06
Imp	Elevation	Unloaded	-0.18 to -0.08	0.10 to 0.19	-0.22 to -0.11	-0.31 to -0.12	-0.08 to 0.09	0.22 to 0.42
		Loaded	-0.16 to -0.10	0.05 to 0.17	-0.21 to -0.12	-0.21 to 0.13	0 to 0.16	0.18 to 0.31
		ARM	-0.07 to -0.02	0.09 to 0.27	-0.04 to 0.07	-0.30 to -0.13	-0.23 to 0.01	0.32 to 0.40
	Lowering	Unloaded	-0.16 to -0.10	0.05 to 0.17	-0.21 to -0.12	-0.21 to -0.13	0 to 0.16	0.18 to 0.31
		Loaded	0.05 to 0.16	0.07 to 0.27	-0.10 to 0.05	-0.29 to -0.20	-0.22 to 0.10	0.19 to 0.32
		ARM	-0.08 to -0.04	0.20 to 0.26	-0.01 to 0.12	-0.31 to -0.17	-0.14 to 0.08	0.28 to 0.43

Where,
Imp = subjects with impingement group
Scap IRER = Scapular internal/external rotation
Scap UR = Scapular upward rotation
GH = glenohumeral

Table F Correlation coefficients between kinematic variables and demographic variables

Variable	Group	Scapular IRER	Scapular UR	Scapular tilt	GH elevation	GH plane	GH axial rotation
Age (years)	Healthy	0.01 to 0.21	0.16 to 0.34	0.10 to 0.20	-0.33 to 0.10	-0.27 to -0.10	-0.21 to 0.08
	Imp	-0.35 to -0.29	-0.22 to 0.04	0.00 to 0.13	0.10 to 0.23	-0.09 to 0.06	-0.29 to -0.16
Height (cms)	Healthy	-0.09 to -0.07	0.02 to 0.06	0.18 to 0.47	0.11 to 0.27	0.00 to 0.17	-0.17 to 0.03
	Imp	0.07 to 0.21	0.00 to 0.20	0.08 to 0.20	-0.19 to 0	-0.49 to -0.22	-0.22 to 0.12
Weight (kgs)	Healthy	0.13 to0.34	0.01 to 0.07	-0.12 to 0.21	-0.12 to 0.14	-0.19 to -0.08	-0.50 to -0.20
	Imp	0.21 to 0.33	-0.22 to -0.06	-0.05 to 0.09	-0.08 to 0.21	-0.55 to -0.32	-0.31 to 0.03
BMI (kg/m$_2$)	Healthy	0.17 to 0.44	-0.05 to -0.03	-0.31 to -0.14	-0.28 to 0.00	-0.27 to -0.10	-0.51 to -0.34
	Imp	0.27 to 0.33	-0.41 to -0.38	-0.29 to -0.15	0.01 to 0.37	-0.34 to -0.24	-0.28 to -0.11

Where,
Imp = subjects with impingement group
Scap IRER = Scapular internal/external rotation
Scap UR = Scapular upward rotation
GH = glenohumeral
BMI = body mass index

Table G Correlation coefficients between kinematic variables and humerothoracic plane of elevation at 90°

	Kinematic Variable	**30°**	**60°**	**90°**	**120°**
Healthy	Scapular internal/external rotation	-0.08	0.04	0.15	0.18
	Scapular upward rotation	-0.25	0.02	0.08	-0.09
	Scapular tilt	0.09	0.13	0.16	0.05
	Glenohumeral angle of elevation	0.46	0.30	0.03	0.09
	Glenohumeral plane of elevation	0.44	0.59	0.65	0.55
	Glenohumeral axial rotation	0.13	0.03	-0.11	0.08
Impingement	Scapular internal/external rotation	0.25	0.26	0.25	0.23
	Scapular upward rotation	-0.05	-0.07	-0.09	-0.10
	Scapular tilt	0.01	0.02	0.03	0.04
	Glenohumeral angle of elevation	0.08	0.07	0.03	0.00
	Glenohumeral plane of elevation	0.50	0.53	0.53	0.52
	Glenohumeral axial rotation	0.12	0.10	0.07	0.05

Appendix 13

ANCOVA results for Scapular internal/external rotation and tilt

Dependent Variable	**Factor**	**DF**	**F-ratio**	**P value**
Scapular Internal/External Rotation (Lowering phase)	Group	1/46	0.10	0.750
	Condition	2/92	10.98	<0.001
	Angle of elevation	3/138	5.88	<0.001
	Group x Condition	2/92	1.09	0.342
	Group x Angle of elevation	3/138	0.83	0.480
	Condition x Angle of elevation	6/272	1.44	0.198
	Group x Condition x Angle of elevation	6/272	0.54	0.779
	Pectoralis Minor (Degrees)	2/272	5.96	<0.001
Scapular tilt (Elevation phase)	Group	1/43	0.02	0.887
	Condition	2/86	2.96	0.057
	Angle of elevation	3/128	1.47	0.227
	Group x Condition	2/86	0.10	0.901
	Group x Angle of elevation	3/128	0.02	0.997
	Condition x Angle of elevation	6/255	4.50	<0.001
	Group x Condition x Angle of elevation	6/255	0.46	0.840
	Pectoralis Minor (Degrees)	4/255	3.66	0.006

Appendix 14

Surface electrode validation for serratus anterior.

Cross correlation results

Subject	**Cross-correlation coefficients**	
	Latissimus dorsi with surface serratus anterior signal	Latissimus dorsi with fine wire serratus anterior signal
1	0.81	0.82
2	0.83	0.80
3	0.71	0.72
4	0.79	0.79
Average	0.79	0.78

Muscle activity during the elevation phase

Condition	**Motion increment**	**Latissimus Dorsi (n=5)**	**Serratus Anterior (Surface) (n=5)**
Unloaded condition	1	1.31 %	6.22 %
	2	2.73 %	13.43 %
	3	3.49 %	24.27 %
Loaded condition	1	1.77 %	10.25 %
	2	3.57 %	20.54 %
	3	5.95 %	36.96 %

Where 1, 2 and 3 are 30% motion increments for the elevation phase.

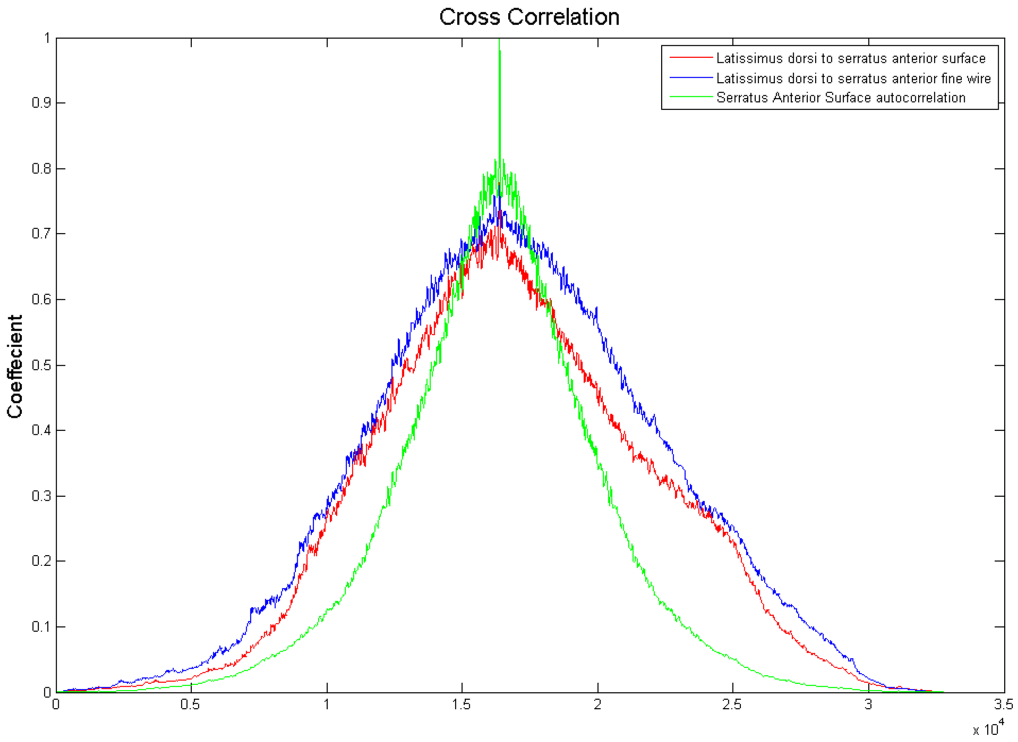


Figure A: Representative plot of a subject for cross correlation coefficients between the signals of latissimus dorsi, serratus anterior (surface electrode), serratus anterior (fine wire electrode) and autocorrelation of serratus anterior surface electrode.

Appendix 15

Logistic regression results: Group prediction by kinematic variables at 30 degrees of humerothoracic elevation during the unloaded condition

Parameter	DF	Estimate	Wald's Chi-square	P value	Odds-ratio	95% Walds CI
Intercept	1	0.75	0.00	0.958		
Scapular IRER	1	-0.10	2.19	0.139	0.91	0.80-1.03
Scapular UR	1	0.03	0.01	0.941	1.03	0.46-2.29
Scapular tilt	1	-0.19	0.79	0.374	0.83	0.55-1.25
GH plane of elevation	1	-0.22	1.82	0.177	0.80	0.58-1.11
GH angle of elevation	1	-0.03	0.01	0.934	0.97	0.45-2.09
GH axial rotation	1	-0.01	0.07	0.796	0.99	0.94-1.05
Normalized Pectoralis Minor length	1	0.52	0.76	0.382	1.68	0.52-5.38

Logistic regression results: Group prediction by kinematic variables at 120 degrees of humerothoracic elevation during the unloaded condition

Parameter	DF	Estimate	Wald's Chi-square	P value	Odds-ratio	95% Walds CI
Intercept	1	28.41	1.54	0.214		
Scapular IRER	1	-0.02	0.11	0.743	0.98	0.89-1.08
Scapular UR	1	0.30	2.89	0.130	1.35	0.92-1.99
Scapular tilt	1	-0.12	2.08	0.150	0.89	0.76-1.04
GH plane of elevation	1	-0.01	0.01	0.921	0.99	0.88-1.12
GH angle of elevation	1	0.24	1.64	0.201	1.28	0.88-1.85
GH axial rotation	1	0.14	0.31	0.578	1.01	0.97-1.07
Normalized Pectoralis Minor length	1	0.54	1.10	0.294	1.72	0.63-4.70

Where, CI = confidence intervals

Appendix 16 a

Values of scapular kinematic variables across the sub groups based on type of impingement

Condition	**Phase**	**Angle**	**Scapular IRER**				**Scapular Upward Rotation**				**Scapular Tilt**			
			H	Int	Sub	Ambi	H	Int	Sub	Ambi	H	Int	Sub	Ambi
Unloaded	Elevation	30°	33.1	34.6	30.1	31.7	-5.8	-2.6	-4.3	-5.4	-9.9	-10.1	-9.0	-10.4
		60°	36.0	36.9	33.1	34.9	-14.7	-11.0	-12.6	-15.0	-7.1	-6.9	-6.7	-7.0
		90°	37.5	38.1	35.0	37.6	-25.7	-20.9	-21.9	-25.1	-4.7	-2.7	-4.1	-5.6
		120°	34.7	35.7	33.6	36.6	-35.3	-30.5	-30.9	-34.0	0.9	4.1	0.4	-2.2
	Lowering	30°	35.1	35.5	31.6	33.0	-9.1	-5.4	-4.6	-5.1	-9.0	-8.0	-9.6	-8.6
		60°	36.4	37.1	33.7	35.3	-19.8	-15.3	-14.6	-16.2	-5.9	-4.3	-7.5	-5.4
		90°	35.5	36.5	33.4	35.8	-31.1	-25.1	-23.8	-27.1	-2.3	0.6	-3.5	-3.7
		120°	31.3	32.8	30.2	33.7	-37.9	-32.6	-31.8	-35.1	2.5	6.7	3.5	-0.7
Loaded	Elevation	30°	35.7	39.9	33.4	34.6	-5.4	-1.6	-5.2	-3.9	-9.4	-9.8	-10.4	-11.1
		60°	38.0	42.6	35.9	37.8	-16.6	-11.1	-15.1	-15.1	-6.2	-5.8	-7.4	-6.8
		90°	38.2	42.7	36.9	38.7	-28.2	-23.6	-26.0	-27.1	-3.7	-1.8	-5.4	-4.9
		120°	34.0	38.0	35.2	35.6	-36.6	-34.3	-34.9	-36.2	1.0	4.0	-1.8	-1.7
	Lowering	30°	37.0	41.8	35.1	34.9	-5.3	-4.2	-2.9	-1.4	-10.4	-8.7	-11.3	-11.3
		60°	38.5	44.0	36.4	37.5	-17.9	-14.3	-16.0	-15.0	-6.0	-4.1	-7.1	-6.1
		90°	37.5	42.8	35.7	36.6	-29.5	-25.1	-27.3	-27.3	-2.5	0.9	-2.9	-3.1
		120°	32.9	37.4	32.2	33.9	-36.6	-34.0	-36.0	-36.2	0.02	0.05	0.02	-.06
After repetitive Motion	Elevation	30°	36.5	40.0	36.6	34.1	-5.1	-2.0	-8.9	-4.5	-10.5	-11.9	-8.6	-11.7
		60°	38.4	42.6	39.3	37.0	-17.3	-12.8	-19.1	-15.7	-8.0	-8.4	-7.3	-8.2
		90°	37.9	42.4	41.3	38.0	-29.7	-25.7	-29.9	-27.4	-5.7	-5.4	-7.6	-6.6
		120°	33.4	37.3	39.4	35.3	-38.0	-35.4	-39.3	-35.1	-1.2	0.4	-5.5	-4.1
	Lowering	30°	36.5	40.4	37.8	33.7	-5.7	-3.4	-8.2	-0.8	-11.3	-11.2	-10.7	-12.4
		60°	38.6	42.6	39.7	36.2	-18.9	-14.3	-20.8	-14.8	-7.2	-7.1	-8.0	-7.5
		90°	37.6	41.9	39.3	36.6	-31.1	-25.1	-31.1	-27.0	-4.2	-2.6	-5.2	-5.4
		120°	33.0	37.3	35.7	35.0	-38.1	-33.7	-39.3	-34.7	0.3	2.2	-0.9	-3.2

Where H=healthy subjects (n = 25); Int = internal impingement only sub group (n = 7); Sub = subacromial impingement only sub group (n = 8) and Ambi = ambiguous impingement sub group (n = 9)

Appendix 16 b Values of scapular kinematic variables across the sub groups based on level of involvement

Condition	**Phase**	**Angle**	**Scapular IRER**		**Scapular Upward Rotation**		**Scapular Tilt**	
			Low (n = 15)	High (n = 9)	Low (n = 15)	High (n = 9)	Low (n = 15)	High (n = 9)
Unloaded	Elevation	30°	31.6	32.8	-3.6	-5.4	-9.3	-10.9
		60°	34.5	35.5	-11.9	-14.9	-6.5	-7.4
		90°	36.6	37.4	-21.7	-24.7	-3.7	-5.2
		120°	35.7	34.6	-30.4	-34.5	1.1	-0.5
	Lowering	30°	32.4	34.7	-4.3	-6.2	-8.0	-10.0
		60°	34.7	36.3	-14.4	-17.2	-5.2	-6.7
		90°	34.9	35.8	-24.1	-27.8	-1.6	-3.6
		120°	32.3	32.1	-32.2	-35.0	4.1	0.7
Loaded	Elevation	30°	35.8	35.7	-2.5	-5.6	-9.7	-11.8
		60°	38.7	38.4	-12.5	-16.4	-6.2	-7.6
		90°	39.8	38.3	-24.4	-28.0	-3.7	-4.9
		120°	37.4	34.1	-34.1	-37.1	0.0	-0.2
	Lowering	30°	36.4	37.8	-1.9	-4.0	-9.5	-12.4
		60°	39.3	38.5	-13.6	-17.8	-5.0	-7.4
		90°	38.9	36.9	-25.2	-29.1	-0.8	-3.6
		120°	35.4	32.7	-34.6	-37.0	3.1	0.8
After repetitive Motion	Elevation	30°	37.8	34.7	-4.7	-6.2	-10.5	-11.2
		60°	40.8	37.2	-15.3	-17.0	-8.0	-7.9
		90°	42.2	37.4	-27.0	-29.0	-7.0	-5.9
		120°	39.9	32.9	-35.4	-38.6	-4.3	-1.6
	Lowering	30°	37.6	36.0	-3.9	-4.2	-10.6	-13.0
		60°	40.6	37.0	-15.7	-18.2	-7.2	-8.1
		90°	41.1	35.8	-26.4	-30.1	-4.3	-4.8
		120°	38.5	31.5	-34.4	-38.5	-0.9	-0.8