Towards Restoring Scapular Position and Motion Through Mapping Muscle Excitation of the Trapezius

by:

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Abstract

Context: Scapular dyskinesis (SD) is associated with increased risk of shoulder pain and pathology, however its etiology remains unclear. Alterations in muscle excitation of the three regions of the trapezius are believed to contribute to altered scapular kinematics, yet research regarding trapezius excitation in individuals with SD is limited. Therefore, the influence of the trapezius on SD is not fully understood.
Objective: To determine if differences in the mean location of spatial distribution of excitation ($BARY_Y$) exist within each region of the trapezius during shoulder elevation (FE) and lowering (FL) in those with (DYS) and without (CON) scapular dyskinesis.
Methods: Fifty-six healthy right-handed participants were assessed using the Scapular Dyskinesis Test (SDT): 28 had normal scapular motion (CON: 15 Female, 27±9 years) and 28 had dyskinesis (DYS: 22 Female, 24±7 years). Participants completed five repetitions of weighted shoulder flexion while high-density surface electromyography (HDsEMG) of the upper (UT), middle (MT), and lower (LT) trapezius was collected. Kinematics were collected to extract $BARY_Y$ within 30-degree ranges (30°-60°, 60°-90°, 90°-120°) of glenohumeral flexion. A two-way (group*angle) between-within (BW) analysis of variance (ANOVA) was conducted for each grid at each angle to determine the effect of group on $BARY_Y$.
Results: A significant interaction was found for LT_FE (p=.025, η_p^2=.079). $BARY_Y$ was more cranial in DYS, with differences of 4.6% (CON=35.2±6.6%, DYS=30.6±7.9%, p=.022) for 30°-60°, 5.0% (CON=35.1±6.7%, DYS=30.1±8.2%, p=.015) for 60°-90°, and 5.8% (CON=35.7±7.3%, DYS=29.9±8.4%, p=.008) for 90°-120°. A significant interaction was found for LT_FL, (p=.041, η_p^2=.073). $BARY_Y$ was more cranial in DYS at 120°-90° and 90°-60°, with differences of 5.9% (CON=36.3±6.9%, DYS=30.4±8.5%, p=.006) and 4.5% (CON=36.1±6.2%, DYS=31.6±7.9%, p=.021), respectively. No significant interactions were found for UT or MT during FE or FL.
Conclusions: Differences in spatial distribution of excitation exist within the LT during both phases of shoulder flexion between individuals with SD and healthy controls. As the magnitude of the differences was small, it is unclear if this differing neuromuscular strategy contributes to alterations in scapular kinematics. No differences in excitation were observed during either phase in the UT or MT, indicating the neuromuscular strategies utilized for each group did not differ.

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List of Abbreviations

AC – acromioclavicular joint

AMC – acromion marker cluster

$BARY_Y$ – mean barycentre y-coordinate

CON – control group

DYS – dyskinesis group

EMG – electromyography

FE – shoulder flexion (elevation phase)

FL – shoulder flexion (lowering phase)

GH – glenohumeral joint

HDsEMG – high-density surface electromyography

IZ – innervation zone

LT – lower trapezius

MT – middle trapezius

MU – motor unit

RMS – Root Mean Square

PI – Principal Investigator

SA – serratus anterior

SC – sternoclavicular joint

SD – scapular dyskinesis

SDT – scapular dyskinesis test

ST – scapulothoracic joint

UT – upper trapezius

1. Introduction

Musculoskeletal disorders are a financial burden on the Canadian healthcare system, with direct and indirect expenses exceeding $25 billion annually[1]. The shoulder is one of the most commonly reported areas of musculoskeletal pain in the general population, with an overall prevalence of 21%[2] and a lifetime prevalence of up to 67%[3]. Shoulder pain and pathology can be debilitating[4] and are typically associated with occupational or athletic tasks that involve frequent use of the arm at, or above, shoulder level[5].

The scapula plays a pivotal role in normal shoulder function, providing a stable base for the highly mobile humerus at the glenohumeral joint and attachment sites for the muscles acting on the shoulder joint[6]. Alterations to the position or motion of the scapula, known as scapular dyskinesis (SD)[6,7], are highly prevalent in both symptomatic and healthy individuals[8,9]. Despite this, SD has been widely associated with an increased risk of shoulder pain[5,10–13], impaired shoulder function[14–16], as well as numerous shoulder pathologies[7,12,17–30]. For example, decreased scapular posterior tilt, external rotation, and elevation may reduce the subacromial space during arm elevation and lead to shoulder impingement[7,23–28], one of the most commonly reported causes of shoulder pain[31].

While several mechanisms have been proposed to contribute to SD[32], the full etiology remains unclear. Alterations in scapulothoracic muscle excitation are believed to contribute to abnormal scapular kinematics due to their dynamic influence on the scapula[25,33–36]. One of the most important muscles for scapular motion and stabilization is the trapezius[34,37–41], a broad, fan-shaped muscle overlying the scapula. Due to its differing fibre orientation the trapezius is sub-divided into three distinct regions: the upper trapezius (UT), middle trapezius (MT), and lower trapezius (LT). Each region produces different actions on the scapula[42,43], which help to maintain normal scapular position and motion. Increased excitation of the UT and decreased excitation of the MT or LT has been demonstrated in individuals with SD during dynamic movement[44–47], leading authors to believe the resulting muscle imbalances may contribute to altered scapular kinematics. Previous studies measuring muscle excitation in the three regions of the trapezius in individuals with SD have used bi-polar EMG[44–53], which provides limited information on the neuromuscular excitation of a muscle or muscle region. To overcome this, high-density surface electromyography (HDsEMG) was developed[54] to provide global information regarding the

neuromuscular excitation of a muscle or muscle region by mapping its distribution over a grid of multiple surface electrodes. This myoelectric map is a representation of the spatial distribution of a muscle[55].

Several studies have investigated the spatial distribution of excitation of the UT[56–66], with shifts in mean location of distribution (i.e. barycentre coordinate) found during various tasks and conditions. These changes in the spatial distribution of excitation in the UT are examples of motor variability, the body's ability to maintain task performance through the selection of various motor options[67–69]. Motor variability may be achieved through the selective excitation of fibres with different orientations found within the UT[42,43], as sub-regions of the UT are capable of providing different actions on the scapula[42,63,70]. Shifts in the spatial distribution of excitation may also be a result of the recruitment of additional MUs[71–73] or the derecruitment and/or substitution of MUs during a sustained contraction[57,62,74–76]. Further, shifts may be influenced by muscle fibre diameter[77], proportion of fibre/MU type[64,78], and fibre/MU distribution[77,78], which vary across the UT. For the MT and LT, changes in spatial distribution have been demonstrated during various shoulder movements[79], though research in these regions is limited. Muscle fibre orientation appears to be relatively homogenous in the MT while fibre orientation of the LT shares similar heterogeneity to the UT[42], indicating more motor variability may be expected in the LT. Other factors that could impact spatial distribution, such as MU and fibre characteristics, have yet to be explored in either region.

While the MT and LT appear to be critical to the normal movement of the scapula[25,34,39,80–82], the results of previous investigations using HDsEMG[79,83–85] as well as bi-polar EMG[44–53] do not provide sufficient information to understand the neuromuscular strategies utilized by each region. Selective excitation of different muscle fibres within a muscle sub-region (i.e. motor variability) may influence scapular kinematics; however, no investigations to-date have explored the spatial distribution of excitation within any region of the trapezius in individuals with SD. Further, while it is known that excitation within the UT can be redistributed to meet differing task demands, it is not known whether the MT and LT possess the same motor variability to achieve task performance.

Thus, the following work aims to address this knowledge gap by examining the spatial distribution of excitation of the UT, MT, and LT in individuals with SD during a dynamic shoulder task. The document will begin with a comprehensive review of the current literature

regarding the function of the scapula, anatomy and actions of the trapezius, the etiology and assessment of SD, as well an overview of electromyography studies of the trapezius and in individuals with SD. Next, methodological details will be presented followed by statistical analysis, results, discussion, limitations and future directions, and conclusions. The document will conclude with references followed by appendices.

2. Review of Literature

2.1 Overview of Anatomy of the Shoulder Complex

The shoulder complex is the connection of the appendicular skeleton, or upper extremity, to the axial skeleton, or thorax. It consists of bony structures as well as various soft tissue structures that act to provide stability to the joint and improve function. The four bones which make up the shoulder joint are the sternum, clavicle, scapula, and humerus. These bones articulate to create four joints: the sternoclavicular (SC) joint, the acromioclavicular (AC) joint, the glenohumeral (GH) joint and the scapulothoracic (ST) joint. These joints work in a coordinated manner to provide the upper limb with a vast range of motion. Due to its high mobility, the shoulder complex relies on both dynamic (e.g. muscular) and static (e.g. ligamentous) soft tissue structures to provide stability.

2.2 Function of the Scapula

The scapula lies on the posterolateral thorax overlying ribs 2-7 and is defined by its flat, triangular shape and its various bony landmarks which act as attachment sites for muscles and articulations to form joints[43] ***(Figure 1).*** The only bony connection between the uniquely shaped scapula and the axial skeleton is via the AC joint, with additional stability provided by the ST joint. The ST joint is not considered a “true” joint as it does not have the characteristics of a fibrous, cartilaginous, or synovial joint. Instead, the scapula is a floating bone nearly completely stabilized by its surrounding musculature[86] to maintain congruency with the thoracic wall.

The scapula serves several roles to facilitate the stability and mobility of the upper extremity to allow for optimal shoulder function. One of the primary roles is maintaining a coordinated relationship with the humerus to allow for maximal range of motion of the upper extremity. By maintaining this congruency, the scapula creates a stable surface for the articulating humerus at the GH joint[6]. This proper alignment allows for the most efficient position of the intrinsic shoulder muscles (i.e. rotator cuff) to provide dynamic compression to the glenoid socket[6] creating maximal concavity and compression of the GH joint[33]. Appropriate

orientation of the scapula also optimizes the length-tension relationship of the ST muscles, maximizing excitation and force production[24,37,87–89].

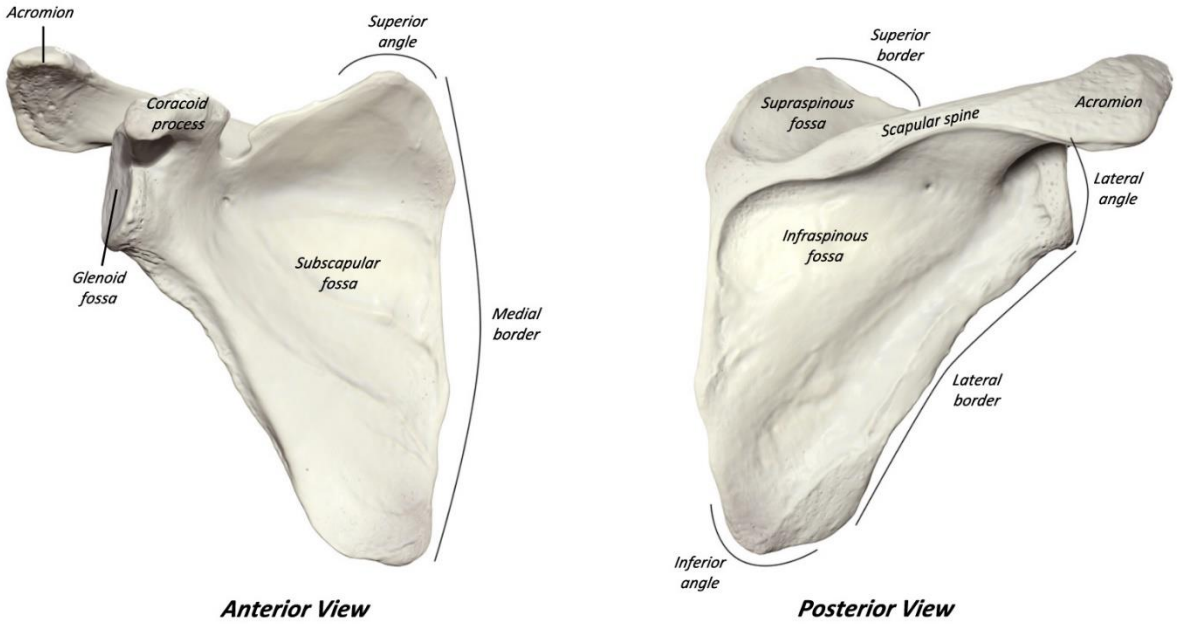


Figure 1. Anatomical Landmarks of the Scapula
Adapted from https://www.anatomystandard.com/Upper_Limb/Shoulder_Girdle/Scapula.html

2.2.1 Muscles Influencing the Scapula

The scapula serves as an attachment site for the muscles influencing dynamic movement and stability of the upper extremity. There are 17 muscles which attach to the scapula, however only six of these muscles are responsible for the majority of dynamic stabilization of the ST joint[86,90]: 1) the levator scapula; 2 & 3) the rhomboid major and minor; 4) the pectoralis minor; 5) the serratus anterior (SA); and 6) the trapezius. Along with the SA, the trapezius is widely considered one of the most important muscles responsible for scapular motion and stabilization[34,37–41]. Indeed, the trapezius and SA act synergistically to produce many actions of the scapula. Generally speaking, the trapezius is seen as the primary stabilizer of the scapula, while the SA is seen as the primary mover, though considerable overlap exists[91]. Due to the differing fibre orientation of the trapezius, the upper, middle and lower portions of the muscle create distinct anatomical as well as functional subdivisions[92] that produce different actions on the scapula[42,43] ***(Figure 2)***:

1) Upper trapezius (UT) – The descending fibres of the UT originate on the superior nuchal line and ligamentum nuchae and attach on the lateral third of the clavicle. These fibres act to elevate, upwardly rotate and externally rotate the scapula during shoulder elevation[42,91,93]. As the UT does not have a direct attachment to the scapula, it accomplishes these motions through its action on the clavicle. When the UT is contracted, an elevation and retraction force is applied to the clavicle at the SC joint[42,93,94], causing indirect motion to occur at the scapula.
2) Middle trapezius (MT) – The transverse fibres of the MT extend from the spinous processes of C7 and T1 with the C7 fibres attaching on the acromion and T1 fibres on the spine of scapula. These fibres act to stabilize and retract the scapula throughout shoulder elevation[42,91,95]. While not considered an agonist or antagonist of the UT, proper excitation of the MT is imperative to normal scapular position and motion[25,34,80].
3) Lower trapezius (LT) – The ascending fibres of the LT originate on the spinous processes of T2 to T12 and insert on the deltoid tubercle of the scapular spine, located on the medial most aspect of the scapular spine. These fibres act to depress and externally rotate the scapula during shoulder elevation[42,91]. Additionally, the LT works in tandem with the SA and to a lesser extent the UT to upwardly rotate the scapula[81,82], particularly in the early and middle stages of shoulder elevation[93]. Excitation of the LT is essential to maintain the normal path of the instant centre, or axis, through which scapular upward rotation occurs during shoulder elevation. This is due to the straight line of pull the LT has on the deltoid tubercle of the scapular spine as the scapula upwardly rotates, providing a strong mechanical advantage[81]. During the lowering phase from an elevated position, the LT operates to maintain the scapula against the thorax[39]. The LT may also have some influence on scapular posterior tilting, though the primary muscle for this action is the SA[93,94].

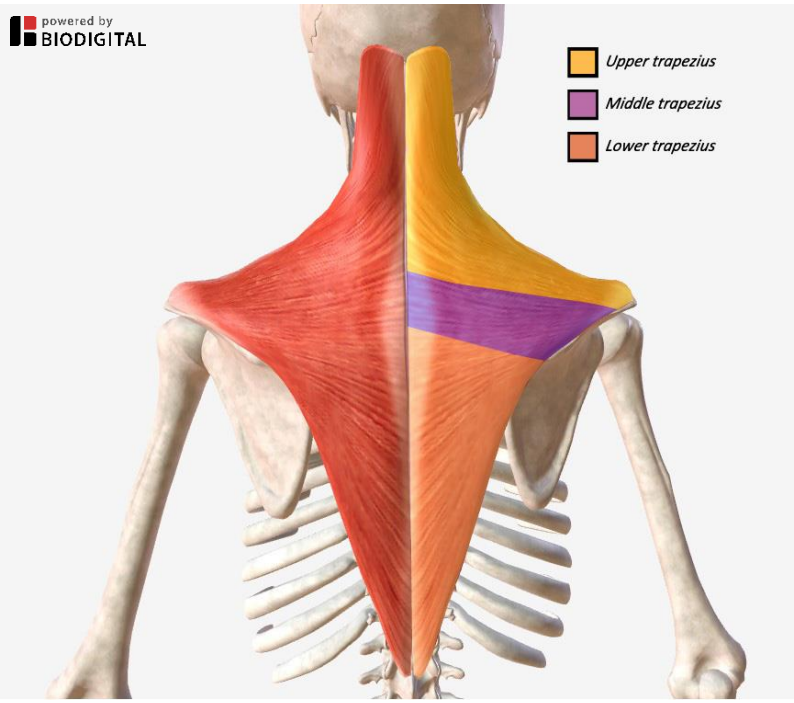


Figure 2. Anatomical Subdivisions of the Trapezius Muscle (shown on right side) Adapted from https://human.biodigital.com/edit?id=production/maleAdult/male_complete_anatomy_13

Scapular positioning and motion primarily relies on the synergistic actions of the trapezius and SA[96]. This is known as a force couple, or the coordinated action of two or more muscles or muscle subdivisions to create or control motion at a joint[6]. For example, the UT, LT and SA work as a force couple to upwardly rotate the scapula[89]. As the arm begins to elevate, the moment arms of UT, LT and SA are long, allowing for effective action on the scapula. The SA acts to move the scapula laterally around the thoracic wall, while the LT resists this motion to stabilize the axis of rotation. The UT then exerts an upward rotation moment about the axis[42]. As elevation increases, the UT moment arm decreases while the LT and SA moment arms remain long, continuing scapular upward rotation[97]. The balanced activity within force couples are essential to allow for smooth, coordinated motion of the scapula and are considered more important to normal shoulder function than the absolute strength of individual muscles[6].

All three regions of the trapezius are innervated by the spinal accessory nerve (cranial nerve XI). Complete trapezius palsy significantly impairs scapular position and motion, particularly during upper extremity motions[86], demonstrating the importance of the trapezius in normal shoulder function. This presents as a shoulder “droop” with excessive downward rotation

and lateral translation of the scapula or scapular "winging" where excessive internal rotation occurs as the other scapular stabilizers are unable to effectively compensate[98].

2.3 Scapular Kinematics

The ability to assess and identify abnormalities in scapular position and motion can only occur once normal position and motion of the scapula are understood[99]. At rest (i.e. anatomical position), the scapula is typically positioned with the superior angle of the scapula and lateral border of the acromion level with the T2/T3 spinous processes, the root of the scapular spine level with the T3/T4 spinous processes, and the inferior angle lying somewhere between the T7-T10 spinous processes[89]. The scapula is oriented 30° from the frontal plane[100] with the glenoid facing inferiorly[101] and the medial border parallel to the thoracic spine[102]. The medial border and inferior angle are congruent with the contours of the thoracic wall[103], and the orientation of the scapula on the dominant side may be marginally inferolateral relative to the nondominant side[102].

Movement of the scapula on the thoracic wall relative to the movement of the humerus during shoulder elevation is known as scapulohumeral rhythm and coordinated through the balanced actions of the surrounding musculature[45,91]. During the first 30° of humeral flexion and 60° of humeral abduction, scapular motion is minimal and has significant variation from person to person[104] as a stable base for the humeral head is established[105]. This is known as the setting phase, as the scapula seeks to find the optimal position for function[104]. As the humerus moves beyond 30° of flexion, motion becomes more uniform with a 2:1 ratio of GH to ST movement[104,106]. For example, as the upper extremity moves from 0° of elevation to 180° at the GH joint, the humerus accounts for 120° of total motion while upward rotation of the scapula accounts for 60°. However, while upward rotation is the most apparent motion, smaller accessory motions occur to refine the position of the scapula during arm elevation[91]. Early studies of scapulohumeral rhythm[104,107] observed only two-dimensional kinematics and therefore the complex three-dimensional nature of the scapula was not fully understood, impacting the accuracy of early scapular kinematic measurements[108,109].

Three dimensional motion of the scapula occurs along three axes: upward/downward rotation along a horizontal axis perpendicular to the plane of the scapula; internal/external rotation along the vertical axis; and anterior/posterior tilt along a horizontal axis parallel to the plane of the scapula[33] ***(Figure 3)***. The scapula is also capable of two translational motions along the thoracic wall: elevation/depression, which translates the scapula superiorly and inferiorly,

respectively, as well as medial/lateral translation where movement occurs in a horizontal direction[39,108] . The coupling of external rotation, upward rotation, posterior tilting and medial translation is known as scapular retraction, while the coupling of internal rotation, downward rotation, anterior tilt and lateral translation is known as scapular protraction[33].

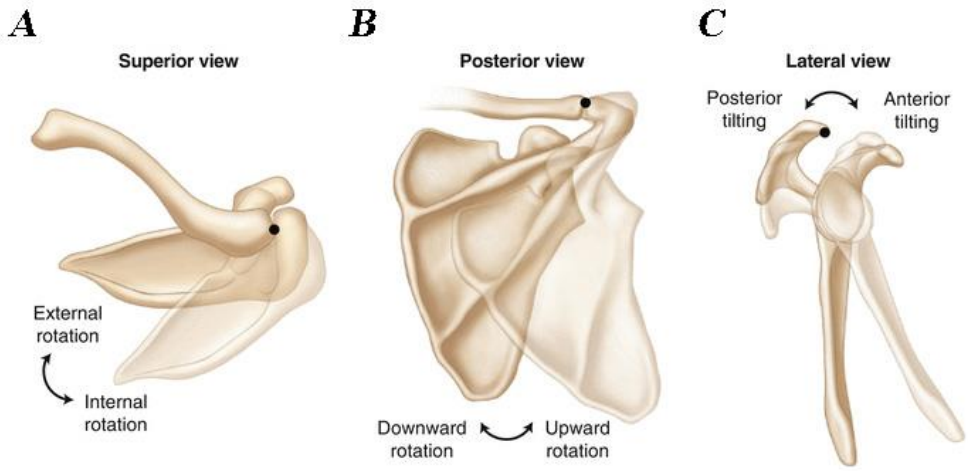


Figure 3. Motions of the Scapula Along its Three Axes[110]
(A) Scapular external and internal rotation occurring along a vertical (i.e. longitudinal) axis in the transverse plane. (B) Scapular upward and downward rotation occurring along a sagittal (i.e. anterior-posterior) axis in the frontal plane. (C) Scapular anterior and posterior tilting occurring along a frontal (i.e. left-right) axis in the sagittal plane.

As the humerus elevates during flexion and abduction, the scapula typically upwardly rotates, posteriorly tilts, and externally rotates[25,28,34,99,108,111]. Upward rotation is the most significant motion occurring during arm elevation and is assisted by a posterior tilt to translate the acromion posteriorly and away from the elevating humeral head, thereby increasing the subacromial space. This movement reduces the likelihood of contact between the humeral head and acromion or other soft tissue structures, preventing potential subacromial impingement and other shoulder pathology[25,28,91,99,112]. Additionally, upward rotation during elevation is critical in lending mechanical stability to the shoulder joint, maintaining congruency between the glenoid and humeral head[113]. While external rotation of the scapula is most common, some authors have noted internal rotation during elevation[114–116]. However, the functional significance of the discrepancy between internal and external rotation is unclear with some authors speculating external rotation is more desirable to reduce the demands on the GH joint during shoulder

elevation[91]. Internal and external rotation of the scapula may also be dependent on the plane of elevation or the specific range in which the arm is currently elevating[99,117].

2.4 Scapular Dyskinesis

Scapular dyskinesis is a collective term referring to any alteration in normal scapular motion and/or position[6,7]. The term 'dyskinesis' can be separated into two parts: 'dys' (alteration of) and 'kinesis' (motion). While dyskinesis is often used interchangeably with 'dyskinesia', the latter term describes a loss of voluntary motion resulting from neurological factors (e.g. tardive dyskinesia) while dyskinesis is a broader term encompassing varying causative factors[35]. Indeed, the term 'scapular dyskinesis' does not suggest etiology or infer a pattern correlated to specific shoulder pathology[118]. Another common term is 'scapular winging', describing a prominence of the medial border of the scapula (i.e. decreased external rotation)[119]. However, this term does not indicate whether the abnormality is static, dynamic or both, nor does it encompass all possible variations of altered scapular kinematics[39]. Changes in scapular orientation during motion may include a reduced posterior tilt, excessive elevation, and a decreased external rotation which results in posterior displacement of the medial or inferior border of the scapula from the thoracic wall[7,25,28,116] ***(Figure 5)***. Absence of a smooth, coordinated rhythm is another common characteristic of SD, most often presenting as excessive scapular elevation during shoulder elevation or a rapid downward rotation during shoulder lowering[10,13].

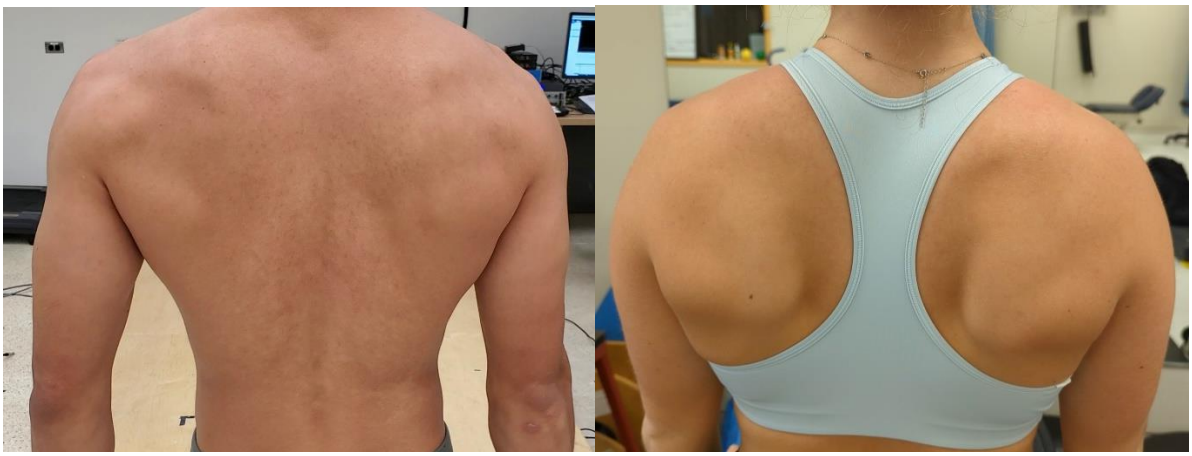


Figure 4. Example of Scapular Dyskinesis
(A) Normal scapular positioning. (B) Decreased external rotation and posterior tilting resulting in a medial and inferior border prominence of the scapula bilaterally.

2.4.1 Assessing Scapular Dyskinesis

The goal when assessing for SD is to identify the presence of altered kinematics, determine the potential relationship between these alterations and clinical symptoms, and establish the underlying factors leading to SD[36,39,120]. However, it is important to note that SD is not itself an injury or diagnosis, but a clinical impairment indicating a deviation from optimal scapular kinematics[32,120]. As scapular dyskinesis is a common finding, it can be challenging to determine if these deviations are clinically relevant and contributing to an individual's symptoms. However, in a consensus statement by Kibler et al.[10] it was recommended that scapular kinematics be evaluated during a clinical assessment prior to rehabilitation of patients with shoulder disorders. Clinical determination of the presence of SD can also prove challenging as it relies primarily on visual observation[121]. Additionally, interindividual variability in scapular kinematics is significant[122] and may be impacted by various factors such as the plane and degree of shoulder elevation[27,99,123], as well as the current level of muscle fatigue[124].

Several methods have been developed to assess scapular position and motion, including static measures[125], symptom alteration tests[24,87,117], dynamic measures[125–128], and the use of 3-dimensional motion capture systems. However, due to the drawbacks of static measures[129] and symptom alteration tests[130], as well as the resources required for the use of motion capture systems[8], dynamic measures have been recommended in the clinic assessment of SD[131].

2.4.1a Dynamic Measures

The first visual assessment method to be described was by Kibler et al. (2002)[126], with scapular motion categorized into four different types. This 4-type method allowed for a more specific description of scapular kinematics to differentiate between various abnormalities in scapular motion. Different patterns of dyskinesis have also been noted when comparing elevation to lowering phases[13,44,132] or during motion in different planes[123]. However, the reliability of Kibler's method has been thoroughly explored[13,103,127,128] and found to be relatively low, therefore its use in clinical settings was not warranted. As a result, simplified classification methods were introduced[127,128].

The Scapular Dyskinesis Test (SDT) is a visual assessment method developed by McClure et al.[127] involving dynamic motion of the scapula. Unlike Kibler's 4-type method, the SDT does not attempt to distinguish among the pattern(s) of dyskinesis present, as the authors believed patterns were not mutually exclusive. This removed the necessity of an examiner to decide on a

single predominant pattern of dyskinesis when multiple abnormalities may be present[25,126]. Instead, scapular motion was rated as "normal", "subtle abnormality" or "obvious abnormality" based on operational definitions of scapular motion **(Tables 1 – 3 in Experimental Methods)**. Additionally, while Kibler's method accounted for scapular symmetry[126], McClure's method evaluated each scapula independently relative to the thorax. A full description of the SDT can be found in the Experimental Methods section of this paper.

The SDT has been shown to have a moderate to substantial inter-rater reliability while intra-rater reliability was found to be substantial[127,133,134]. Strong concurrent validity for the SDT was also demonstrated among overhead athletes using 3-dimensional kinematic measures[135]. However, several studies have investigated the predictive ability of the SDT for injury risk with mixed results[136–139]. One of these studies reported observable dyskinesis could predict risk of injury[136] while three others reported no predictive ability[137–139], leading some authors to question the clinical value of the SDT[9].

The use of visual observation with dynamic measures is limited as it relies on the subjective interpretation and experience of the examiner[121,126,130]. Accurate assessment of scapular kinematics can prove challenging due to the varying planes of movement in which the irregularly shaped scapula can move[140], as well as movement artifact of the soft tissue overlying the scapular region[10,123,140]. Indeed, Maclean et al.[129] found dynamic measures frequently under-estimated scapular angles when compared to static measures, particularly at higher elevation angles. However, these standardized visual assessment methods are preferred over static measures or symptom alteration tests[10,125,128].

Regardless of their reliability or validity, static or dynamic measures as well as symptom alteration tests do not allow for diagnosis of shoulder pain or pathology. In a systematic review by Wright et al. (2013)[120], the outcomes of various SD tests did not have a significant impact on the post-test probability of shoulder pain or pathology diagnosis. The authors suggested two potential reasons for these findings: 1) SD tests are not valid or reliable enough to diagnose shoulder pain or pathology; and 2) SD is a broad and inadequately defined term encompassing several impairments, limiting the use of a single test to discriminate between those with and without shoulder dysfunction.

2.4.2 Etiology of SD

The prevalence of SD in the literature varies[8,9,141]. In a systematic review by Burn et al.[8], 61% of overhead athletes and 33% of non-overhead athletes were found to have SD based on the results of 12 studies. This higher prevalence in overhead athletes has been proposed to be due to heavy reliance on unilateral upper extremity function[121]. A meta-analysis by Hogan et al. (2020)[9] also focused on SD in athletes found the presence of SD ranged from 22% to 56% based on the results of 7 studies. However, the prevalence increases to 68% to 100% when investigating individuals who have experienced a shoulder injury[7,142]. As the majority of studies related to SD have focused on athletes, making inferences regarding prevalence rates in the general population is difficult. Therefore, future investigations into SD prevalence in this population are needed.

Despite its prevalence in both symptomatic and asymptomatic populations, the precise mechanisms behind altered scapular kinematics are still unclear. When considering the complex kinematics of the scapula in relation to the multi-axial GH joint, as well as the multi-directional influence of the scapulothoracic muscles, it becomes evident that a combination of underlying factors could lead to the presence of SD. Therefore, several etiologies have been suggested in the literature[32]. However, the relationship between scapular dyskinesis and shoulder pain and pathology remains ambiguous[10,35,135]. Longitudinal studies following individuals with SD are difficult, leading SD to be explored primarily using cross-sectional methods. Due to this limitation, it is difficult to discern whether SD represents a cause or effect of pathology[5,6,10,46,90,91,120,136,138,143,144], making inferences regarding its etiology difficult[10,11]. For example, SD may predispose an individual to injury by creating pathomechanics that adversely affect function (i.e. cause), or the pathomechanics may be in response to injury and lead to a further reduction in function (i.e. effect)[35]. Alternatively, SD may be due to avoidance of painful kinematic patterns leading to alterations in scapular motion[145].

The passive positioning and dynamic functional stability of the scapula are known to be controlled by the scapulothoracic musculature[50,81,119,146,147], leading several authors to suggest alterations to these muscles to be a primary cause of SD[25,33–36]. While imbalances in the absolute strength of scapulothoracic muscles have been proposed to contribute to abnormal kinematics[34,90,148], much of the literature considers alterations in neuromuscular control of greater significance to SD etiology. Indeed, alterations to the excitation ratio, timing or recruitment

patterns of scapulothoracic muscles such as the trapezius and serratus anterior have all been proposed to alter scapular kinematics[14,25,34,44–46,48,91,144,146,148].

Extensive research has been conducted regarding electromyographic alterations in individuals with shoulder pathology, with research specifically examining those with SD becoming more prevalent in recent years[44–53]. Several studies found an increased excitation of the UT in those with SD relative to the control group during various dynamic motions, while excitation of the MT, LT, and/or SA were decreased or delayed[44–47]. Other studies found only an increase in UT excitation[50] or a decrease in MT excitation[49] with all other scapulothoracic muscles showing no differences. In contrast, Zakharova-Luneva et al. (2012)[48] found no significant differences in muscle excitation between individuals with SD and healthy controls. The altered ratio of excitation and differences in the timing of excitation found in the majority of these studies are similar to those found in the literature when analyzing the muscle excitation of individuals with shoulder impingement[5,25,45,47,146,149,150].

Unique alterations in muscle excitation are also found for different patterns, or kinematic variations, of SD. When contrasting the muscle excitation of each dyskinesis pattern to healthy controls, those with prominence of the inferior angle of the scapula were found to have decreased LT and SA excitation[44,151], medial border prominence were found to have increased UT excitation and decreased MT and SA excitation[44,151], while those with types where both patterns are combined had increased UT excitation and decreased LT and SA excitation[44]. For those with excessive scapular elevation, increased excitation in the UT has been found[12,14,27,28]. Along with excessive elevation, an overactive UT likely leads to impaired upward rotation[36] and excessive anterior tilt of the scapula[5,94]. Decreased MT excitation may lead to excessive internal rotation of the scapula during arm elevation and reduce medial stabilization[91], while a reduction in scapular upward rotation may be the result of decreased LT excitation[94,148].

Ebaugh et al. (2005)[152] suggested a minimum level of muscle excitation, termed activation threshold, is necessary to produce normal scapulothoracic motion. If this threshold is not reached during motion, scapulothoracic control will be compromised, leading to SD. Muscle excitation during the lowering phase of motion has been shown to be less than muscle excitation during the elevation phase[13,153,154], potentially falling below this activation threshold. Indeed, EMG excitation during lowering was reduced by 61%, 42%, and 39% when compared to the elevation phase for the UT, LT, and SA, respectively[153]. This minimum threshold theory may provide a

potential explanation for the increased prevalence of SD during shoulder lowering[6,44]. Scapulothoracic muscle excitation is also dependent on plane of movement, as differences between flexion and scaption or abduction have been found[25,50,155–157]. For example, UT and MT have shown greater excitation during shoulder abduction than flexion, while LT displayed an inverse effect having greater excitation during flexion[155]. Flexion has also been shown to elicit greater SA excitation than abduction in those with normal scapular kinematics, leading some authors to believe the increased prevalence of scapular winging in flexion to be the result of reduced SA excitation[104,128,156,157].

Inter-individual differences in innervation to the trapezius may play a role in SD, as Shiozaki et al. (2007)[158] discovered significant variability in the branching of the accessory nerve to the three regions of the trapezius muscle. This may result in individuals with greater innervation of the MT or LT having more inherent control over scapular position and motion. Nerve pathology, or palsy, is often cited as a primary source of altered scapular kinematics[39,159,160], yet these mechanisms only account for approximately 5% of all individuals with SD[35]. Lesions of the spinal accessory nerve inhibit the trapezius muscle, leading to lateral translation and excessive protraction of the scapula due to the unopposed actions of the serratus anterior and pectoralis minor. Injury or palsy of the long thoracic nerve leads to dysfunction within the serratus anterior, causing excessive internal rotation, elevation and medial translation of the scapula to occur as the trapezius and pectoralis minor become predominant[32,119].

The inhibitory effect of pain on muscle excitation may also contribute to SD, altering normal muscle excitation patterns[6,36,48,59,61,145,146,153]. Inhibition can reduce the ability of a muscle to produce torque and provide stability, leading to disruptions in scapular function[7,119]. In a study by Falla et al. (2007)[145], a saline solution was injected into the UT to determine the effect of experimental muscle pain on trapezius excitation during a dynamic task. The results found a decrease in muscle excitation in the UT accompanied by an increase in excitation of the LT. The authors suggested this reorganization of synergistic muscle coordination was a compensatory mechanism in response to the inhibitory effects of painful stimuli. Such alterations in muscle excitation are supported by the pain adaptation model[161], which suggests muscle amplitude is affected by pain due to the inhibition of agonist muscles. More recently, Hodges & Tucker (2011)[162] proposed a more in-depth model regarding pain and muscle excitation. The authors suggested that pain leads to a redistribution of excitation within and between muscles leading to

alterations in the kinematics of the associated body segments. This redistribution of excitation is proposed to create short-term benefits by protecting the structure(s) from further pain or injury but may incite long-term mechanical changes that adversely affect function. Shoulder pathologies (e.g. subacromial impingement, GH instability) are often cited as a potential mechanism for SD[6,36,144,163], however it may be the pain associated with these pathologies that underlies the development of altered kinematics opposed to the nature of the pathology itself.

Different alterations in scapular kinematics have been shown with short-term fatigue to the scapulothoracic muscles[40,124,164–167]. Tsai et al. (2003)[164] found decreases in scapular upward rotation, posterior tilting and external rotation while changes in scapular resting position were observed, with similar changes found in several other studies following a fatigue protocol[166,167]. Conversely, Chopp-Hurley et al. (2016)[165] found a significant increase in scapular upward rotation following a fatiguing protocol, though considerable variability existed among participants. Increased upward rotation was also found by Ebaugh et al. (2006)[40] which was accompanied by increased scapular external rotation. However, these studies did not specifically isolate the scapular stabilizers during the fatigue protocol, confounding the results as extrinsic muscles (e.g. deltoid) also demonstrated fatigue[5].

Flexibility deficits in soft tissue may restrict or influence scapular movement, contributing to altered kinematics[168–170]. For example, a shortened pectoralis minor or short head of biceps brachii can produce an anterior tilt and excessive protraction of the scapula due to their attachment at the coracoid[168,171,172]. Aberrant posture (e.g. forward head posture, thoracic kyphosis) have also been shown to impact scapular kinematics by promoting scapular protraction[7,25,88,108,173,174], and may lead to adaptive shortening of postural muscles (i.e. flexibility deficits) or imbalances in muscle strength[88,174]. Specific impairments in flexibility are often associated with specific dyskinesis patterns[47]. Inferior angle prominence may be related to flexibility deficits in the pectoralis minor[168], while medial border prominence has been associated with posterior capsular tightness[169].

2.4.3 SD and Pathology

SD is believed to adversely affect shoulder function[14–16], with several authors suggesting abnormal scapula kinematics increase the risk of shoulder pain[5,10–13]. Further, there is a growing body of evidence associating SD with shoulder pathologies including shoulder impingement[7,23–28], GH instability[7,17,29,30], rotator cuff weakness[175] or pathology[18], adhesive capsulitis[19–22], labral

tears[12] and AC pathology[16]. It has been suggested SD may be the cause of pathology, or may simply exacerbate pre-existing symptoms[33].

A combination of upward rotation, external rotation and posterior tilting of the scapula during shoulder elevation are optimal to minimize the risk of shoulder pathology[10,25,33,39,91,99,108,111,144,176]. Individuals with SD display sub-optimal control and are often limited in one or more of these motions. For example, individuals with subacromial impingement syndrome often present with decreased scapular posterior tilt during arm elevation[23,25,28,34]. This reduces the space between the acromion and humeral head, creating friction on the soft tissue structures passing through the subacromial space. With repetitive movements, this increased friction may lead to inflammation, pain and weakness. Increased protraction and reduced elevation of the scapula may also create decreased subacromial space and rotator cuff compression, leading to subacromial impingement and a decrease in rotator cuff strength[32,37,87]. Further, excessive protraction and reduced elevation may increase the strain on anterior GH ligaments, increasing the risk of internal impingement[32]. Indeed, shoulder impingement is believed to be one of the most common causes of shoulder pain[31].

SD can also affect the length-tension relationship of scapulothoracic muscles, reducing their force producing capabilities and thus, their control of the scapula during upper limb tasks[16,42]. Consequently, the scapula is unable to provide a stable base for the humerus, further disrupting the normal kinematics of the shoulder. Efficient energy and force transfer occurring from the torso to the upper extremity via the kinetic chain is impacted[118], resulting in the subsequent links of the kinetic chain (e.g. GH joint) producing greater force to compensate for the compromised ST joint. With repetitive use of upper extremity, particularly in overhead tasks producing significant force (e.g. tennis player), these overloaded tissues may lead to the development of shoulder injuries[6].

Despite the association between SD and numerous pathologies, several authors have questioned the relationship to shoulder pain[36,50,133,135,138,144] and whether SD increases the risk of pathology[9,136,143,177]. In a systematic review and meta-analysis by Hickey et al. (2018)[11], the authors concluded that SD in asymptomatic athletes increased risk of future shoulder pain by 43%. However, these findings were criticized by several authors[9,178] as static tests for SD were included, contrary to current expert consensus recommending dynamic measures for the clinical assessment of SD[10]. Additionally, the total number of injuries did not meet statistical

recommendation for risk factor identification[179] and the risk range reflected significant uncertainty[178]. In their own meta-analysis, Hogan et al. (2020)[9] found a 7% increased risk of shoulder pathology in those with SD, however the results were not statistically significant. The authors reported normal scapular motion in 54% of athletes, with 21% developing a shoulder injury over the course of one to two seasons. In contrast, 46% of athletes were found to have SD with 25% developing a shoulder injury over the same period. While methodological differences (e.g. sample size, demographic variables, definition of injury, etc.) of the studies included in these meta-analyses may have contributed to the conflicting results, the multi-factorial nature of injuries makes the development of prospective studies on SD and shoulder pathology difficult[9]. Indeed, biological and psychosocial factors as well as their complex interactions must be considered confounding factors of these studies, and results should be interpreted with caution[180].

Many individuals presenting with SD are not experiencing pain or pathology[11,128,134,177,181,182] with some authors reporting a similar incidence of SD in asymptomatic individuals when compared to symptomatic individuals[11,13,128,134,181]. In a systematic review by Wright et. al (2012)[120] it was determined that the presence of SD cannot be used to diagnose the presence or absence of shoulder pain. This has led several authors to suggest SD may not be a pathological clinical presentation, but instead a natural adaptation to an individual's long or short-term demands[8,117,120,134,178,181,183]. For example, Thomas et al. (2009)[184] found scapular position changed over the course of a season in response to the demand of an athlete's sport, potentially allowing for maximized performance[11], while Madsen et al. (2011)[124] found SD increased from 37% of swimmers to 82% after a single-hour of training. Alternatively, SD could be a compensatory strategy to protect against injury or avoid stress on pain-sensitive structures[10,46,143]. To further support this hypothesis, no pattern of kinematic alteration has consistently been found to relate to a specific pathology[5,10,111]. Individuals with shoulder impingement have demonstrated both increased posterior tilting[26,27] and decreased posterior tilting[23,25,28,34], as well as increased upward rotation[27,112] and decreased upward rotation[23,25], while others have found increased internal rotation[7,25] and superior translation[27,28]. These mixed findings have lead some authors to believe SD may be an example of a normal human adaptation that has been pathologized[178]. However, regardless of the relationship between SD and pathology, determining the potential association between changes in spatial distribution of the

trapezius and alterations in kinematics will help to further our understanding regarding scapular neuromuscular control.

2.5 Electromyography

Electromyography (EMG) is a technique used to measure the electrical potentials of muscles during contraction to provide a representation of neuromuscular excitation[185]. EMG provides information such as the intensity of muscle contractions or recruitment strategy of motor units (MUs), furthering our understanding of the coordination of skeletal muscle and regulation of forces exerted across joints[186,187].

2.5.1 Intramuscular vs. Surface EMG

Electrodes used to collect EMG can be broadly categorized into two types: intramuscular and surface. Intramuscular electrodes, composed of either a needle or fine-wires, are inserted directly into a muscle to measure electrical excitation within a specific region[188]. One of the primary advantages of these electrodes is their anatomical specificity, sampling from a small number of muscle fibres and discriminating between individual MUs[189,190]. Electrodes can be inserted into deeper musculature while providing consistent measurements throughout movement[191]. Indeed, several studies investigating neuromuscular excitation of the trapezius have utilized intramuscular electrodes[70,76,147]. However, the invasive nature of these electrodes limits their utility in research, and their small detection zone is often not practical in larger muscles or where global muscle excitation is of interest. A high failure rate has also been noted with fine-wire EMG, with Heuberer et al. (2015)[192] reporting low quality EMG signals in 32% of their recordings compared to <1% using surface EMG.

Surface electrodes adhere to the skin overlying a muscle to provide an indirect measure of neuromuscular excitation (i.e. electrical potential present on skin)[193]. These electrodes are typically composed of silver-silver chloride (Ag-AgCl)[186,189,193] and vary in size and shape. Surface electrodes are commonly used due to their non-invasive nature and low cost[191]. In most cases, two surface electrodes are placed on the skin in a bi-polar configuration[188] parallel to the muscle fibres at a specific interelectrode distance (typically 10-20 mm)[194]. The electrodes are placed between the innervation zone of a MU and tendon of the muscle, with the measurement resulting from the difference between two monopolar electrodes. By taking the differential of two signals, electrical excitation from external sources (e.g. electrical excitation of nearby muscles, ambient noise, etc.) is significantly reduced[186]. Bi-polar surface EMG has been widely

used in the literature, including investigations into the neuromuscular excitation of the trapezius in those with SD[44–53]. However, alignment error is common with bi-polar EMG as it is assumed that both electrodes are overlying the same muscle fibres. This is particularly of concern in muscles that are multi-pennate with varying fibre orientations (e.g. trapezius)[42,43], and during changes in joint position or muscle force[187]. Further, while bi-polar EMG measures neuromuscular excitation over a greater area than intramuscular EMG, the detection zone is still relatively small leading to a sampling of a limited number of MUs. As all muscle fibres belonging to a single MU are of the same type[195] and are located within relatively small regions of the muscle[196], recording a select number of MUs may not be representative of excitation of the entire muscle. Indeed, as the excitation within a muscle or muscle region is known to vary and the fibre distribution be non-uniform, the information provided by bi-polar EMG is limited. This limitation is of particular concern when attempting to understand the global impact of excitation of a muscle or muscle region on normal or abnormal biomechanics[197].

2.5.2 High-Density Surface EMG

Continued advancements in EMG technology have led to the development of electrode grids and arrays with multiple surface electrodes in close proximity, termed high-density surface electromyography (HDsEMG)[54]. This novel technology allows for the acquisition of surface electrical potential at a significantly greater spatial resolution through topographical mapping, providing a more representative depiction of the average neuromuscular excitation of a whole muscle in comparison to bi-polar EMG[55,63,64,197]. Despite the advantages of HDsEMG over bi-polar EMG, both surface techniques are only capable of measuring the neuromuscular excitation of superficial musculature. This limitation provides continued utility for intramuscular EMG when deeper muscles are explored[198].

2.5.2a Spatial Distribution of Excitation

HDsEMG is capable of obtaining global information regarding the neuromuscular excitation of a muscle by mapping the distribution of its signal amplitude[55]. Heightened excitation within an area of the grid corresponds to the recruitment of specific MUs, while areas of reduced excitation correspond to regions of the muscle with lower recruitment rates. This myoelectric map represents the spatial distribution of excitation of a muscle. Determination of the mean location of the distribution of excitation allows for shifts or differences in excitation to be observed and quantified in the space of the electrode grid. This mean location of excitation is

known as the barycentre, often referred to as the centroid or centre of mass[199]. The barycentre is defined using a x-coordinate and y-coordinate with a pre-defined origin (i.e. 0,0) located on the grid[57].

Several studies have examined the spatial distribution of excitation and barycentre location of the UT using HDsEMG during various tasks and conditions[56–66]. In healthy participants, a shift in the cranial direction of the barycentre has been seen during dynamic repetitive tasks or sustained contractions inducing fatigue[57,58,61–65]. This upward shift may be due to the recruitment of additional MUs[71–73] or the derecruitment and substitution of MUs in a discrete portion of the muscle to minimize neuromuscular fatigue[57,62,74–76]. Conversely, participants experiencing pain have shown a relative caudal shift in barycentre location during similar or identical tasks[56,59,60,65]. Increased excitation in the lower fibres of the UT may be considered an inefficient motor strategy by individuals experiencing pain and has been proposed to further contribute to the painful condition[56]. Alternatively, the caudal shift may be a motor strategy designed to protect the region from injury. The cranial fibres of the UT act as an agonist of movement, while the more caudal fibres provide stabilization of the scapula[42,63,70]. Indeed, a caudal shift in barycentre is also seen with increasing force production[63], possibly due to an increased need for scapular stabilization. Therefore, increased MU recruitment of the lower regions of the UT may be a mechanism to avoid undue stress to the tissues.

The changes seen in the spatial organization of the UT during various tasks and conditions are examples of motor variability, the body's ability to select various options for maintaining task performance. Motor variability leads to alterations in kinetics, kinematics, and muscle excitation and is proposed to reduce the incidence of musculoskeletal disorders of the shoulder[67–69]. Indeed, the availability of multiple motor options (i.e. high motor variability) may be a mechanism to prevent tissues from being overloaded during task performance, therefore reducing the risk of pain and pathology[67,200]. For muscle excitation, motor variability includes synergistic muscle actions, recruitment or de-recruitment of MUs within a muscle, as well as changes in individual MU characteristics within a muscle[67,200,201] that occur during dynamic movement and static postures[200]. Evidence of motor variability has been shown in studies involving those with chronic muscle pain, with individuals experiencing pain demonstrating less variation in muscle excitation during a task[202–204]. Further, increased motor variability has been shown to reduce the development of muscle fatigue as the heterogeneity of muscle excitation increases[57,62,201,205].

The presence of motor variability is further supported by the known heterogeneity of muscle fibre/MU type[64,78], fibre/MU distribution[77,78] as well as fibre diameter[77] and orientation[42,43] found in the upper trapezius leading to an uneven spatial distribution with excitation[55,62,63]. This heterogenic spatial distribution of excitation is more evident during submaximal contractions where fewer MUs are recruited compared to maximal contractions as the number of active MUs from the beginning to the end of the task may vary significantly[62]. Indeed, spatial distribution of excitation changes with the level of force[55,63,64,73,199] and length of contraction[65,73], providing additional evidence of motor variability within a muscle. This variable recruitment has been hypothesized to occur due to compartmentalization within regions of the trapezius muscle for specific biomechanical functions[62,206] as selective excitation of sub-regions of a muscle have been previously reported[92,207–209]. The muscle fibres of a MU within a sub-region will exert force in a specific direction based on their orientation and line of action[210], which has been demonstrated by changes in spatial distribution of excitation within a muscle when performing different muscle actions[208,211]. The development of individualized motor strategies may explain the inter-individual variability seen in previous studies regarding patterns of trapezius excitation[212–214].

Despite the growing literature on the spatial distribution of excitation of the UT, literature on the spatial distribution of the LT and MT are limited and have only investigated healthy individuals[79,83–85]. Bohunicky et al. (2021)[83] recently explored the excitatory effects of kinesio tape on the spatial distribution of excitation of the LT, discovering no differences between groups during a repeated elevation task. Afsharipour et al. (2016)[79] investigated muscle excitation of the MT and LT in violin and cello players using spatial maps, finding differences in amplitude within selected regions of the grid depending on the instrument played and movement performed. These differences indicated changes in spatial distribution were found, though changes over the entire spatial map were not examined. The remaining two studies on these muscle regions examined the MT and LT in various scapular positions[84] and during a typing task with biofeedback for postural correction[85]. However, a single 64-electrode array was used to capture excitation of the MT and LT, not allowing the spatial maps of each region to be viewed independently. The limited number of studies on the spatial distribution of excitation of these regions makes it difficult to draw any conclusions or make any inferences on the neuromuscular strategies implemented by the MT or LT, particularly in those with altered kinematics or

shoulder pathology. Indeed, to our knowledge no study has yet to explore the spatial distribution of excitation of the UT, MT, or LT in individuals with SD.

3. Purpose

Scapular dyskinesis is a prevalent clinical finding in both symptomatic and healthy individuals, and has been associated with an increased risk of shoulder pain[5,10–13] and impaired shoulder function[14–16] as well as numerous shoulder pathologies[7,12,17–30]. Several studies have shown increased excitation of the UT and decreased excitation of the MT or LT in individuals with SD[44–47], leading authors to believe that an imbalanced ratio may be contributing to altered scapular kinematics.

Excitation of the three parts of the trapezius in individuals with SD has only been measured using traditional bi-polar EMG[44–53], providing a limited window into the neuromuscular excitation over the whole muscle or muscle region. The development of novel technology (i.e. HDsEMG)[54] has allowed for acquisition of surface electrical potential at a significantly greater spatial resolution to provide a more representative depiction of neuromuscular excitation of a muscle (i.e. spatial distribution)[55]. Shifts in spatial distribution of excitation during various tasks and conditions have been demonstrated in the UT[56–66], which may be the result of differing MU recruitment strategies (i.e. motor variability)[63,70]. These neuromuscular strategies may be influenced by differences in fibre orientation[42,43], diameter[77], proportion[64,78] and distribution[77,78] within each region that may lead to the selective excitation of specific muscle fibres and contribute to the differences seen in the spatial distribution of excitation. Similar motor variability be present in the MT and LT, however limited studies on the MT[79,84,85] and LT[79,83–85] using HDsEMG have been conducted. As each region of the trapezius plays a critical role in scapular function[25,34,39,80–82], further exploring the neuromuscular strategies of these regions may provide a better understanding of how motor variability within the trapezius relates to changes in scapular kinematics. However, no studies to date were found to have investigated the spatial distribution of excitation in any region the trapezius in individuals with SD.

Therefore, determining the modifications in the spatial distribution of excitation in those with SD in each region of the trapezius may contribute to a greater understanding of SD, filling an existing gap in the literature. We hope to gain insight into the physiological mechanisms that control scapular position and motion that can be utilized to better understand the association

between SD and pathology, as well as improve clinical outcomes through the development of appropriate corrective strategies to restore normal scapular kinematics.

4. Objectives

The proposed investigation will aim to answer questions related to the spatial distribution of excitation within the three regions of the trapezius during shoulder elevation and lowering in the sagittal plane in those with scapular dyskinesis (DYS) and those with normal scapular motion (CON):

1) Does the barycentre of excitation of the UT differ between groups throughout shoulder elevation and lowering in the sagittal plane (i.e. flexion) in the y-direction?
2) Does the barycentre of excitation of the MT differ between groups throughout shoulder elevation and lowering in the sagittal plane (i.e. flexion) in the y-direction?
3) Does the barycentre of excitation of the LT differ between groups throughout shoulder elevation and lowering in the sagittal plane (i.e. flexion) in the y-direction?

5. Hypothesis

The following are the corresponding hypotheses for each research question:

1) The Y-coordinate of the UT will be located more caudally in the SD group relative to the CON group.
2) The Y-coordinate of the MT will not differ between groups.
3) The Y-coordinate of the LT will be located more cranially in the SD group relative to the CON group.

The above hypotheses regarding the UT in those with SD is based on the caudal shift in spatial distribution of excitation found in those with muscle pain. Individuals with SD may invoke motor strategies that increase excitation in the caudal stabilizing fibres of the UT while minimizing excitation in the cranial fibres to counteract the alterations in scapular kinematics. For the MT, due to the homogeneity of its fibre direction, no difference in spatial distribution of excitation is expected. For the LT, a cranial shift in SD is hypothesized as the upper fibres of the LT involved in stabilization are selectively excited while the lower fibres that elicit movement are inhibited.

Barycentre coordinate in the y-direction (i.e. cranial-caudal) will be analyzed as shifts in the spatial distribution of excitation related to recruitment of MUs primarily occur in this

plane[62,64,65]. Barycentre coordinate in the x-direction (i.e. medial-lateral) will not be analyzed as differences are not expected. As electrodes in the x-direction run parallel to muscle fibre direction, the distribution should remain relatively unaltered[62,186]. Further, differences in the x-direction are proposed to be a result of positioning or misalignment errors, or due to skin artifact as the muscle fibres move relative to the overlying tissues and tendinous regions may pass under the grid surface[63,66]. As electrode grids were placed on participants by the same research assistant throughout the study, such errors were expected to be minimized.

6. Experimental Methods

6.1 Overview of Methods

To achieve the objectives of the proposed study, a cross-sectional design was used to analyze the spatial distribution of excitation of the UT, MT, and LT during a dynamic overhead shoulder task in two different groups: a scapular dyskinesis group (DYS) and a control group (CON). Eligible participants were independently sorted into groups by two certified athletic therapists using the scapular dyskinesis test (SDT)[126]. Participants were asked to complete a dynamic weighted shoulder task by moving through a full range of motion in the sagittal plane (i.e. flexion): The task consisted of an elevation phase (0°–180°) and a lowering phase (180°–0°) creating two distinct phases: shoulder flexion during elevation (FE); and shoulder flexion during lowering (FL). A shoulder abduction task was also completed following the shoulder flexion task, however this task was not included in our analysis. Due to the volume of data, the current study focused on shoulder flexion as abnormalities in scapular kinematics are more obvious during flexion when compared to abduction[19,127,215]. Therefore, potential differences in barycentre location between DYS and CON would likely be more apparent during this task. HDsEMG of each region of the trapezius was recorded during the task, as well as kinematics of the upper extremity and torso. A flow chart of the study design is presented in ***Figure 5***.

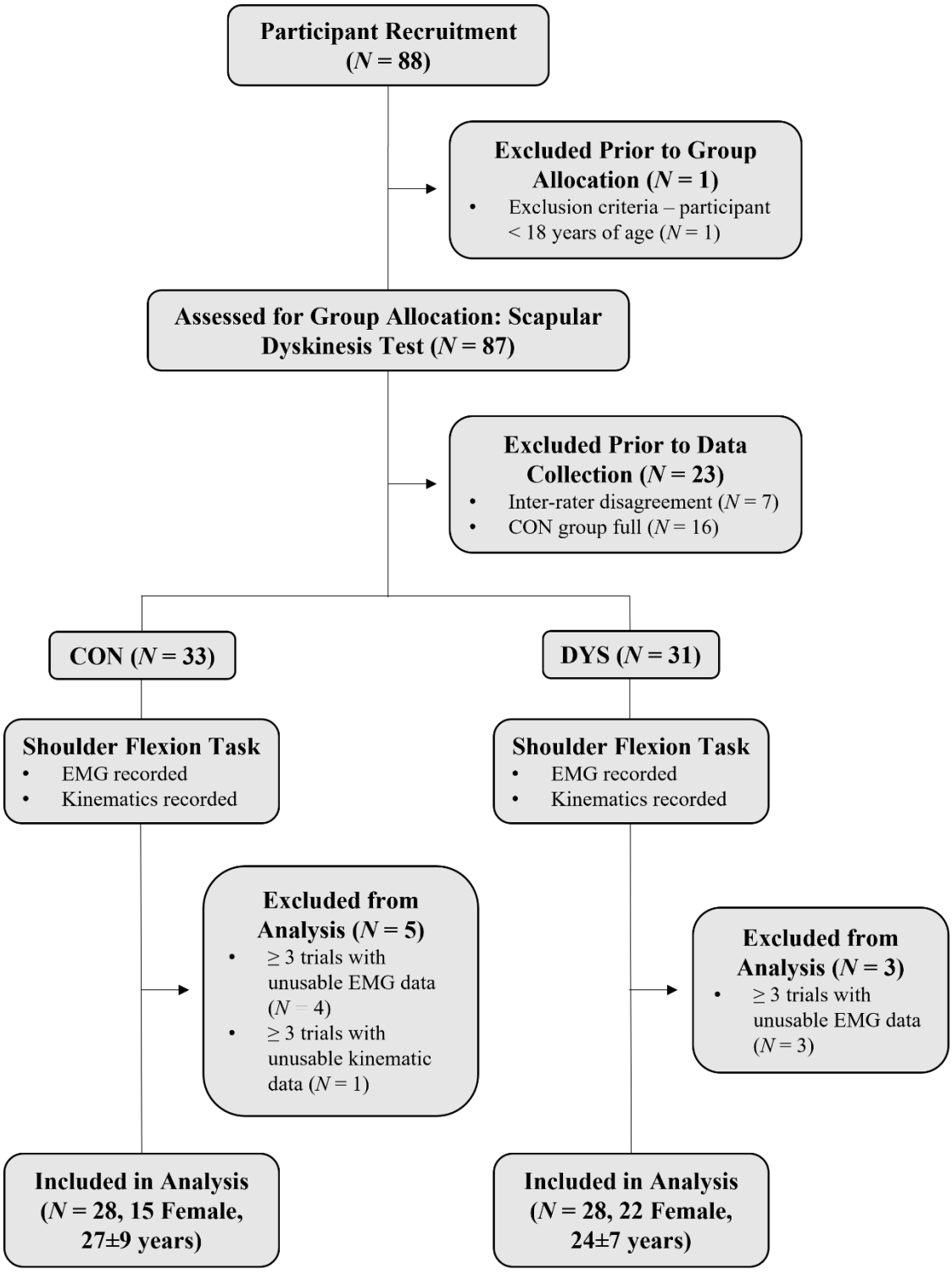


Figure 5. Flow Chart of Study Design

6.2 Participants

A convenience sample of 56 right-hand dominant individuals were analyzed in order to complete the proposed research. Participants were sorted into two equal groups using the SDT: 28 in the control group (CON: 15 Female, 27±9 years) and 28 in the dyskinesis group (DYS: 22 Female, 24±7 years). Potential participants were recruited from the student and faculty population at the University of Manitoba and the surrounding community through posters, social media posts, and word of mouth. Individuals meeting the following criteria were eligible to participate: between the ages of 18 and 60; not currently experiencing pain on the right side in the shoulder, upper back, or neck; and no history of injury or orthopedic disorder to the shoulder, upper back or neck (e.g. rotator cuff tears, whiplash, fractures, etc.) in the past year. Right-handed participants were recruited as differences in scapular motion[176,181] and muscle excitation[156,216] have been found between dominant and non-dominant arms. Individuals with neurological (e.g. Multiple Sclerosis, Parkinson's Disease) or musculoskeletal disorders (e.g. Muscular Dystrophy) were ineligible to participate. Additionally, any individuals allocated to a group (CON, DYS) where recruitment had been completed were excluded from the study. Participant screening took place either over email or in person prior to attending the experimental session to ensure eligibility.

6.3 Experimental Session

Participants attended a single experimental session for the study lasting approximately one hour in duration. At the beginning of the experimental session, informed consent was obtained via an intake form as well as participant age for demographic information.

6.3.1 Scapular Dyskinesis Assessment

Eligible participants were recruited into an experimental group using the SDT as described by McClure and colleagues[127] ***(Figure 6)***. Male participants were asked to remove their shirt and female participants will be asked to wear a shirt that allows visual access to the scapula and shoulder complex, such as a spaghetti strap, halter tank-top or any other tank-top that allows access to the upper-middle back. Modified shirts that allow for visual access to the scapula were available for participants if needed.

Prior to beginning the test, a research assistant demonstrated each task for the participant and asked them to practice several times until the movement was performed correctly and the participant was comfortable with the task. Two movements were performed bilaterally: dynamic

weighted shoulder flexion in the sagittal plane and dynamic weighted shoulder abduction in the frontal plane. Both movements began with the arms at the side of the body with the elbows straight and torso erect. Flexion occurred with the forearm in a neutral position (i.e. mid-way between pronation and supination) with the thumb oriented anteriorly at the start of the movement. For abduction, the participant began in anatomical position (i.e. thumb facing away from midline, or supinated). The participant was instructed to "lead with their thumbs" throughout each movement as they elevate their arms overhead through a full range of motion (i.e. 0°–180°) then lower their arms to return to the start position (i.e. 180°–0°). Each movement occurred at a cadence of 3 seconds per direction (i.e. elevation and lowering phases) continuously, guided by a metronome set to 60 beats per minutes. Once a participant was comfortable with each movement, the SDT began, and the participant was asked to perform 5 repetitions each of flexion and abduction. All repetitions of flexion were completed first, followed by all repetitions of abduction. Throughout all repetitions, participants held a handheld weight determined by body weight: 1.4 kg (3 lb.) for participants <68.1 kg (150 lb.) and 2.3 kg (5 lb.) for participants >68.1 kg[127]. Dumbbells were used for the 1.4 kg weight while kettlebells were used for the 2.3 kg weight, based on equipment availability.

During the SDT, scapular position and motion on the right side was observed and evaluated independently by two certified athletic therapists. Four examiners in total were used: the principal investigator (PI) was an examiner for every participant throughout the study, with the other examiner being one of three individuals based on availability. All examiners were certified athletic therapists whose years certified ranged from 5 to 12 years. Further, examiners were trained in the operational definitions and rating scales used by the SDT prior to the beginning of the study. Examiners stood approximately 2 meters behind participants during their evaluation. For instances when a second examiner could not be present, a video recording of the SDT was sent to the second examiner by the PI to be rated. Examiners did not communicate during the evaluation and were blinded to each other's assessment[217]. Participants' scapular motion on the right side was categorized as "normal", "subtle abnormality", or "obvious abnormality" using McClure and colleagues' [127] operational definitions **(Table 1)** and rating scale **(Table 2)**. Examiner ratings were compared for agreement following the SDT by the PI, and participants were excluded from the study in cases where final ratings differed. Each participant was grouped based on the combined evaluation of flexion and abduction[127] **(Table 3)**.

Those rated as "normal" were allocated to the control group (CON), while those rated as "subtle abnormality" or "obvious abnormality" were allocated to the dyskinesis group (DYS).

Table 1. McClure and colleagues' operational definitions of scapular motion[127].

Normal scapulohumeral rhythm: The scapula is stable with minimal motion during the initial 30° to 60° of humerothoracic elevation, then smoothly and continuously rotates upward during elevation and smoothly and continuously rotates downward during humeral lowering. No evidence of winging is present.
Scapular dyskinesis: Either or both of the following motion abnormalities may be present: *Dysrhythmia:* The scapula demonstrates premature or excessive elevation or protraction, non-smooth or stuttering motion during arm elevation or lowering, or rapid downward rotation during arm lowering. *Winging:* The medial border and/or inferior angle of the scapula are posteriorly displaced away from the posterior thorax.

Table 2. McClure and colleagues' rating scale for scapular motion in flexion and abduction[127].

Normal motion: No evidence of abnormality
Subtle abnormality: Mild or questionable evidence of abnormality, not consistently present
Obvious abnormality: Striking, clearly apparent abnormality, evident on at least 3/5 trials (dysrhythmias or winging of 1 in. [2.54 cm] or greater displacement of scapula from thorax)

Table 3. McClure and colleagues' final rating scale for scapular motion based on combined flexion and abduction test movements[127].

Normal: Both test motions are rated as normal, or 1 motion is rated as normal and the other as having subtle abnormality
Subtle abnormality: Both flexion and abduction are rated as having subtle abnormalities
Obvious abnormality: Either flexion or abduction is rated as having obvious abnormality

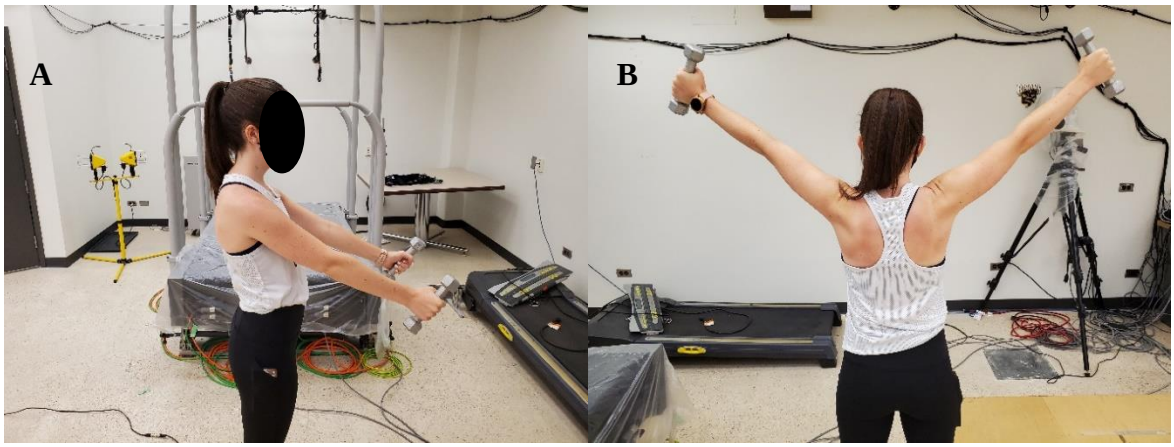


Figure 6. Scapular Dyskinesis Test
Prior to kinematic marker and electrode grid placement, participants completed the scapular dyskinesis test to determine which group they were placed in. Scapular motion was evaluated by two certified athletic therapists during bilateral dynamic shoulder flexion (A) and dynamic shoulder abduction (B) through the elevation phase (0°-180°) and a lowering phase (180°-0°).

6.3.2 Kinematics

Kinematics of the torso and right upper extremity were collected using retroreflective markers (PM-14.0, B & L Engineering, Santa Ana, CA, USA) 14mm in diameter. Retroreflective marker placement was based on the International Society of Biomechanics (ISB) recommendations for anatomical landmarks ***(Figures 7 & 8)***[218]. Anatomic landmarks were identified and marked by the same certified athletic therapist throughout data collection to improve the reliability of retroreflective marker placement. An acromion marker cluster (AMC) made up of three retroreflective markers ***(Figure 9)*** was placed on the acromion at its junction with the scapular spine. The location for the AMC was chosen as it has been found to minimize soft-tissue deformation and reduce error[219]. The AMC was fixed to the participants' skin using a combination of double-sided tape and retention tape (Hypafix, BSN Medical Canada, Laval, Quebec, Canada) and was used to estimate the position of the GH joint for the local coordinate system during kinematic processing. Eighteen total retroreflective markers were placed on the participant: six markers were used only during initial static calibration (i.e. calibration markers), while 12 markers (including AMC) remained on the participant throughout the experimental task (i.e. tracking markers) ***(Figure 7)***. All markers were fixed on participants' skin or clothing using double-sided tape. Markers allowed for the position and orientation of the participants' body segments in space to be quantified via the reflection of infrared light back to the cameras surrounding the collection space.

Kinematic data was collected using an 8-camera motion capture system (model 2.2, Vicon Motion Systems, Los Angeles, CA, USA). Cameras were calibrated to the collection space via a calibration wand before each experimental session, and the origin of the axes of the global coordinate system set. After calibration of the collection space was completed and all retroreflective markers attached, the participant stood in the centre of the collection space facing the researcher.

A static calibration of the participant in anatomical position (i.e. 0° shoulder elevation) was recorded for a 5-second duration. The calibration helps determine the local coordinate system positions and orientations of each segment (i.e. trunk, upper limb) using the calibration and tracking markers. After the calibration trial and prior to the start of the experimental task, the calibration markers were removed. Prior to the start of the experimental task but after electrode grids had been placed, the participant was asked to perform a single repetition of shoulder flexion and abduction through a full range of motion to visually inspect for gaps in marker trajectories (i.e. camera blind spots). If gaps were found, the participant was repositioned in the collection space and asked to perform the task again until no gaps could be visualized.

A BNC cable was connected from the Vicon to the Quattrocento bioelectrical signal amplifier (Quattrocento, 400-channel EMG amplifier, OT Bioelettronica, Turin, Italy) that is responsible for collection of EMG data. Connecting the two systems allowed for the data to be synchronized to create identifiable timestamps during data processing.

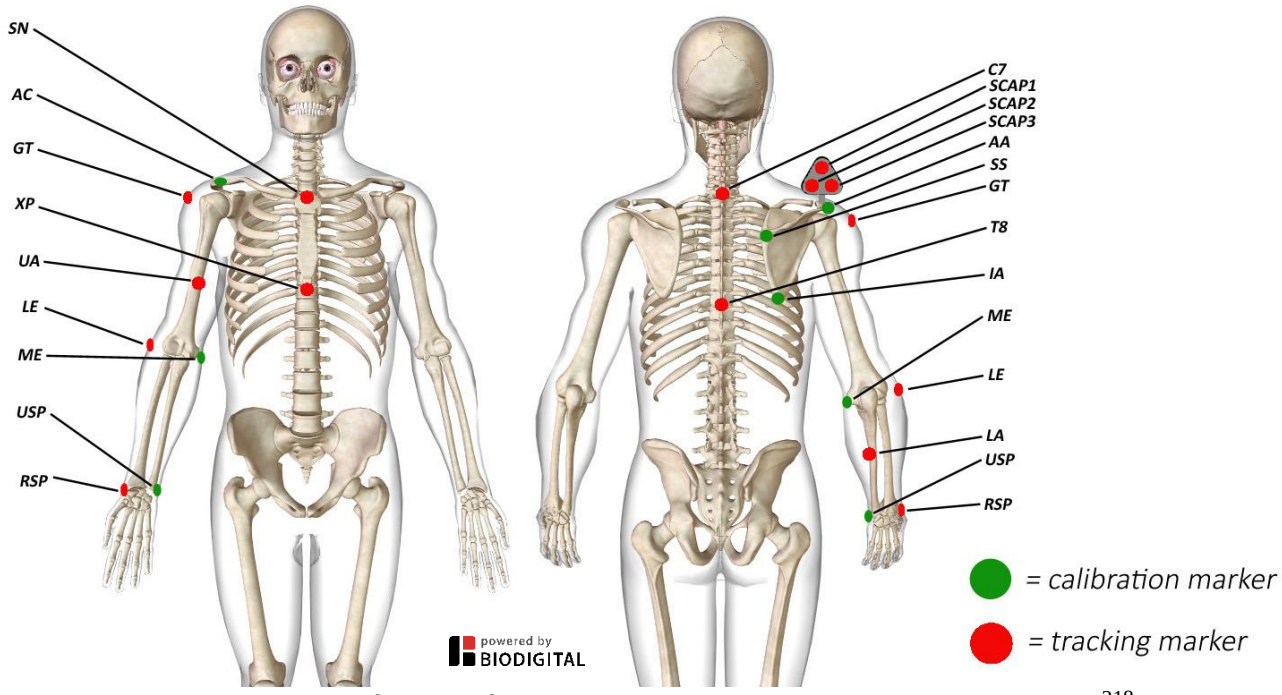


Figure 7. Locations of Retroreflective Kinematic Markers on Anatomic Model[218]
Calibration markers: AA (acromial angle); AC (acromioclavicular joint); IA (inferior angle); ME (medial epicondyle); SS (scapular spine); USP (ulnar styloid process). Tracking markers: C7 (C7 spinous process); GT (greater tubercle); LA (lower arm); LE (lateral epicondyle); RSP (radial styloid process); SCAP 1, 2, 3 (acromion marker cluster); SN (sternal notch); T8 (T8 spinous process); UA (upper arm); XP (xiphoid process).
Adapted from
https://human.biodigital.com/edit?id=production/maleAdult/male_complete_anatomy_13

Figure 8. Locations of Retroreflective Kinematic Markers on Participant
Prior to kinematic marker placement, locations were identified and marked on the participant. Female participants were asked to wear a shirt that exposed the scapula and was tight fitting to reduce movement of the T8 and XP markers.

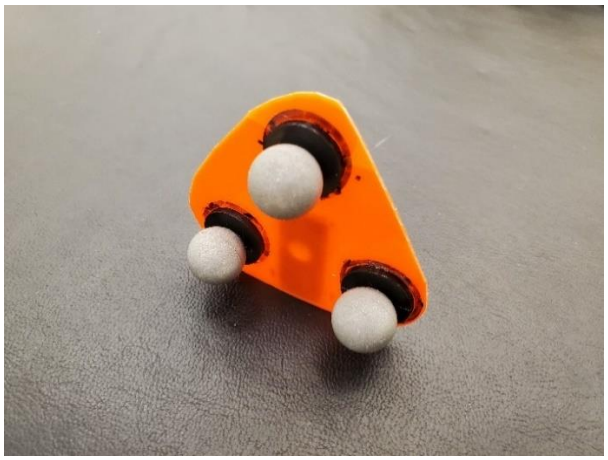


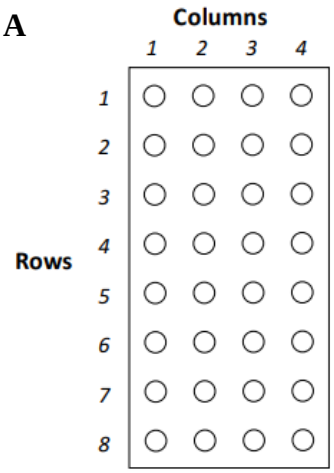
Figure 9. Acromion Marker Cluster

The AMC is composed of three retroreflective markers fixed to a rigid surface that are equidistant to one another (i.e. form an isosceles triangle). The AMC was fixed to the acromion of each participant.

6.3.3 High-Density Surface Electromyography

High-density surface electromyography of the right UT, MT and LT fibres was collected using three, two-dimensional grids of 32 electrodes (GR10MM0804, OT Bioelettronica, Turin, Italy) arranged in four columns and eight rows ***(Figures 10A & 10B)***. Prior to placement of the electrode grids the skin was shaved (if necessary), cleaned with abrasive paste (Nuprep, Weaver and Company, Aurora, Colorado, USA) and wiped clean with an alcohol swab (70%).

A



B

Figure 10. 32-Electrode Grid

(A) Schematic of a 32-electrode grid with four columns and eight rows of electrodes. (B) 32-electrode grid used for data collection. Grid was secured to the skin using adhesive disposable matrices filled with conductive paste secured using retention tape. Grids were placed medial to the innervation zone with rows running parallel to muscle fibre direction.

The innervation zones (IZ) of the UT, MT and LT were identified by first estimating their location via anatomical landmarking[197] **(Table 4, *Figure 11*)**. Next, a linear array of 8 electrodes (SA 8/10, OT Bioelettronica, Turin, Italy) were placed over this location and positioned parallel to the muscle fibre direction in a single differential configuration. With the linear array in place, the participant performed a 5-second isometric contraction against manual resistance targeting the respective region of the trapezius. Positions for manual muscle testing were based off previously published standardized method[95] and modified to allow the participant to stand upright throughout ***(Figure 12).*** For UT, the participant's arm remained at their side in a neutral position. Manual resistance was applied just superior to the elbow as the participant attempted to elevate the shoulder girdle, shrugging the shoulder superiorly. For MT, the participant abducted the arm to 90° and manual resistance was applied at the posterior aspect of the upper arm as the participant horizontally abducted. For LT, the participant abducted the arm to 135° and manual resistance was applied at the posterior aspect of the upper arm, with the participant applying force in a posterior direction. Once IZs were identified, a mark was made on the participants' skin using washable marker and an electrode grid was positioned medial to the IZ ***(Figure 13)***. Placing the grid medial to the IZ allowed for a more accurate estimation of EMG variables and reduced signal noise[56,197].

Table 4. Barbero and colleagues' anatomical landmark frames for determination of innervation zones[197].

Trapezius Region	Anatomical Landmark Frames
Upper Trapezius	A line from the distal portion of the clavicle to the spinal process of the sixth cervical vertebrae
Middle Trapezius	A line between the acromial angle and the spinous process of the seventh cervical vertebrae
Lower Trapezius	A line between the scapular spine and the spinous process of the twelfth thoracic vertebrae

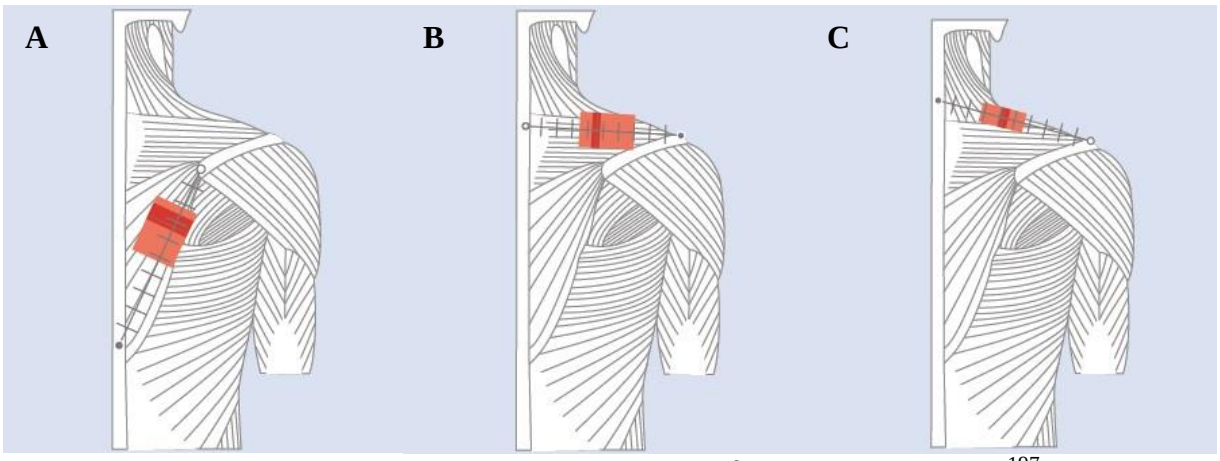


Figure 11. Anatomical Landmark Frames for Innervation Zones[197]
Innervation zone was determined for the LT (A), MT (B), and UT (C) using the Anatomical Landmark Frames as reference for placement of the linear electrode array.

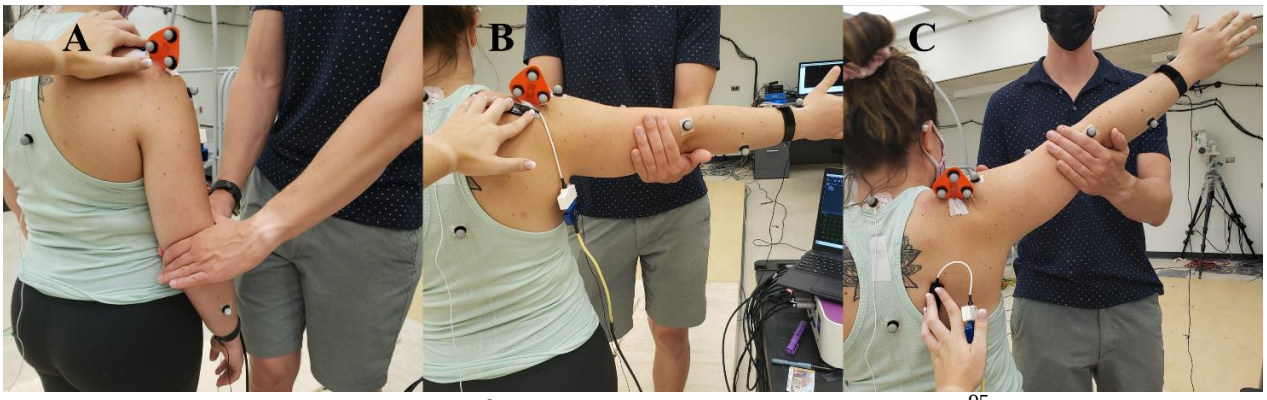


Figure 12. Modified Manual Muscle Testing Positions[95]
Linear array was positioned over the corresponding region of the trapezius during manual muscle testing. A 5-second isometric contraction was held as the researcher applied resistance during the following movements: (A) UT – shoulder girdle elevation (i.e. shoulder shrug); (B) MT – shoulder horizontal abduction from 90°; (C) LT – shoulder horizontal abduction from 135°.

Rows of the electrode grids were aligned to run parallel to the estimated fibre direction of each region of the trapezius. Electrode grids were then secured to the skin using adhesive disposable matrices filled with conductive paste (Ten20, Weaver and Company, Aurora, Colorado, USA), and further secured using retention tape. A ground reference electrode (T3545, OT Bioelettronica, Turin, Italy) was fixed to the spinous process of the C7 vertebrae and connected to the adapter of each electrode grid. A grounding wrist strap (WS1, OT Bioelettronica, Turin, Italy) was wet and placed around the participants' wrist to reduce noise and improve signal quality. The electrode grids and grounding wrist strap were then connected to

the Quattrocento bioelectrical signal amplifier, completing the experimental set-up ***(Figure 14)***. Prior to the start of the experimental task, raw EMG signals were visually inspected for the presence of unresponsive channels (e.g. contact issues, short circuits).

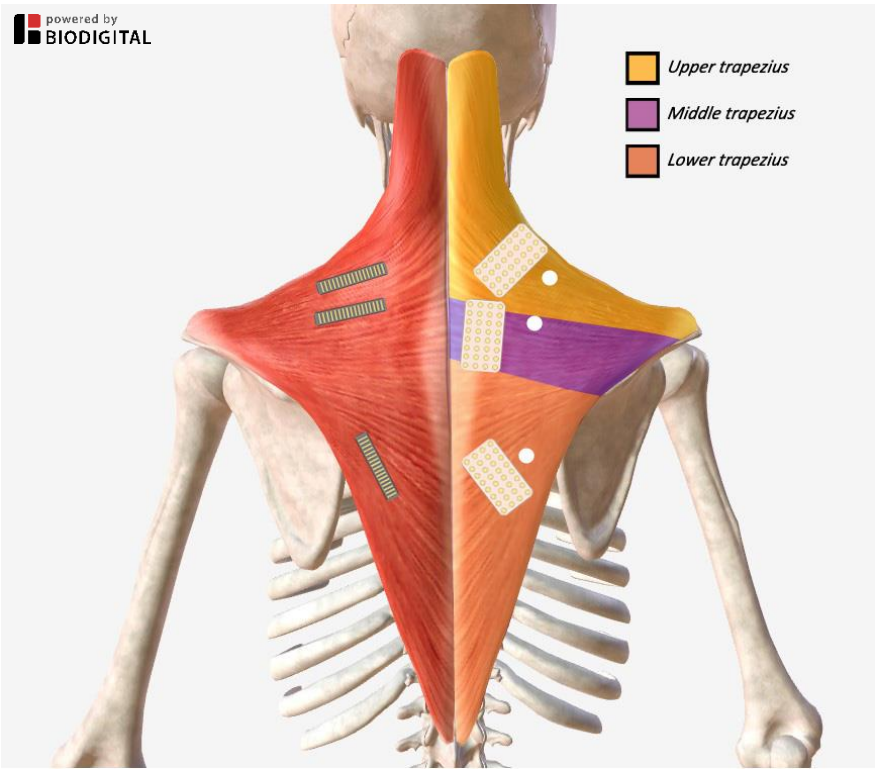


Figure 13. Innervation Zone and Electrode Grid Approximated Locations
Approximate position of the linear electrode arrays for determination of innervation zones (depicted on the left). Estimated electrode grid placements on the right upper, middle and lower trapezius medial to innervation zones (white circles). Determination of innervation zone location was performed on the right side but is depicted on the left for comparison to the approximate location of the innervation zones and electrode grids (both shown on the right). Reference electrodes are not depicted in this figure.
Adapted from
https://human.biodigital.com/edit?id=production/maleAdult/male_complete_anatomy_13

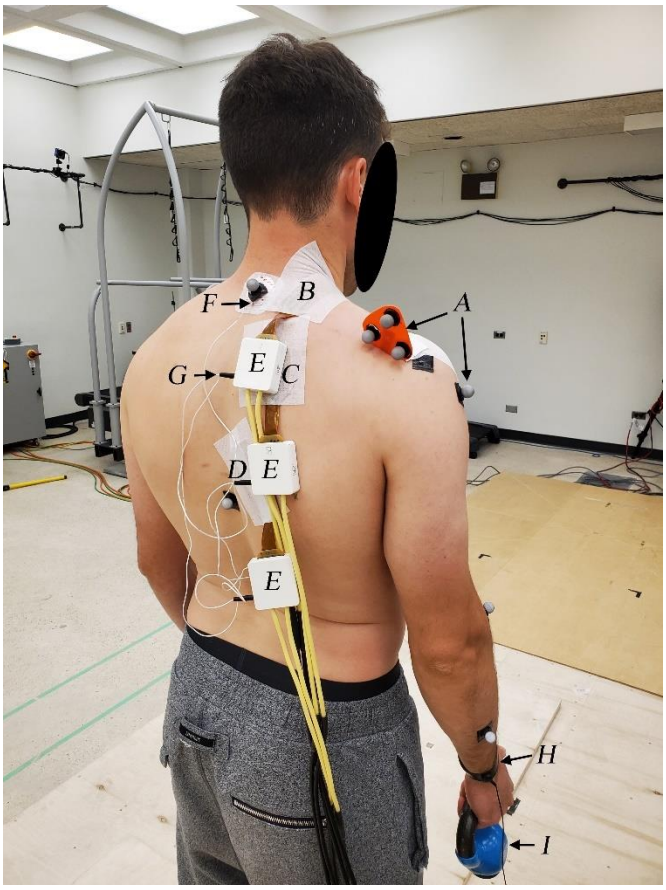


Figure 14. Completed Experimental Setup

Kinematic markers (A) were attached at specified landmarks, followed by electrode grids at the upper trapezius (B), middle trapezius (C), and lower trapezius (D). Electrode grids were placed medial to the determined innervation zones and secured with retention tape, then connected to a matrix adapter (E). A grounding electrode was placed on the C7 spinous process (F) with a grounding cable (G) connecting the ground electrode to each adapter. A grounding wrist strap and grounding cable (H) were placed on the participant's right wrist, which connected directly to the signal amplifier. The participant was given a handheld weight (I) for use in the experimental task.

6.3.4 Dynamic Shoulder Task

Following completion of the SDT, placement of kinematic markers, IZ determination and electrode placement, participants were asked to complete the same flexion task performed during the SDT using a similar protocol. All parameters for the experimental task were identical to the SDT (i.e. range of motion, cadence, weight), however the participant now completed the task unilaterally (i.e. right side only). Additionally, motion from one repetition to the next was no longer continuous. Instead, participants stopped after each repetition (i.e. individual trial) and began their next trial when prompted. Trials were separated by approximately 10 seconds to allow time for the researchers to save the collected EMG and kinematic data and prepare for the

subsequent trial to be recorded ***(Figure 15)***. Each trial began using a verbal cue from a research member to indicate when to start the motion (e.g. “3, 2, 1, go”). Kinematic recordings were started before EMG recordings for each trial in order to ensure data could be synchronized during processing.

Participants completed the same protocol for abduction following the flexion task. However, only data from the flexion task was included in the current analysis.

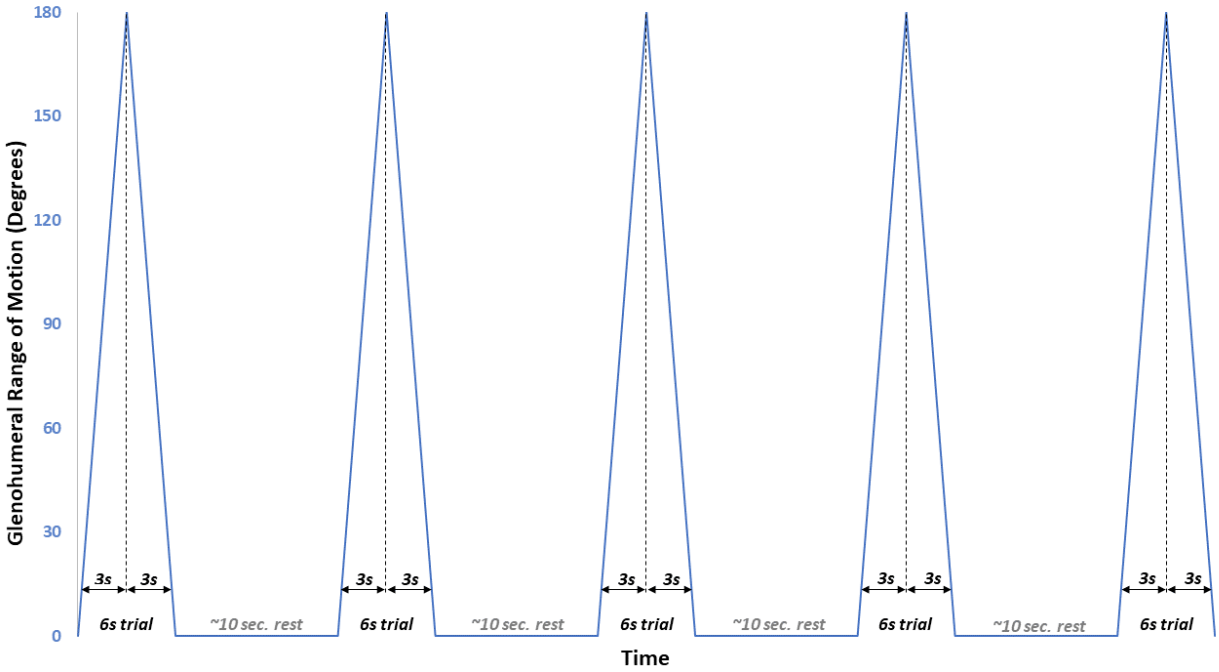


Figure 15. Schematic of Experimental Protocol for Shoulder Flexion Task

6.4 Data Processing

6.4.1 Kinematic Processing

Kinematic data was collected using Vicon Nexus software (version 2.12.0, Vicon Motion Systems, Los Angeles, CA, USA) sampled at 64 Hz throughout the experimental task. All kinematic data was processed using Vicon Nexus by running custom pipelines for the static calibration and movement trials. All kinematic data was visually inspected to ensure marker trajectories were correctly labelled throughout the trial. A minimum of 3 cameras were required to begin a marker trajectory. Interpolation of missing marker trajectory data was performed using a Woltring Quintic spline to fill gaps up to 32 frames (0.5s) in length with a 3rd order polynomial function and numerical fit of three frames before and after the gap. Reconstructed data was

closely inspected to ensure it followed a normal trajectory. For larger gaps or markers missing from the entire trial, 2 cameras were used to begin a marker trajectory. Affected markers were closely inspected to ensure a normal trajectory throughout. Kinematic trials with errors in reconstruction (e.g. unnatural segment movement) or that included gaps in marker trajectory data after gap filling or changing the minimum cameras were excluded from analysis. A minimum of 3 corresponding EMG and kinematic trials per phase were required for analysis. However, all 5 trials in a phase were used for analysis if they met the above criteria for inclusion. All marker trajectories were then exported to .csv files.

A custom Matlab code (The MathWorks, Natick, MA, USA) was used to rotate the global coordinate system to ISB standards[218] and filter raw marker trajectories using a low-pass, zero-lag, fourth-order Butterworth filter with a 6 Hz cut-off[220]. Filtered data were used to create local coordinate systems for each segment to describe relevant rotations. The GH joint was calculated as 60mm below the acromion parallel to the Y vector of the torso[221]. Thoracohumeral (i.e. humerus relative to the thorax) rotations were then calculated with the Y-X'-Y" sequence[218] representing plane of elevation, elevation, and axial rotation, respectively (***Figure 16)***.

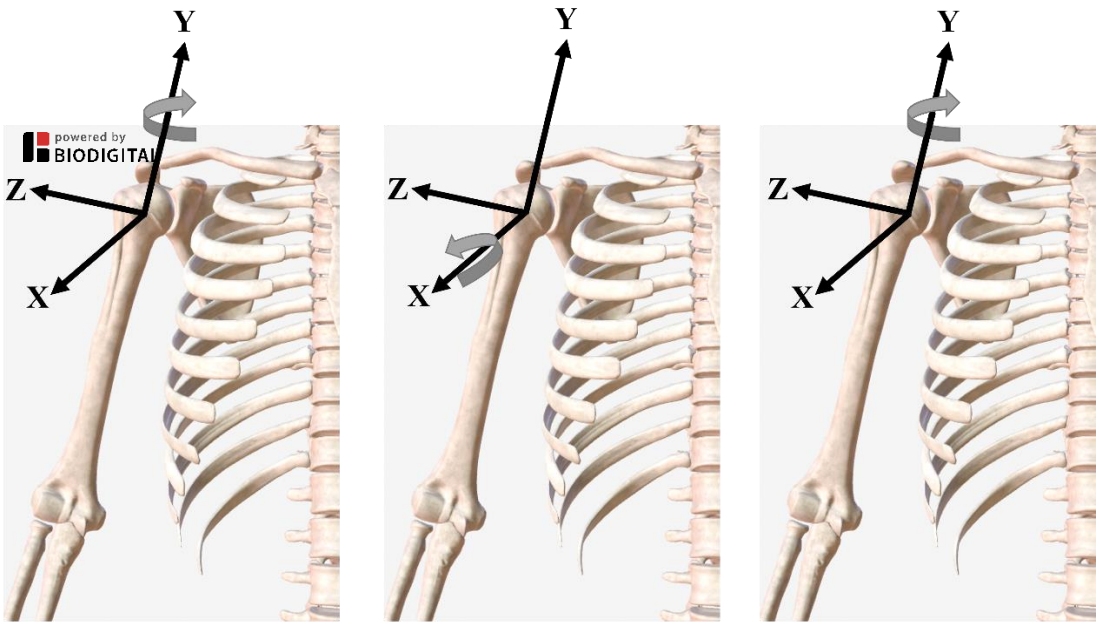


Figure 16. Joint Coordinate System for Calculating Thoracohumeral Rotations[218]
Global coordinate system was rotated to ISB standards prior to the creation of local joint coordinate systems for each segment. Rotation of the humerus relative to the thorax was then calculated using the Y-X'-Y" sequence[218], shown here from left to right.
Adapted from
https://human.biodigital.com/edit?id=production/maleAdult/male_complete_anatomy_16

A trigger was used to collect raw voltage data in conjunction with trajectory data to synchronize the EMG and kinematic data. As EMG began recording for each trial, a simultaneous spike in voltage occurred which was recorded by Vicon Nexus and exported to a .csv file. This synchronization data was used to identify the start of each trial. The kinematic data were then partitioned into 30° increments (i.e. ranges) based on the elevation angle (i.e. rotation about the rotated X-axis) for FE (i.e. 30°–60°, 60°–90°, 90°–120°) and FL (i.e. 120°–90°, 90°–60°, 60°–30°) as these ranges have been used previously[44,154]. While the study intended to analyze data from 0°–150°, data from 0°–30° was not utilized as participants did not achieve a true 0° GH angle at the starting position (i.e. arm at rest). Further, data from 120°–150° was not analyzed as several participants did not reach 150° of arm elevation. No participants reached 180° of elevation, however data from 150°–180° was not intended to be analyzed as it was anticipated not all participants would achieve a true 180° of GH range of motion **(*Figure 17*)**. The frame number corresponding to the beginning and end of each range was then extracted

from the Matlab code. As kinematic data was collected at 64 Hz, all exported frames were multiplied by 32 to correspond with the EMG data collected at 2048 Hz.

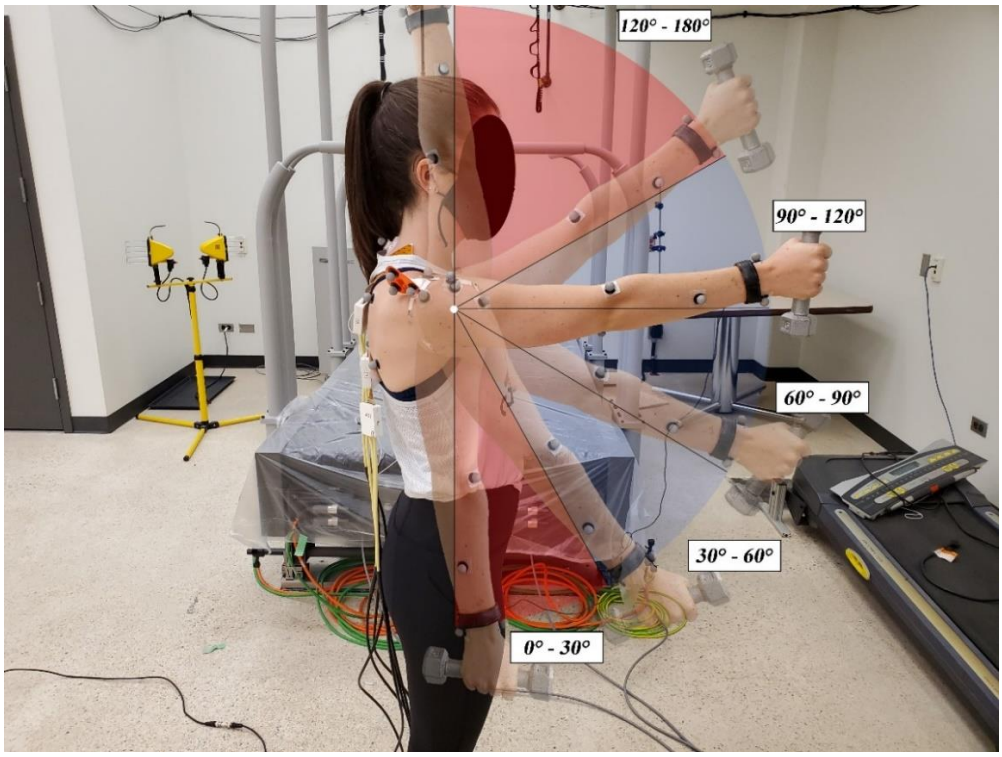


Figure 17. Arc of Analysis for Shoulder Flexion Task
Participants will perform the elevation and lowering task through a full range of motion, with ranges partitioned into 30° increments. However, only ranges between 30°-120° will be analyzed (blue region) while between 0°-30° and 120°-180° will be excluded (red region).

6.4.2 EMG Processing

Raw EMG data was collected using OT BioLab+ software (version 1.5.6.0, OT Bioelettronica, Turin, Italy) on a Dell laptop (Latitude 5500, Dell Canada, Toronto, ON, Canada). EMG signals were measured in volts and acquired in a monopolar configuration with a gain of 500, band-pass filtered (-3 dB bandwidth, 10–500 Hz), sampled at 2048 Hz, and converted to digital data by a 16-bit A/D converter (Quattrocento, OT Bioelettronica, Turin, Italy). A digital high-pass filter at 30 Hz was utilized to remove electrocardiogram contamination from the EMG signal[222]. Visual inspection of raw EMG signals was performed to determine the presence of poor-quality channels (e.g. zero amplitude, non-physiological amplitude spikes, etc.) that was reconstructed using interpolation of neighbouring channels when necessary. EMG trials displaying poor signals in >2 channels in the same row, or >4 channels per grid were excluded from analysis. Single differential spatial filtering was performed using OT BioLab+ resulting in

an 8 x 3 channel matrix. The resulting data from the 24 channels was exported to .csv files for further processing.

EMG files exported from OT Biolab+ were imported into LabChart (version 8.1.19, ADInstruments, Colorado Springs, CO, USA). A custom macro was used to calculate and export the average Root Mean Square (RMS) of each channel over each specified range for every grid during all included trials. The RMS value is widely utilized to quantify the electric signal as it reflects the physiological excitation in the MUs during contraction[57,59,60,223]. RMS data for each participant was exported to an Excel file and averaged between trials to create a single 8 x 3 RMS grid for each range for FE (30°–60°, 60°–90°, 90°–120°) and for FL (120°–90°, 90°–60°, 60°–30°) for each grid (UT, MT, LT), resulting in 18 RMS grids per participant. Data from each grid was then inputted into a custom Excel formula based on Madeleine et al. (2006)[65] to calculate and visualize the mean barycentre location (i.e. y-coordinate), which was subsequently used for statistical analysis. Weighted sums were created in the y-direction (i.e. cranial-caudal) by multiplying channel values by their respective column number. Weighted sums were then multiplied by the relative contribution of a single channel to the entire grid (i.e. 1/total grid RMS). The resulting value provided an absolute location of barycentre on the grid in cm (i.e. 0 cm to 7 cm) in the y-direction. Barycentre data was then expressed as a percentage of total distance from the most cranial (i.e. grid origin, y = 0%) electrode to the most caudal (i.e. 100%) electrode **(*Figures 18 & 19*)**. Thus, for each participant the final data set consisted of a barycentre y-coordinate for each of the 18 RMS grids. The average number of trials included for analysis from each participant was 4.43 for CON and 4.57 and DYS.

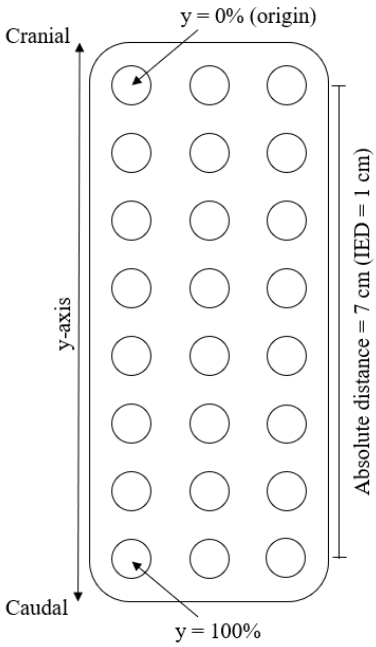


Figure 18. Schematic representation of electrode grid with y-coordinate axis and origin for defining barycentre coordinate. IED = inter-electrode distance.

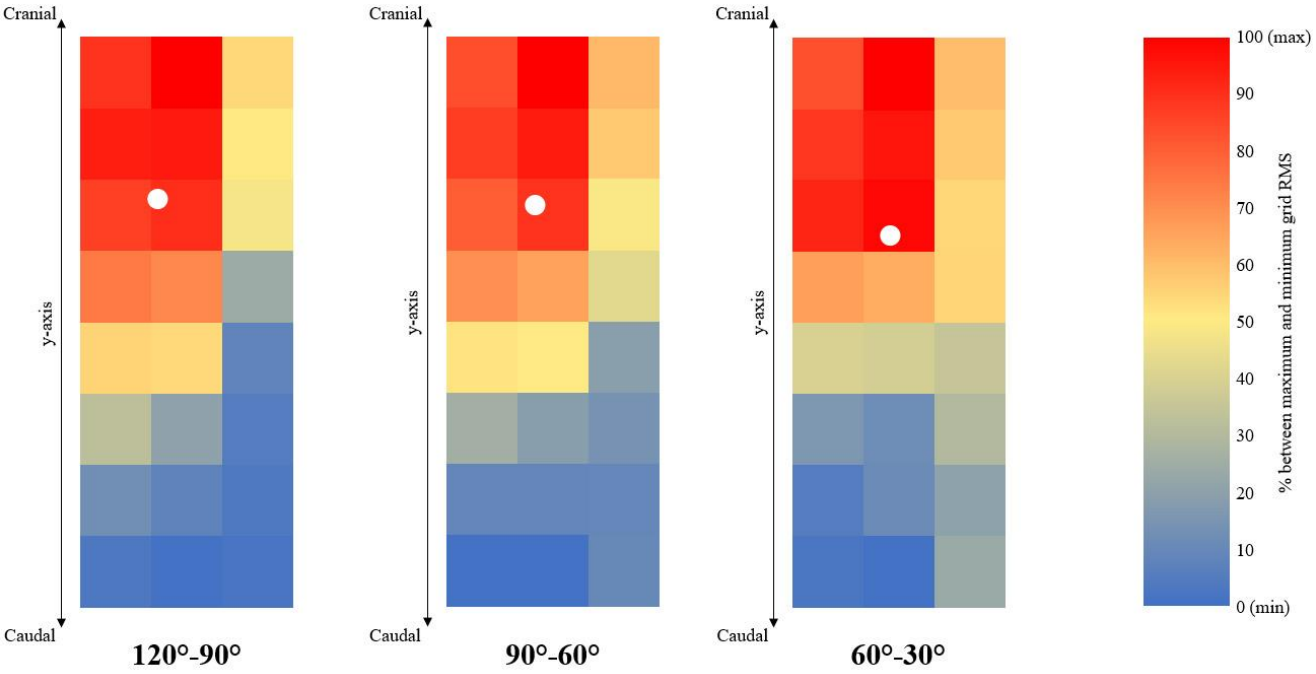


Figure 19. Visualization of EMG amplitude distributions during the lowering (eccentric) phase of the flexion task for one participant in the lower trapezius. The lowest root-mean-square (RMS) value within each grid represents the minimum (0%) while the highest RMS value represents the maximum (100%). All other grid RMS values are expressed as a relative percentage between the maximum and minimum values. Mean location of RMS over the entire grid is represented by a white circle (i.e. barycentre).

6.5 Statistical Analysis

Statistical analysis was completed using SPSS (27.0 for Windows, Chicago, Il, USA). A two-tailed, two-way (group*angle) between-within (BW) analysis of variance (ANOVA) was conducted to determine the effect of group (SD or CON) on the dependent variable (mean barycentre y-coordinate [$BARY_Y$]) at three ranges (30°–60°, 60°–90°, 90°–120°) during two movements (FE, FL) in three grids (UT, MT, LT), resulting in six total two-way BW ANOVAs (2 phases x 3 grids).

Before statistical analysis, outliers were identified via studentized residuals with values greater than ±3 standard deviations. When outliers were detected, the raw data of the trial containing the outlier was inspected for poor-quality EMG channels or issues with kinematic marker trajectories that may have been missed during initial data processing. If no errors were found during inspection, all variable data (i.e. barycentre coordinates) from the participant containing the outlier value(s) was removed from the analysis and the two-way BW ANOVA was re-run. Results from the original ANOVA were compared with the ANOVA in which outliers were removed, and if a different result was not seen (e.g. notable change in *p*-value, substantial difference in group mean or standard deviation, etc.) the outliers were not removed from the final analysis. Normality of the data was determined using Shapiro–Wilk's test for normality as well by assessing kurtosis and skewness values. For the Shapiro–Wilk test, data was considered normally distributed if $p > .05$. Skewness and kurtosis were converted into *z*-scores by dividing each value by their respective standard error. Values > ±1.96 were considered significant at $p < .05$[224]. However, ANOVAs are considered robust to deviations of normality[225,226], particularly with equal group sizes[227]. Therefore, violations were reported but no transformations were performed on the data.

To determine if the variance between CON and DYS were equal Levene's test was performed, with a violation of the assumption of homogeneity of variances considered $p \leq .05$. If Levene's test is significant, Welch's *F*-test which assumes unequal variance was run and the results were reported. Welch's *F*-test is designed to be accurate when the homogeneity of variance is violated [228]. To determine homogeneity of covariance of the data, Box's test of equality of covariance matrices was used, with a violation of equality of covariance considered $p \leq .001$.

Prior to interpreting the result of the two-way BW ANOVA interaction, Mauchly's test of sphericity was interpreted to determine if the assumption of sphericity had been violated (p <.05). If the assumption was violated, a Greenhouse-Geisser correction was used to adjust the degrees of freedom used in calculating the p-value and epsilon (ε) was reported [229]. Significant interactions found in the two-way BW ANOVA were followed by testing for simple main effects of group and angle. Simple main effects of group were determined using one-way ANOVAs, while simple main effects of angle were determined using one-way repeated measures ANOVAs. If a one-way repeated measures ANOVA produced a significant result, it was followed by a multiple pairwise comparison using a Bonferroni adjustment to determine where the differences occurred. If no significant interactions were found during the two-way BW ANOVAs, they were followed by testing for main effects. Main effects of group were determined using a one-way ANOVA, while main effects of angle were determined using one-way repeated measures ANOVAs. If a one-way repeated measures ANOVA produced a significant result, it was followed by a multiple pairwise comparison using a Bonferroni adjustment to determine where the differences occurred. Results were reported as mean and standard deviation (SD). Statistical significance was accepted at $p < 0.05$. The minimum effect size representing an effect was considered $\eta_p^2 = 0.04$, a moderate effect size was considered $\eta_p^2 = 0.25$, and a large was considered $\eta_p^2 = 0.64$[230].

A priori power analysis for sample size determination was not conducted as no previous literature has investigated the spatial distribution of excitation within the UT, MT and LT in individuals with and without SD. Investigations reporting changes in spatial distribution of excitation within the upper trapezius in response to various interventions in healthy individuals have utilized sample sizes between 9-22[58,59,61–66,145], while investigations comparing healthy individuals to those with fibromyalgia[60] and myofascial pain[56] have utilized groups of 10 (N=20) and 12/13 (N=25), respectively. In the only study conducted on the spatial distribution of excitation of the LT, a sample size of 22 healthy individuals was utilized[83]. Our study analyzed 28 participants per group (N=56) to be able to detect potential differences in spatial distribution of excitation that may exist between groups.

Inter-rater reliability of the SDT was also calculated for all screened participants (N = 87) using percentage of agreement and weighted kappa (κ_w) with linear weights. Inter-rater reliability

was considered excellent if $\kappa_w > 0.8$, substantial if $\kappa_w = 0.61–0.8$, moderate if $\kappa_w = 0.41–0.6$, fair if $\kappa_w = 0.21–0.4$, and poor if $\kappa_w = 0–0.2$[231].

7. Results

The results of the two-way BW ANOVAs are presented in Appendix F, including effect size, while mean group $BARY_Y$ are presented in Appendix G. Between-group differences in $BARY_Y$ are presented in Appendix H, while combined-group $BARY_Y$ are presented in Appendix I.

7.1 Upper Trapezius

7.1.2 Elevation Phase

There were three outliers with studentized residual values of 3.16, 3.21, and 3.32. The data was normally distributed as assessed by Shapiro-Wilk's test ($p > .05$) for CON 30°-60°, CON 60°-90°, CON 90°-120°, and DYS 30°-60°. The data was not normally distributed ($p < .05$) for DYS 60°-90° ($p = .048$) and DYS 90°-120° ($p = .022$). Kurtosis *z*-scores of 4.259 and 4.636 were found for DYS 60°-90° and DYS 90°-120°, respectively, while all other skewness and kurtosis *z*-scores fell below ±1.96. There was homogeneity of variances ($p > .05$) between groups at each range, as assessed by Levene's test of homogeneity of variances. There was homogeneity of covariances ($p > .001$) as assessed by Box's M test. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 52.085$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .615$). For the interaction effect, observed power = .059 with an effect size of $\eta_p^2 = .001$, indicating no magnitude of difference between groups.

There was no statistically significant interaction between group and angle on $BARY_Y$, $F(1.230, 66.432) = 0.077$, $p = .833$, $\eta_p^2 = .001$ ***(Figure 20)***. Therefore, main effects of group and angle were explored. The main effect of group on $BARY_Y$ was not significant, $F(1, 54) = 0.512$, $p = .477$, $\eta_p^2 = .001$, a difference of 0.99% (95% CI, -1.79 to 3.78). The main effect of angle was significant, $F(1.230, 66.432) = 29.080$, $p < .001$, $\eta_p^2 = .350$, with $BARY_Y$ shifting cranially from 57.0 ± 5.1% at 30°-60°, to 55.7 ± 5.4% at 60°-90°, and to 55.1 ± 5.5% at 90°-120°. Post hoc pairwise comparisons using a Bonferroni adjustment revealed a statistically significantly difference in $BARY_Y$ between 30°-60° and 60°-90° (1.3% [95% CI, .8 to 1.8], $p < .001$), 30°-60° and 90°-120° (1.9% [95% CI, 1.1 to 2.8], $p < .001$), and 60°-90° and 90°-120° (0.6% [95% CI, .1 to 1.1], $p = .013$).

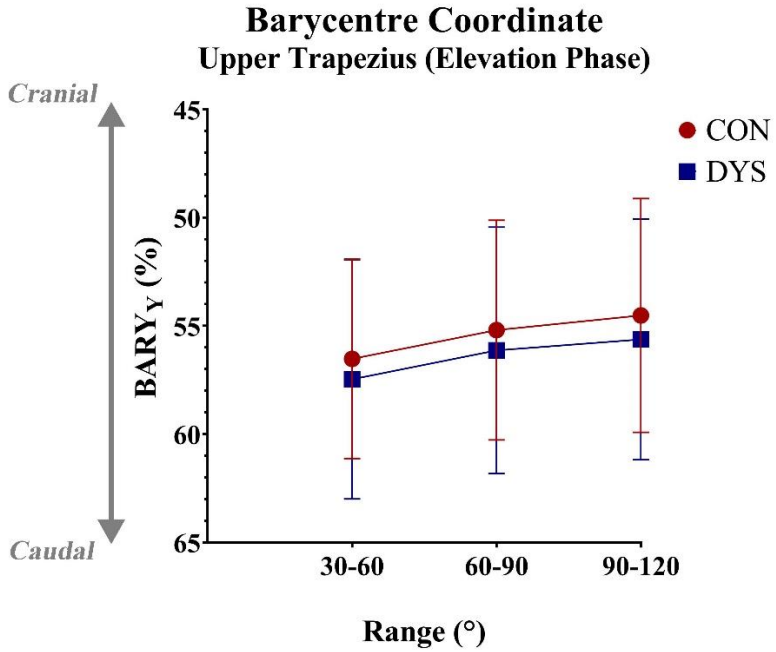


Figure 20. Mean (±SD) y-coordinate of barycentre ($BARY_Y$) at each range for control group (CON) (*n* = 28) and dyskinesis group (DYS) (*n* = 28) during the elevation phase of the flexion task in the upper trapezius (i.e. UT_FE). Data are expressed in percentages (0% to 100%) with respect to distance from grid origin (i.e. 0)

7.1.2 Lowering Phase

There was a single outlier with a studentized residual value of 3.04. The data was normally distributed as assessed by Shapiro-Wilk's test (p > .05) for both groups at each range. Kurtosis z-scores of 3.698 and 2.305 were found for DYS 120°-90° and DYS 90°-60°, respectively, while all other skewness and kurtosis z-scores fell below ±1.96. There was homogeneity of variances ($p > .05$) between groups at each range, as assessed by Levene's test of homogeneity of variances. There was homogeneity of covariances ($p > .001$) as assessed by Box's M test. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 32.608$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .685$). For the interaction effect, observed power = .454 with an effect size of $\eta_p^2 = .051$, indicating a small magnitude of difference between groups.

There was no statistically significant interaction between group and angle on $BARY_Y$, $F(1.370, 73.998) = 2.887$, $p = .081$, $\eta_p^2 = .051$ ***(Figure 21)***. Therefore, main effects of group and angle were explored. The main effect of group on $BARY_Y$ was not significant, $F(1, 54) = 0.912$, $p = .344$, $\eta_p^2 = .017$, a difference of 1.4% (95% CI, -1.5 to 4.3). The main effect of angle was

significant, $F(1.370, 73.998) = 6.321$, $p = .008$, $\eta_p^2 = .105$, with $BARY_Y$ shifting caudally from 55.5 ± 5.5% at 120°-90°, to 55.7 ± 5.6% at 90°-60°, to 56.4 ± 5.4% at 60°-30°. Post hoc pairwise comparisons using a Bonferroni adjustment revealed a statistically significantly difference in $BARY_Y$ between 120°-90° and 60°-30° (-.9% (95% CI, -1.7 to -.1), $p = .028$), and 90°-60° to 60°-30° (-.7% [95% CI, -1.3 to 0.0], $p = .034$), but not between 120°-90° and 90°-60° (-.3% [95% CI, -.7 to .2], $p = .506$). However, differences between 90°-60° and 60°-30° were not significant when outliers were removed from analysis (-.6% [95% CI, -1.2 to 0.0], $p = .056$).

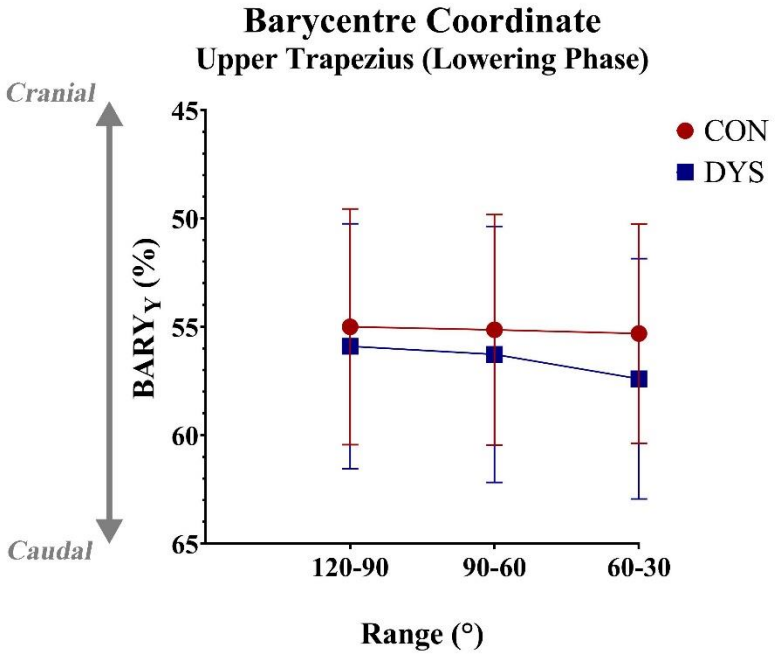


Figure 21. Mean (±SD) y-coordinate of barycentre ($BARY_Y$) at each range for control group (CON) ($n = 28$) and dyskinesis group (DYS) ($n = 28$) during the lowering phase of the flexion task in the upper trapezius (i.e. UT_FL). Data are expressed in percentages (0% to 100%) with respect to distance from grid origin (i.e. 0)

7.2 Middle Trapezius

7.2.1 Elevation Phase

There were no outliers with studentized residual values greater than ±3 standard deviations. The data was normally distributed as assessed by Shapiro-Wilk's test ($p > .05$) for CON 90°-120° and DYS 90°-120°. The data was not normally distributed ($p < .05$) for CON 30°-60° ($p = .035$), CON 60°-90° ($p = .025$), DYS 30°-60° ($p = .023$), and DYS 60°-90° ($p = .020$). Skewness z-scores of -2.570 and -2.749 were found for DYS 30°-60° and DYS 60°-90°, respectively, while a kurtosis z-score of 2.422 was found for DYS 60°-90°. All other skewness and kurtosis z-scores

fell below ±1.96. There was homogeneity of variances ($p > .05$) between groups at each range, as assessed by Levene's test of homogeneity of variances. There was homogeneity of covariances ($p > .001$) as assessed by Box's M test. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 45.512$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .634$). For the interaction effect, observed power = .052 with an effect size of $\eta_p^2 < 0.001$, indicating no magnitude of difference between groups.

There was no statistically significant interaction between group and angle on $BARY_Y$, $F(1.269, 68.515) = 0.015$, $p = .942$, $\eta_p^2 < 0.001$ ***(Figure 22)***. Therefore, main effects of group and angle were explored. The main effect of group on $BARY_Y$ was not significant, $F(1, 54) = 0.559$, $p = .458$, $\eta_p^2 = .010$, a difference of 1.8% (95% CI, -3.0 to 6.6). The main effect of angle was significant, $F(1.269, 68.515) = 5.669$, $p = .014$, $\eta_p^2 = .095$, with $BARY_Y$ shifting caudally from 60.5 ± 9.0% at 30°-60° to 61.2 ± 8.9% at 60°-90°, and cranially from 60°-90° to 90°-120°, with a barycentre coordinate of 59.7 ± 9.6%. Post hoc pairwise comparisons using a Bonferroni adjustment revealed a statistically significantly difference in $BARY_Y$ between 60°-90° and 90°-120° (1.5% [95% CI, .6 to 2.4], $p < .001$), but not from 30°-60° to 60°-90° (-.6% [95% CI, -1.5 to .2], $p = .188$) or 30°-60° to 90°-120° (.9% [95% CI, -.6 to 2.4], $p = .453$).

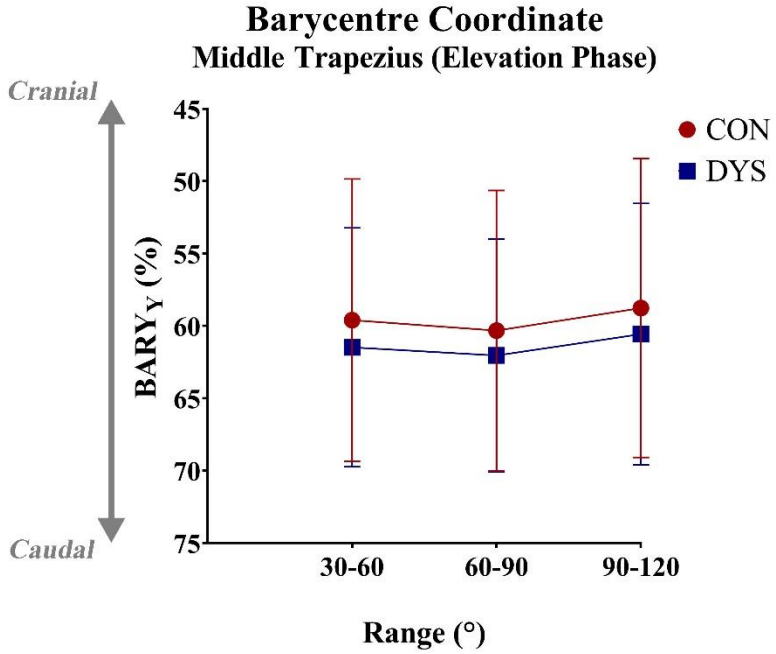


Figure 22. Mean (±SD) y-coordinate of barycentre ($BARY_Y$) at each range for control group (CON) (*n* = 28) and dyskinesis group (DYS) (*n* = 28) during the elevation phase of the flexion task in the middle trapezius (i.e. MT_FE). Data are expressed in percentages (0% to 100%) with respect to distance from grid origin (i.e. 0)

7.2.2 Lowering Phase

There were no outliers with studentized residual values greater than ±3 standard deviations. The data was normally distributed as assessed by Shapiro-Wilk's test ($p > .05$) for CON 60°-30°, DYS 120°-90°, DYS 90°-60°, and DYS 60°-30°. The data was not normally distributed ($p < .05$) for CON 120°-90° ($p = .022$), and CON 90°-60° ($p = .005$). A skewness z-score of -2.105 was found for CON 90°-60° while all other skewness and kurtosis z-scores fell below ±1.96. There was homogeneity of variances ($p > .05$) between groups at each range, as assessed by Levene's test of homogeneity of variances. There was homogeneity of covariances ($p > .001$) as assessed by Box's M test. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 36.323$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .668$). For the interaction effect, observed power = .268 with an effect size of $\eta_p^2 = 0.028$, indicating no magnitude of difference between groups.

There was no statistically significant interaction between group and angle on $BARY_Y$, $F(1.337, 72.189) = 1.576$, $p = .217$, $\eta_p^2 = 0.028$ ***(Figure 23)***. Therefore, main effects of group and angle were explored. The main effect of group on $BARY_Y$ was not significant, $F(1, 54) =$

0.288, $p = .594$, $\eta_p^2 = .005$, a difference of 1.3% (95% CI, -3.4 to 5.9). The main effect of angle was significant, $F(1.337, 72.189) = 26.853$, $p < .001$, $\eta_p^2 = .332$, with $BARY_Y$ shifting caudally from 58.8 ± 9.3% at 120°-90° to 58.9 ± 8.8% at 90°-60°, and cranially from 90°-60° to 60°-30°, with a barycentre coordinate of 55.7 ± 8.7%. Post hoc pairwise comparisons using a Bonferroni adjustment revealed a statistically significantly difference in $BARY_Y$ between 120°-90° and 60°-30° (3.1% [95% CI, 1.5 to 4.7], $p < .001$) and 90°-60° and 60°-30° (3.2% [95% CI, 2.2 to 4.2], $p < .001$), but not from 120°-90° to 90°-60° (-.1% [95% CI, -1.1 to .8], $p = 1.000$).

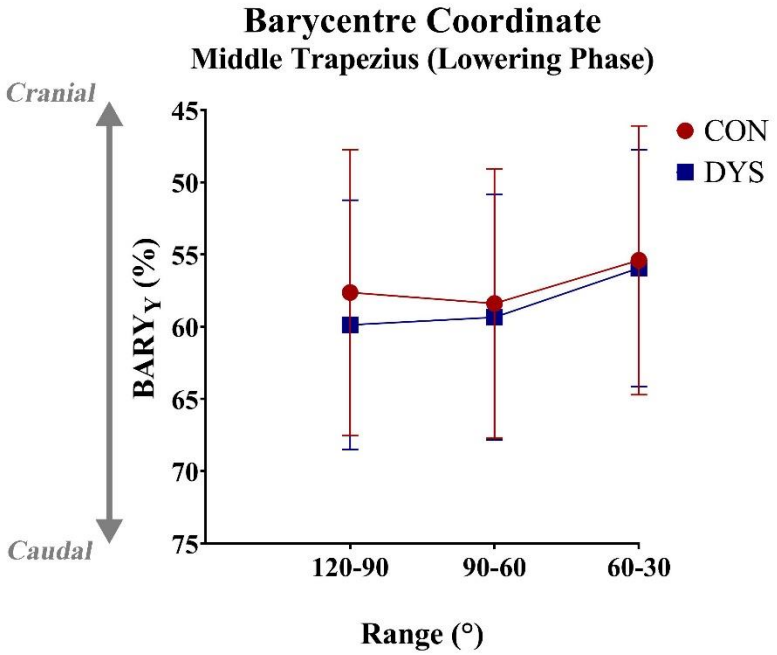


Figure 23. Mean (±SD) y-coordinate of barycentre ($BARY_Y$) at each range for control group (CON) ($n = 28$) and dyskinesis group (DYS) ($n = 28$) during the lowering phase of the flexion task in the middle trapezius (i.e. MT_FL). Data are expressed in percentages (0% to 100%) with respect to distance from grid origin (i.e. 0)

7.3 Lower Trapezius

7.3.1 Elevation Phase

There was a single outlier with a studentized residual value of 3.04. The data was normally distributed as assessed by Shapiro-Wilk's test ($p > .05$) for CON 30°-60°, CON 60°-90°, CON 90°-120°, and DYS 30°-60°. The data was not normally distributed ($p < .05$) for DYS 60°-90° ($p = .026$), and DYS 90°-120° ($p = .006$). Skewness z-scores of 2.001 and 2.109 were found for DYS 60°-90° and DYS 90°-120°, respectively, while all other skewness and kurtosis z-scores fell below ±1.96. There was homogeneity of variances ($p > .05$) between groups at each range, as

assessed by Levene's test of homogeneity of variances. There was homogeneity of covariances ($p > .001$) as assessed by Box's M test. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi^2(2) = 36.438$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .668$). For the interaction effect, observed power = .646 with an effect size of $\eta_p^2 = .079$, indicating a small to moderate magnitude of difference between groups.

There was a statistically significant interaction between group and angle on $BARY_Y$, $F(1.336, 72.136) = 4.635$, $p = .025$, $\eta_p^2 = .079$ ***(Figure 24)***. Therefore, simple main effects of group and angle were explored. A statistically significant difference between groups was found at each range. A difference in $BARY_Y$ of 4.6% (95% CI, .7 to 8.5) was seen for 30°-60° ($F[1, 54] = 5.532$, $p = .022$, $\eta_p^2 = .093$), 5.0% (95% CI, 1.0 to 9.1) for 60°-90° ($F[1, 54] = 6.291$, $p = .015$, $\eta_p^2 = .104$), and 5.8% (95% CI, 1.6 to 10.0) for 90°-120° ($F[1, 54] = 7.646$, $p = .008$, $\eta_p^2 = .124$). For simple main effects of angle, Mauchly's test of sphericity indicated that the assumption of sphericity was violated for CON, $\chi^2(2) = 16.211$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .683$). A statistically significant difference in angle was not seen for CON, $F(1.336, 36.887) = 1.969$, $p = .165$, $\eta_p^2 = .068$. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for DYS, $\chi^2(2) = 23.703$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .626$). A statistically significant difference in angle was seen in DYS, $F(1.251, 33.789) = 4.120$, $p = .042$, $\eta_p^2 = .132$, however post hoc pairwise comparisons using a Bonferroni adjustment revealed no statistically significantly differences between any of the ranges.

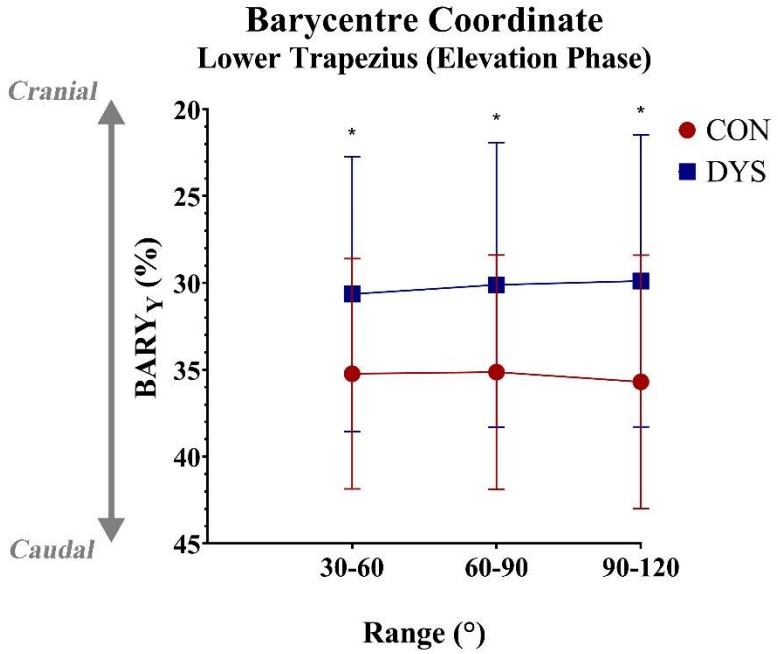


Figure 24. Mean (±SD) y-coordinate of barycentre ($BARY_Y$) at each range for control group (CON) (*n* = 28) and dyskinesis group (DYS) (*n* = 28) during the elevation phase of the flexion task in the lower trapezius (i.e. LT_FE). Data are expressed in percentages (0% to 100%) with respect to distance from grid origin (i.e. 0)
* indicates a significant difference in mean barycentre coordinate between groups ($p < .05$)

7.3.2 Lowering Phase

There was a single outlier with a studentized residual value of 3.11. The data was normally distributed as assessed by Shapiro-Wilk's test ($p > .05$) for CON 120°-90°, CON 90°-60°, CON 60°-30°, DYS 90°-60°, and DYS 60°-30°. The data was not normally distributed ($p < .05$) for DYS 120°-90° ($p = .050$). A skewness z-score of 2.028 was found for DYS 120°-90° while all other skewness and kurtosis z-scores fell below ±1.96. There was homogeneity of variances ($p > .05$) between groups at 120°-90° and 90°-60°, but unequal variance for 60°-30° ($p = .010$), as assessed by Levene's test of homogeneity of variances. There was homogeneity of covariances ($p > .001$) as assessed by Box's M test. Mauchly's test of sphericity indicated that the assumption of sphericity was violated for the two-way interaction, $\chi2(2) = 108.321$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .535$). For the interaction effect, observed power = .546 with an effect size of $\eta_p^2 = .073$, indicating a small to moderate magnitude of difference between groups.

There was a statistically significant interaction between group and angle on $BARY_Y$, $F(1.069, 57.740) = 4.272$, $p = .041$, $\eta_p^2 = .073$ ***(Figure 25)***. Therefore, simple main effects of group and angle were explored. A statistically significant difference between groups was found for 120°-90° ($F[1, 54] = 8.111$, $p = .006$, $\eta_p^2 = .131$), and 90°-60° ($F[1, 54] = 5.661$, $p = .021$, $\eta_p^2 = .095$), with differences in $BARY_Y$ of 5.9% (95% CI, 1.7 to 10.0) and 4.5% (95% CI, .7 to 8.3), respectively. As there was unequal variance for 60°-30°, results from the Welch ANOVA were interpreted. A statistically significant difference between groups was not seen, Welch's $F(1, 46.815) = 2.808$, $p = .100$, with a difference of 3.0% (95% CI, -.6 to 6.6). For simple main effects of angle, Mauchly's test of sphericity indicated that the assumption of sphericity was violated for CON, $\chi^2(2) = 49.917$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .540$). A statistically significant difference in angle was seen for CON, $F(1.079, 29.136) = 5.036$, $p = .030$, $\eta_p^2 = .157$, with $BARY_Y$ shifting cranially from 36.3 ± 6.9% at 120°-90° to 36.1 ± 6.2% at 90°-60°, and caudally from 90°-60° to 60°-30°, with a $BARY_Y$ of 34.9 ± 7.9%. Post hoc pairwise comparisons using a Bonferroni adjustment revealed a statistically significantly difference in $BARY_Y$ between 90°-60° and 60°-30° (-1.8% [95% CI, -3.3 to -.2], $p = .024$), but not between 120°-90° and 90°-60° (.2% [95% CI, -.6 to .9], $p = 1.000$) or 120°-90° and 60°-30° (-1.6% [95% CI, -.3.7 to .5], $p = .184$). Mauchly's test of sphericity indicated that the assumption of sphericity was violated for DYS, $\chi^2(2) = 55.650$ ($p < .001$), therefore a Greenhouse Geiser correction was applied ($\varepsilon = .531$). A statistically significant difference in angle was seen in DYS, $F(1.062, 18.293) = 18.293$, $p < .001$, $\eta_p^2 = .404$, with $BARY_Y$ shifting caudally from 30.4 ± 8.5% at 120°-90°, to 31.6 ± 7.9% at 90°-60°, to 34.9 ± 7.9 at 60°-30°. Post hoc pairwise comparisons using a Bonferroni adjustment revealed a statistically significantly difference in $BARY_Y$ between 120°-90° and 90°-60° (-1.2% [95% CI, -2.1 to -.3], $p = .006$), 120°-90° and 60°-30° (-4.5% [95% CI, -7.1 to -1.8], $p = .001$), and 90°-60° and 60°-30° (-3.3% [95% CI, -5.2 to -1.4], $p < .001$).

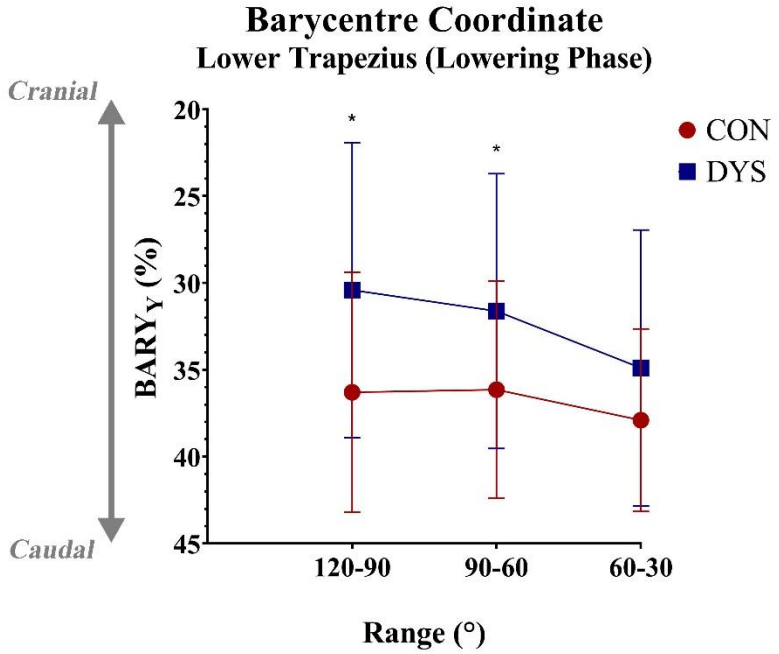


Figure 25. Mean (±SD) y-coordinate of barycentre ($BARY_Y$) at each range for control group (CON) (*n* = 28) and dyskinesis group (DYS) (*n* = 28) during the lowering phase of the flexion task in the lower trapezius (i.e. LT_FL). Data are expressed in percentages (0% to 100%) with respect to distance from grid origin (i.e. 0)
* indicates a significant difference in mean barycentre coordinate between groups ($p < .05$)

7.4 Inter-Rater Reliability of SDT

For shoulder flexion, the two raters were in agreement on 80/87 participants (92%). For the agreement ratings, 35 participants were rated as having normal scapular motion, 34 rated as subtle dyskinesis, and 11 as obvious dyskinesis. For shoulder abduction, the two raters were in agreement on 72/87 participants (83%). For the agreement ratings, 48 participants were rated as having normal scapular motion, 23 rated as subtle dyskinesis, and 1 as obvious dyskinesis. For final rating, the two raters were in agreement on 80/87 participants (92%). For the agreement ratings, 49 participants were rated as having normal scapular motion, 19 rated as subtle dyskinesis, and 12 as obvious dyskinesis. There was a statistically significant agreement between raters for flexion, $\kappa_w = 0.89$ (95% CI, 0.81 to 0.97), $p < .0005$, with the strength of agreement classified as excellent. For abduction, a statistically significant agreement was found, $\kappa_w = 0.66$ (95% CI, 0.51 to 0.81), $p < .001$, with the strength of agreement classified as substantial. There was a statistically significant agreement for final rating, $\kappa_w = 0.86$ (95% CI, 0.75 to 0.97), $p < .0005$, with the strength of agreement classified as excellent.

8. Discussion

Scapular dyskinesis (SD) refers to any alteration in the position and/or motion of the scapula[6,7] and is seen commonly in both symptomatic and healthy individuals. Scapular dyskinesis has been associated with an increased risk of shoulder pain[5,10–13], impaired shoulder function[14–16], as well as numerous shoulder pathologies[7,12,17–30], yet the precise etiology behind SD remains unclear. The purpose of the current study was to determine the differences in spatial distribution of excitation (i.e. barycentre y-coordinate) within the three regions of the trapezius during shoulder elevation and lowering in the sagittal plane between those with scapular dyskinesis (DYS) and those with normal scapular motion (CON). It was hypothesized that during both elevation and lowering through all ranges, the y-coordinate of DYS would be located more caudally in the UT and more cranially in the LT relative to CON, while no differences would be seen in the MT. In line with this hypothesis, the y-coordinate was significantly more cranial in the LT during elevation and lowering for DYS compared to CON. Additionally, no significant difference was seen in the MT, as expected. However, the results from the UT did not support the hypothesis of a more caudally located y-coordinate in DYS, as no significant differences were found.

8.1 Upper Trapezius

Contrary to the stated hypothesis, the location of the barycentre was not significantly more caudal for DYS than for CON in the UT. For the elevation phase, the y-coordinate was located 0.9%, 0.9%, and 1.1% more caudally for DYS compared to CON at 30°-60°, 60°-90°, and 90°-120°, respectively. For the lowering phase, the y-coordinate was located 0.9%, 1.1%, and 2.2% more caudally for DYS compared to CON at 120°-90°, 90°-60°, and 60°-30°, respectively.

The fibres of the UT descend from the superior nuchal line and ligamentum nuchae to attach on the lateral third of the clavicle. During shoulder elevation, the heterogeneous fibre architecture[42,43] of the UT creates differing lines of action to elevate, upwardly rotate, and externally rotate the scapula[42,91,93]. Further, as the distribution[77,78] and proportion of MU/fibre-type[64,78], as well as the fibre diameter[77] are not homogenous within the UT, significant motor variability would be expected. Indeed, HDsEMG studies examining the effect of sustained contractions or fatigue on barycentre location in the UT have found a cranial shift in barycentre over time[57,58,61–65]. Conversely, muscle pain appears to result in a cranial shift in barycentre relative to healthy controls[56,59,60,65], which has also been seen with increased force production[63].

These differing neuromuscular strategies appear to be adaptations to the task or condition to optimize the distribution of excitation within the muscle, reducing muscular fatigue[57,62,74–76] or improving stabilization[42,63,70]. Therefore, it is clear the UT is capable of displaying motor variability within its fibres. However, in the current study the distribution of muscle excitation did not differ between those with and without SD, thus differences in spatial distribution of the UT may not contribute to observable alterations in scapular position or motion.

While the present study did not detect any differences in $BARY_Y$, motor variability between groups may still exist. We recorded muscle amplitude over the entire grid and determined the average of the mean location of excitation over 30° ranges for each group. However, the homogeneity of the distribution of excitation between groups was not compared. For example, those with normal scapular kinematics may have excited fibres more homogenously along the entire grid surface, or utilized greater variability (i.e. recruitment-derecruitment of MUs) while those with SD may have excited fibres in a more concentrated location (i.e. more heterogenous distribution). This may ultimately produce similar results regarding mean barycentre location, despite stark contrasts in excitation patterns. Alternatively, spatial distribution may be relatively homogenous in both groups despite the excitation of different MUs. For example, individuals with SD may recruit MU with smaller diameter fibres capable of producing less force, while those with normal scapular motion may recruit larger diameter fibres capable of providing adequate scapular stabilization. If the distribution of the recruited fibres for each group are diffuse within the muscle region, detectible differences between groups may not be seen on spatial maps. Therefore, if different MUs within the UT are excited, the resulting spatial map may look similar.

Amplitude of muscle excitation in the UT has been previously explored in individuals with SD, with differences in UT excitation noted compared to healthy controls[44–47,50]. For example, Tooth et al. (2020)[46] found a 23% to 31% increase in UT excitation during loaded and unloaded shoulder flexion tasks when compared to the control group. However, due to the small detection surface of bi-polar electrodes used in these studies, it is possible differences in amplitude, as well as the ratio of activation between each region, were not accurately quantified. Indeed, spatial distribution within each region is known to be heterogenous[62,63,205], therefore the mean amplitude of a muscle region would be more accurately observed through HDsEMG. This information

could provide a greater understanding of whether amplitude differences between groups do exist over the entire muscle region that could be contributing to altered scapular kinematics.

The influence of other scapulothoracic muscles must also be taken into consideration when discussing mechanisms for SD. Primarily, the serratus anterior (SA) has frequently been noted to contribute to altered scapular kinematics[34,44–47] due to its action on the scapula, upwardly rotating and protracting the scapula while counteracting the forces of the supraspinatus and deltoid acting to downwardly rotate, anteriorly tilt, and internally rotate the scapula[151]. Therefore, the SA acts to keep the scapula congruent with the thoracic wall while facilitating scapular movement through synergistic actions with other scapular muscles, such as the trapezius[96]. Similar to the trapezius, studies conducted on those with SD[44–47] have utilized bi-polar EMG. Due to the morphological structure of the muscle, HDsEMG would be ineffective at providing an accurate representation of whole muscle excitation. Further, as the SA attaches to the anterior aspect of the scapula, regions of the muscle are inaccessible for EMG analysis. Alternative methods for assessing SA excitation may provide more insight into the impact of the muscle on SD. Other scapulothoracic muscles, such as the rhomboid major and minor, pectoralis minor, or levator scapula may also impact scapular kinematics[86,90], though limited research investigating the excitation of these muscles in SD exists[49].

It must also be considered that SD is likely multi-factorial in nature, with various mechanisms contributing to a change in kinematics. Indeed, Lin et al. (2016)[232] suggested restoring scapular kinematics may not be possible through improvements in muscle excitation timing or amplitude alone. For example, reduced SA activation may lead to prolonged internal rotation (i.e. medial scapular border prominence), creating decreased flexibility in the pectoralis minor and further promoting an increase in anterior tilt and internal rotation of the scapula [151,168,171,172]. The shortened musculature may in turn create postural deficits (e.g. forward head posture, thoracic kyphosis) that promote scapular protraction, leading to imbalances in muscle excitation (e.g. increased UT excitation)[151,174]. The cyclic and holistic nature of the mechanisms behind SD may therefore explain why rehabilitation programs targeting just one of these factors have generally proved ineffective[10]. The impact of other anatomical factors, such as shortened/lengthened ligamentous structures or fascial restrictions, have yet to be explored but may also contribute to the development of SD.

While we did not compare the two phases of shoulder flexion, it must be noted that no differences were found between groups for either the elevation or lowering phase. In previous studies, an increased prevalence of SD during shoulder lowering has been noted[6,44], potentially due to the decreased amplitude in muscle excitation seen during this phase. Indeed, Huang et al. (2015)[44] compared individuals with SD to healthy controls and found differences in muscle excitation between groups primarily occurred during the lowering phase of shoulder flexion. In the present study, alterations in observable scapular kinematics were primarily noted during the lowering phase, including medial or inferior scapular border prominence as well as disruptions in rhythm during scapular downward rotation. While these observations were anecdotal in nature, it does not appear that phase of movement had a significant impact on $BARY_Y$ in either group.

Despite no differences between groups being seen, changes in $BARY_Y$ were found between angles during FE and FL when groups were combined. During the elevation phase, $BARY_Y$ shifted cranially between each angle, followed by a caudal shift during the lowering phase. These results are in line with those found by Falla et al. (2017)[61], who performed a box lifting and lowering task between hip and shoulder height in the sagittal plane (i.e. shoulder flexion). The authors investigated the effect of experimental muscle pain on the spatial distribution of the UT in otherwise healthy male participants. While differences between control and the painful condition were found, $BARY_Y$ in both groups was seen to shift cranially with elevation and caudally with lowering. These alterations in distribution provide evidence of motor variability within the UT, as different fibres within the UT are excited in response to a change in glenohumeral angle. However, the shift in $BARY_Y$ in the current study was relatively small. For FE, a total shift of 1.9% was seen within the observed angles, while a shift of 0.9% was seen during FL. This magnitude of change, while statistically significant, does not demonstrate that the excitation of appreciably different regions of the UT was utilized throughout the range of motion. Instead, it appears subtle modifications in MU recruitment are used to excite muscle fibres with a greater mechanical advantage, or to reduce the risk of fatigue to the region.

8.2 Middle Trapezius

In line with the stated hypothesis, there was no significant difference in the barycentre location between groups in the MT. For the elevation phase, the y-coordinate was located 1.9%, 1.7%, and 1.8% more caudally for DYS compared to CON at 30°-60°, 60°-90°, and 90°-120°,

respectively. For the lowering phase, the y-coordinate was located 2.2%, 1.0%, and 0.6% more caudally for DYS compared to CON at 120°-90°, 90°-60°, and 60°-30°, respectively.

The fibres of the MT run transversely from the spinous processes of C7 and T1 to the acromion and spine of scapula, respectively. Due to its relatively linear line of pull, the MT acts to stabilize and retract the scapula during shoulder elevation[42,91,95]. While the MT appears to be critical to the position and motion of the scapula, we did not anticipate differences in motor variability due to the homogeneity of its muscle fibres[42]. Studies conducted on the MT using HDsEMG are very limited[79,84,85], with only a single study found that observes the MT independently from the LT. In this study, Afsharipour et al. (2016)[79] compared the muscle excitation of the MT in violin and cello players using spatial maps. The authors found differences in amplitude within selected regions of the grid for each instrument played and the task performed. These results indicate differences in spatial distribution, however the distribution of excitation over the whole grid was not investigated. Therefore, the current study appears to be the first to investigate global changes in the spatial distribution of the MT.

While limited studies have been conducted using HDsEMG, the MT has been extensively explored using bi-polar EMG, including in those with SD[44–53]. In each of these studies, MT excitation was either decreased or delayed, leading authors to believe MT contributes to the development of SD. While it can be argued bi-polar EMG may not be representative of entire muscle region, the MT represents a smaller and more homogenous portion of the trapezius, therefore different results would not be expected using HDsEMG to investigate differences in muscle amplitude or timing. Despite this, differences in motor variability may still exist in the MT between SD and healthy controls. As was stated for the UT, the current study did not compare the homogeneity of the distribution of excitation, simply the mean location of excitation occurring within each angle. Differences in spatial distribution may be undetectable using $BARY_Y$ despite excitation of differing MUs if the fibre distribution is diffuse or the resulting location of their mean excitation is similar. Therefore, more research is needed to compare whole map homogeneity and MU distribution.

Interestingly, changes in $BARY_Y$ were found between angles during FE and FL when groups were combined. During FE, a 1.5% cranial shift occurred within the observed angles, but only at the top of the range of motion (i.e. between 60°-90° and 90°-120°). In contrast, the lowering phase displayed a 3.2% caudal shift in $BARY_Y$ only at the bottom of the range of

motion (i.e. between 90°-60° and 60°-30°). These subtle shifts may be in response to a need for more stability within these ranges, exciting muscle fibres with the greatest mechanical advantage to provide the required force. However, similar to the changes seen in the UT, the absolute magnitude of change in $BARY_Y$ was relatively small.

8.3 Lower Trapezius

In line with the stated hypothesis, the location of the barycentre was significantly more cranial for DYS than for CON in the LT. For the elevation phase, the y-coordinate was located 4.6%, 5.0%, and 5.8% more cranially for DYS compared to CON at 30°-60°, 60°-90°, and 90°-120°, respectively. For the lowering phase, the y-coordinate was located 5.9% and 4.5% more cranially for DYS compared to CON at 120°-90°, 90°-60°, with no differences found between groups at 60°-30°.

The fibres of the LT ascend from the spinous processes of T2 to T12, inserting on the deltoid tubercle of the scapular spine. During shoulder elevation, the LT acts to depress, externally rotate and posteriorly tilt the scapula[42,91], while assisting the SA and UT in upward rotation[81,82,93]. Similar to the UT, the orientation of the fibres of the LT are relatively heterogenous[42]. As a result of its several actions and differing fibre orientation, it can be speculated variations in fibre diameter as well as MU/fibre-type distribution and proportion exist within the LT much like those found in the UT. However, no studies examining these anatomical characteristics for the LT were found. Further, studies on the spatial distribution of the LT are very limited[79,83–85]. Bohunicky et al. (2021)[83] explored the impact of UT kinesio tape on the spatial distribution of excitation of the LT during a repeated loaded shoulder elevation task, finding no differences between the tape condition and control, while the effects of GH angle or repeated contractions were not investigated. In the same study discussed previously, Afsharipour et al. (2016)[79] examined the LT during various movements in violin and cello players, discovering differences in amplitude within selective regions of the grid. However, spatial maps were not used to determine changes in barycentre over the entire grid surface. The remaining studies conducted using HDsEMG examined healthy participants during various postures[84] and while providing biofeedback[85], however MT and LT distributions were combined making inferences regarding either region difficult.

Bi-polar EMG studies exploring the LT in SD have found a decrease in LT excitation leading to an increased ratio of UT:LT excitation[44–47]. During shoulder flexion and abduction

tasks, Tooth et al. (2020)[46] found a 32% to 65% reduction in excitation in the LT in individuals with SD compared to healthy controls. Specifically, decreases in excitation were noted during unloaded abduction through elevation and lowering. Further, the UT:LT ratio was significantly higher in the dyskinetic group particularly during the lowering phase of flexion and abduction. While the results of these studies provide evidence of differences in excitation between groups, conclusions drawn from them must be taken with caution. Considering the results of the current study, the mean location of excitation differed between groups throughout elevation and lowering. Therefore, while differences in whole-muscle amplitude are possible, different distributions between groups may have led to unrepresentative EMG measurements as the detection zone of bi-polar electrodes are limited.

The results of the current study provide evidence of differing neuromuscular strategies occurring for those with SD compared to healthy controls. While the specific mechanisms behind the differences cannot be determined based on the data from this study, several mechanisms may have led to differing spatial distributions within the muscle region. First, it is possible the fibres of the recruited MUs in the SD group were of a differing orientation to those found in CON, providing different actions on the scapula. As it is believed the more cranial fibres of the LT are involved in stabilization of the scapula[42], it is possible the cranial shift in DYS was in response to the medial or inferior scapular border prominence exhibited with SD, attempting to provide greater stabilization and control for GH movement. Alternatively, the diameter or composition (i.e. type I vs. type II) of excited fibres may have been different between groups. Type I fibres are more fatigue resistant[233] but are smaller and capable of providing less force[67,77]. Differences in excitation amongst these fibres therefore could impact scapular positioning and motion. Next, the number of MU recruited between groups may have differed. Selective excitation of MUs has been shown within the upper limb[234], as well as selective excitation of muscle sub-regions including the trapezis[92,207–209]. Differing motor strategies may therefore exist between groups that utilize different MUs. It is also possible the DYS group recruited a lower number of MU, or was less efficient with rotation or substitution (i.e. recruitment-derecruitment) compared to CON, leading to alterations in kinematics. Finally, differences in spatial distribution between groups may be attributed to other MU characteristics such as discharge rate and conduction velocity, which impact the amplitude of excitation on EMG[235,236].

Anecdotally, the present study noted the greatest alterations in scapular position and motion during the lowering phase, particularly between 90° and 30° of eccentric motion. Increased incidence of SD during the lowering phase is in line with previous studies[6,44], however the results of the current study found similar differences in $BARY_Y$ between groups during elevation and lowering. Further, while differences were seen between groups at each angle in FE, no differences were seen for FL between 60°-30°. Therefore, despite SD being more apparent during the eccentric phase of motion, it does not appear to result in a greater difference in $BARY_Y$ between groups.

While differences in $BARY_Y$ between angles were seen for UT and MT, no meaningful shift in $BARY_Y$ was observed during FE for either DYS or CON in the LT. This indicates that while differences existed between groups, within each group the barycentre remained relatively constant throughout GH elevation. However, differences were seen during FL in both groups. For CON, $BARY_Y$ shifted 1.8% caudally between 90°-60° and 60°-30° while significant shifts in $BARY_Y$ occurred at each angle for DYS, with a total shift of 4.5% caudally from 120°-90° to 60°-30°. These changes in $BARY_Y$ are indicative of greater motor variability during the lowering phase of motion. The shifting barycentre location may be due to an increased demand on the LT to maintain scapular position during FL, particularly in DYS. For example, the LT may employ a more variable MU recruitment strategy during FL to reduce the risk of fatigue or maximize its mechanical advantage. This same motor variability may not be required during elevation where the scapula appears to be less susceptible to altered kinematics. The shifting barycentre location could explain the lower amplitude of muscle excitation demonstrated in the LT during lowering using bi-polar EMG[153].

The differences in $BARY_Y$ found for the LT may have been statistically significant, however their clinical significance must also be considered. Understanding the magnitude of the difference between groups may provide more insight than that provided by the *p*-value, which when interpreted alone can lead to an overestimation or underestimation of the meaning of the results[237]. Indeed, clinical significance provides information regarding the importance and implications for clinical practice. In order to determine the practical value of the results, however, a definition of clinical significance must be established for the outcome variable of interest[237]. One method of defining clinical significance is by examining the effect size, which can be used to inform treatment decisions in a clinical setting[238]. In the present study, the effect

size was found to be small to moderate for the LT for the interaction effect during FE and FL. Alternatively, clinical significance can be defined by determining the minimal important difference, or the smallest difference in an outcome variable between groups that would be of interest[239]. However, no studies to date were found indicating a minimal important difference for EMG outcomes, including differences in barycentre of excitation. In the present study, the largest significant difference in $BARY_Y$ was found in the LT during FL at 120°-90°, a difference of 5.9%. With a total grid distance of 7 cm in the y-direction, a 5.9% difference results in an absolute disparity of 0.4 cm between groups for the mean location of muscle excitation. Therefore, it must be considered whether a difference of this magnitude, despite being statistically significant, can influence scapular position and motion.

8.4 Inter-Rater Reliability of SDT

The SDT was utilized in the current study as a visual assessment method for classification of scapular motion into one of three categories: normal scapular motion, subtle dyskinesis, or obvious dyskinesis[127]. Inter-rater reliability and agreement were determined for all screened participants, with a 92% agreement between raters and excellent strength of agreement when shoulder flexion and abduction ratings were combined (i.e. final rating). Several studies have previously investigated the inter-rater reliability and percentage agreement of the SDT. McClure et al. (2009)[127] found percentage agreement to range from 75% to 82% with a moderate to substantial strength of agreement with κ_w ranging from 0.48 to 0.61. Christiansen et al. (2017)[133] noted percentage agreement to be 86% with a κ_w of 0.59, while Plummer et al. (2017)[134] reported 85% agreement between raters with a κ_w of 0.69.

Our results demonstrate a higher percentage agreement and larger strength of agreement than that found in the literature. The examiners in the current study were provided the operational definitions and rating scale used by McClure and colleagues[127] which they could refer to at any point during the SDT. Scapular motion was independently rated at the time of testing, either live (i.e. in person) or by viewing a video recording. While examiners were blinded to each other's rating during the SDT, they were made aware of the other examiners rating once the SDT was complete. Further, as examiners were often in the same room it may have been possible to observe what the other examiner was focusing on. These factors may have led the examiners to learn each other's tendencies, reducing the rate of disagreement. Therefore, the control for blinding in the current study regarding the SDT may be considered inadequate.

9. Limitations and Future Directions

The current study contained several limitations which should be considered in the interpretation of the results. Regarding the study sample, recruitment was completed at a university campus where the demographic consists of primarily younger adults. The average age of participants in the study was 27 in CON and 23 in DYS, therefore results of the study cannot provide insight regarding differences in muscle excitation in an older population. Further, the results cannot be generalized to those experiencing pain as the sample population was healthy and pain-free. The study excluded those with shoulder pain in order reduce the number of confounding variables on differences in muscle excitation. However, changes in muscle excitation have previously been seen in the trapezius in response to pain[56,59,60] which may contribute to SD in some individuals. Muscle excitation in symptomatic individuals with SD should be explored in future research to determine the impact of pain on scapular kinematics. In addition, while group sizes were equal DYS contained a higher number of females compared to CON (DYS = 22/28; CON = 15/28). Fibre-type composition is believed to be similar between sexes, however females have a smaller fibre cross-sectional area than males[77,240] as well as lower relative strength in the trapezius muscle[241] that may affect the ability to control the scapula. Further, it has been theorized females may demonstrate lower motor variability which may lead to an overload of the excited fibres[67]. While sex-related differences in excitation in those with SD have not been explored in previous studies, the higher number of females in DYS may have contributed to the results of the current study.

The Scapular Dyskinesis Test (SDT) was used to determine the presence of SD for allocation into the appropriate group. Participants with SD were rated as having subtle dyskinesis or obvious dyskinesis depending on the magnitude or frequency of the abnormality. While we initially intended on having three distinct groups (i.e. control, subtle dyskinesis, obvious dyskinesis), there were difficulties in recruiting the appropriate number of individuals with obvious dyskinesis. Indeed, obvious alterations in scapular kinematics appear to be less prevalent than subtle alterations. In a study by Clarsen et al. (2014)[136], 206 male handball players were assessed for scapular dyskinesis using the SDT. The authors found subtle dyskinesis in 86 (42%) participants during flexion and 44 (21%) during abduction, while obvious dyskinesis was found in only 14 (7%) during flexion and 5 (2%) during abduction. Therefore, individuals with subtle and obvious dyskinesis in the present study were grouped together (i.e. DYS) to be analyzed.

Separating subtle and obvious individuals would have established more definitive groups and may have led to larger differences in muscle excitation between CON and the obvious dyskinesis group. Further, the SDT does not distinguish between the pattern of dyskinesis (i.e. medial/inferior border prominence, excessive elevation, dysrhythmia) present. Several patterns of dyskinesis were present in the current study which may have added further variability to the data, making differences in spatial distribution between groups less apparent. While the SDT has been shown to be valid[135] and more reliable[127,133] than methods which account for these patterns[126], generalization of scapular motion does not allow us to determine if different patterns of dyskinesis are associated with unique alterations in muscle excitation. Therefore, future studies should explore the spatial distribution of excitation within each pattern of dyskinesis.

The current study did not account for the effect fatigue may have had on the muscle excitation of the trapezius. Participants held either a 1.4 kg or 2.3 kg weight and completed five repetitions of shoulder flexion over three seconds in each direction, with repetitions separated by approximately ten seconds. Previous studies have shown changes in barycentre location[62–64] as well as scapular kinematics[124] with muscular fatigue, however we do not believe the experimental task was strenuous enough to induce muscular fatigue. Further, as the task was identical between groups any resulting fatigue would affect both groups equally. However, we did not control for participants engaging in physical activity prior to the experimental visit. Individuals may be experiencing muscular fatigue or delayed onset muscle soreness that could alter trapezius excitation and/or scapular kinematics. Future studies should ensure participants do not engage in physical activity for 48 to 72 hours minimum prior to data collection.

Handheld weights of different masses were utilized during the SDT and the experimental task – a 1.4 kg weight for participants <68.1 kg and a 2.3 kg weight for participants >68.1 kg. These relative weights were intended to provide an approximately equal amount of force required to complete the task based on a participant's bodyweight. However, in the current study a dumbbell was used for the 1.4 kg weight while a kettlebell was used for the 2.3 kg weight. As the distribution of mass for each type of weight is different, this may have affected the excitation of the muscles acting to perform the task, including the trapezius. For example, when holding a kettlebell its mass lies further from the axis of rotation (i.e. shoulder) than a dumbbell, increasing the moment arm. Therefore, participants performing the task with the kettlebell may have required greater relative muscle force, and potentially utilized different recruitment strategies

than those using the dumbbell. Future studies should ensure the type of handheld weight used for all participants is the same to reduce any confounding effects it may have.

The present study was the first to examine the differences in spatial distribution of excitation in the UT, MT, and LT in those with SD. To accomplish this, the mean barycentre of muscle excitation was quantified for each region of the trapezius. As discussed previously, while differences in barycentre are indicative of differences in excitation, they do not describe the characteristics of the distributions across the entire grid surface. To accomplish this, previous studies using HDsEMG have analyzed the entropy, or degree of uniformity, of the spatial maps[62,66]. The present study was also the first to utilize HDsEMG in those with SD for any outcome variable. Differences in muscle excitation have been found in those with SD using bi-polar EMG[44–47,49,50], however HDsEMG would allow for a more accurate representation of the amplitude of excitation within a muscle region (i.e. whole-grid RMS) or the ratio of muscle amplitudes between regions. As the present study did not record maximal voluntary contractions of each region of the trapezius prior to the experimental task, it was not possible to normalize muscle amplitude across participants to analyze these variables. Other more complex MU characteristics that contribute to EMG amplitude measures, such as the discharge rate, conduction velocity, and number of active MUs, can also be quantified using HDsEMG through decomposition of MUs[242,243]. Therefore, future studies should examine the homogeneity of excitation between groups, differences in muscle amplitude and ratio of amplitude, as well as other MU characteristics using HDsEMG as they may provide valuable insight into the contributions of the trapezius to SD.

The use of 3-dimensional kinematic measurements allows for the three rotational and two translational movements of the scapula to be quantified. The reliability and validity of these measures have been well established in the assessment of scapular motion[140,244], and their concurrent use with HDsEMG may provide further insight into the results of the current study. For example, significant differences in $BARY_Y$ were found in the LT between 90°-60° during FL. While kinematic differences were determined between groups based on the SDT, we do not know if kinematic alterations were seen in each DYS participant within this range, what the magnitude of the alterations were, or the different patterns of kinematic alterations that may have been present. Future research is needed using 3-dimensional kinematics to determine if specific deviations in scapular kinematics are associated with the selective excitation of sub-regions of

the trapezius. If associations do exist, this more robust data could be used to determine the relationship more accurately between muscle excitation, scapular kinematics, and shoulder pathology through methods such as musculoskeletal simulation and dynamic modeling programs[245]. Clinically, these associations could be the first step in determining whether the location of excitation can be altered, both acutely and chronically, using targeted rehabilitation strategies to improve scapular kinematics and reduce the risk of shoulder pathology.

10. Conclusion

The results of this study demonstrate differences in the spatial distribution of muscle excitation in the LT during the elevation and lowering phases of a shoulder flexion task between individuals with SD and healthy controls. While differing neuromuscular strategies were seen, it is unclear if the results are clinically significant as the magnitude of these differences may be too small to produce alterations in scapular kinematics. More research is needed using 3-dimensional kinematic measurements to determine the association between differences muscle excitation in the LT and alterations in scapular kinematics. Differences in excitation were not seen during either phase in the UT or MT, indicating the same motor variability found in the LT does not exist in these regions between groups. Future research should focus on characteristics of muscle excitation such as homogeneity of excitation, whole-grid muscle amplitude, ratio of excitation between regions, and active motor units in each region to further understand the contributions of the trapezius to alterations in scapular position or motion.

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Appendices

Appendix A – Research Ethics Board Approval

UNIVERSITY OF MANITOBA | Research Ethics and Compliance

Human Ethics
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PROTOCOL APPROVAL

TO: **Trisha Scribbans**
Principal Investigator

FROM: [redacted]

Re: **Protocol #E2018:026 (HS21633)**
Towards Restoring Scapular Position And Motion with Shoulder Pain: Mapping Muscle Activation In The Trapezius

Effective: April 16, 2018 **Expiry:** April 16, 2019

Education/Nursing Research Ethics Board (ENREB) has reviewed and approved the above research. ENREB is constituted and operates in accordance with the current *Tri-Council Policy Statement: Ethical Conduct for Research Involving Humans*.

This approval is subject to the following conditions:

1. Approval is granted only for the research and purposes described in the application.
2. Any modification to the research must be submitted to ENREB for approval before implementation.
3. Any deviations to the research or adverse events must be submitted to ENREB as soon as possible.
4. This approval is valid for one year only and a Renewal Request must be submitted and approved by the above expiry date.
5. A Study Closure form must be submitted to ENREB when the research is complete or terminated.
6. The University of Manitoba may request to review research documentation from this project to demonstrate compliance with this approved protocol and the University of Manitoba *Ethics of Research Involving Humans*.

Funded Protocols:
- **Please mail/e-mail a copy of this Approval, identifying the related UM Project Number, to the Research Grants Officer in ORS.**

Appendix B – Scapular Dyskinesis Test Protocol

Scapular Dyskinesis Test Protocol

This protocol is intended to act as a guide to ensure that the scapular dyskinesis test is performed in a standardized format. Ensure that you explain what each step is and why we are doing it to the participant.

Date: _____ Participant ID:_____

Step	Instructions	Complete
1	Determine the appropriate weight dumbbell based on the participant's body weight: 1.4kg (3lb) for those weighing <68.1kg (150lb) and 2.3kg (5lb) for those >68.1kg (150lb).	
2	Have the participant hold the dumbbells in a forearm neutral position (mid-way between pronation and supination) at their sides.	
3	Instruct to the participant, "Following the cadence of a metronome, I want you to raise your arms out in front of you until they are directly above your head while keeping your hands from rotating in or out. I then want you to lower them back to your sides at the same speed. You will be moving at a rate of 3 seconds per direction and performing the movement a minimum of 5 consecutive times. Next you will perform a similar protocol but raising the arms out to the side while keeping the palms facing forward. You may be required to do more repetitions to allow the examiners to properly assess the movement."	
4	As the participant performs the movements, categorize the scapular motion on their right side as "normal", "subtle abnormality" or "obvious abnormality" for each movement using the following definitions by McClure et al., 2009: *Normal scapulohumeral rhythm*: The scapula is stable with minimal motion during the initial 30° to 60° of humerothoracic elevation, then smoothly and continuously rotates upward during elevation and smoothly and continuously rotates downward during humeral lowering. No evidence of winging is present. *Scapular dyskinesis*: Either or both of the following motion abnormalities may be present. 1) Dysrhythmia: The scapula demonstrates premature or excessive elevation or protraction, non-smooth or stuttering motion during arm elevation or lowering, or rapid downward rotation during arm lowering. 2) Winging: The medial border and/or inferior angle of the scapula are posteriorly displaced away from the posterior thorax. *Subtle:* Mild or questionable evidence of abnormality, not consistently present *Obvious:* Striking, clearly apparent abnormality, evident on at least 3/5 trials	
5	Final rating based on a combination of flexion and abduction test movements is defined as: *Normal:* Both test motions are rated as normal or 1 motion is normal and the other is rated as a subtle abnormality. *Subtle:* Both flexion and abduction are rated as having subtle abnormalities. *Obvious:* Either flexion or abduction (or both) is rated as having obvious abnormality.	
6	For participants with obvious or subtle scapular dyskinesis, further group them into one of the 3 categories for scapular dyskinesis using the following definitions based on Kibler et al, 2002: *Type I: Inferior Angle* - At rest, the inferior medial scapular border may be prominent dorsally. During arm motion, the inferior angle tilts dorsally and the acromion tilts ventrally over the top of the thorax. The axis of the rotation is in the horizontal plane.	

	Type II: Medial Border - At rest, the entire medial border may be prominent dorsally. During arm motion, the medial scapular border tilts dorsally off the thorax. The axis of the rotation is vertical in the frontal plane. *Type III: Superior Border* - At rest, the superior border of the scapula may be elevated and the scapula can also be anteriorly displaced. During arm motion, a shoulder shrug initiates movement without significant winging of the scapular occurring. The axis of this motion occurs in the sagittal plane.	
6	Once two examiners have assessed scapular motion results will be compared. Participants rated as "normal" by both examiners will be allocated to the control group and those rated as "subtle" or "obvious" by both examiners will be allocated to the dyskinesis group. Participants will be excluded from the study if their final ratings differ by examiner.	
7	Thank all participants for their time. If the participant is eligible to participate in the study, move to the first step of the experimental visit.	

Scapular motion - flexion:

Normal ☐ *Subtle abnormality* ☐ *Obvious abnormality* ☐

Scapular motion - abduction:

Normal ☐ *Subtle abnormality* ☐ *Obvious abnormality* ☐

Final rating:

Normal ☐ *Subtle abnormality* ☐ *Obvious abnormality* ☐

Type of scapular dyskinesis (if applicable):

Type I ☐ *Type II* ☐ *Type III* ☐

Brief description of position/rhythm: _____

_____

_____

_____.

Examiner: _____

Appendix C – SDT Rating of Analyzed Participants (N = 56)

		Rater 1				Rater 2		
Group	**Participant**	**FLEX**	**ABD**	**FINAL**		**FLEX**	**ABD**	**FINAL**
CON	**P04**	Subtle	Normal	**Normal**		Subtle	Normal	**Normal**
	P05	Normal	Subtle	**Normal**		Subtle	Normal	**Normal**
	P06	Subtle	Normal	**Normal**		Normal	Normal	**Normal**
	P08	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P09	Normal	Subtle	**Normal**		Normal	Normal	**Normal**
	P12	Subtle	Normal	**Normal**		Subtle	Normal	**Normal**
	P13	Normal	Normal	**Normal**		Normal	Subtle	**Normal**
	P16	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P17	Normal	Subtle	**Normal**		Normal	Normal	**Normal**
	P18	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P19	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P20	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P21	Subtle	Normal	**Normal**		Subtle	Normal	**Normal**
	P27	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P28	Subtle	Normal	**Normal**		Subtle	Normal	**Normal**
	P29	Subtle	Normal	**Normal**		Subtle	Normal	**Normal**
	P31	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P33	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P35	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P36	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P37	Subtle	Normal	**Normal**		Normal	Normal	**Normal**
	P38	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P39	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P41	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P42	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P43	Normal	Normal	**Normal**		Normal	Normal	**Normal**
	P44	Subtle	Normal	**Normal**		Subtle	Normal	**Normal**
	P45	Subtle	Normal	**Normal**		Normal	Normal	**Normal**
Group	**Participant**	**FLEX**	**ABD**	**FINAL**		**FLEX**	**ABD**	**FINAL**
DYS	**P02**	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P10	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P11	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P14	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P15	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P22	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P23	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P24	Obvious	Subtle	**Obvious**		Obvious	Subtle	**Obvious**
	P30	Subtle	Subtle	**Subtle**		Subtle	Subtle	**Subtle**
	P40	Obvious	Normal	**Obvious**		Obvious	Subtle	**Obvious**

	P46	Subtle	Obvious	**Obvious**	Subtle	Obvious	**Obvious**
	P47	Obvious	Subtle	**Obvious**	Obvious	Normal	**Obvious**
	P49	Obvious	Subtle	**Obvious**	Obvious	Normal	**Obvious**
	P51	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P52	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P53	Obvious	Subtle	**Obvious**	Obvious	Subtle	**Obvious**
	P54	Obvious	Subtle	**Obvious**	Obvious	Subtle	**Obvious**
	P55	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P56	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P57	Obvious	Subtle	**Obvious**	Obvious	Subtle	**Obvious**
	P58	Obvious	Subtle	**Obvious**	Obvious	Normal	**Obvious**
	P59	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P60	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P61	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**
	P62	Obvious	Subtle	**Obvious**	Obvious	Obvious	**Obvious**
	P63	Obvious	Subtle	**Obvious**	Obvious	Normal	**Obvious**
	P64	Obvious	Normal	**Obvious**	Obvious	Normal	**Obvious**
	P65	Subtle	Subtle	**Subtle**	Subtle	Subtle	**Subtle**

FLEX- shoulder flexion; ABD- shoulder abduction; FINAL- combined rating from shoulder flexion and abduction.

***Appendix D – Mean Barycentre Y-Coordinate Value* (*N* = 56)**

Upper Trapezius – Elevation and Lowering Phases (grid %)

Outlier values are bolded

Group	Participant	30° - 60°	60° - 90°	90° - 120°	120° - 90°	90° - 60°	60° - 30°
CON	**P04**	57.6	58.4	59.9	59.3	59.6	59.0
	P05	66.6	67.3	65.2	63.2	66.4	65.4
	P06	65.0	64.2	65.4	66.7	63.6	61.5
	P08	63.3	63.0	62.2	62.6	62.3	62.0
	P09	63.6	62.8	62.1	62.1	61.4	62.4
	P12	58.1	56.3	57.3	56.9	56.3	56.8
	P13	56.3	56.3	56.3	52.3	51.4	49.0
	P16	52.1	52.2	51.5	52.7	52.4	52.2
	P17	53.6	52.8	53.5	54.8	57.0	60.4
	P18	48.9	48.5	48.3	49.8	48.5	49.4
	P19	56.6	55.4	54.6	54.6	55.9	55.5
	P20	58.7	58.5	58.8	60.3	59.7	59.6
	P21	56.7	56.1	55.9	58.2	58.4	53.7
	P27	53.2	49.8	49.8	47.8	47.3	47.5
	P28	62.9	60.5	59.0	62.0	62.1	63.9
	P29	56.2	54.3	50.3	56.0	56.6	56.5
	P31	52.0	51.0	50.8	52.0	52.2	51.6

	P33	58.9	57.3	56.5	61.0	60.4	58.9
	P35	56.3	55.7	55.9	53.4	52.3	54.7
	P36	57.3	55.1	54.3	52.6	54.0	54.2
	P37	50.3	50.0	49.2	49.9	49.2	49.5
	P38	54.3	49.1	46.7	46.9	48.3	50.8
	P39	54.9	54.0	52.2	53.5	52.7	51.4
	P41	56.7	54.4	50.7	51.0	53.1	53.5
	P42	55.5	53.5	52.9	52.8	52.7	54.4
	P43	50.5	47.5	44.1	46.3	47.1	48.8
	P44	49.9	48.2	49.4	49.3	50.0	50.2
	P45	56.4	53.3	53.7	52.1	53.0	55.7
DYS	**P02**	56.2	56.3	55.9	57.7	57.4	57.1
	P10	57.6	57.3	58.9	58.8	59.6	60.1
	P11	57.4	55.5	54.1	56.2	57.7	60.7
	P14	66.7	62.8	60.6	58.8	57.8	62.1
	P15	53.1	52.6	53.4	54.1	52.6	51.2
	P22	58.8	58.4	57.9	57.1	57.0	57.9
	P23	54.0	52.6	53.7	53.7	53.8	55.6
	P24	63.5	59.6	57.0	55.5	57.6	61.3
	P30	53.9	54.0	55.1	56.8	55.5	53.5
	P40	59.1	57.7	55.1	58.2	58.2	59.8
	P46	49.3	47.8	47.8	49.5	49.6	50.3
	P47	45.6	41.0	40.4	**39.3**	40.1	43.9
	P49	59.1	57.3	53.5	53.5	57.6	60.2
	P51	60.9	54.9	52.8	53.8	55.0	60.5
	P52	59.7	59.9	59.8	60.0	60.4	58.8
	P53	63.9	62.2	60.9	63.7	64.2	64.3
	P54	52.3	54.3	58.2	51.7	50.0	54.9
	P55	56.1	55.2	56.1	58.7	58.2	60.7
	P56	53.5	51.1	50.0	49.7	51.5	51.2
	P57	**73.2**	**73.7**	**72.9**	71.3	71.0	69.5
	P58	55.3	54.6	54.4	57.6	59.0	58.7
	P59	52.3	51.9	49.9	52.4	52.3	52.6
	P60	58.6	58.5	58.5	54.9	55.2	55.7
	P61	54.9	54.5	54.2	52.0	49.5	49.4
	P62	61.5	59.6	59.3	59.5	61.3	61.9
	P63	62.0	61.2	60.1	63.7	66.6	66.3
	P64	53.8	51.6	51.9	52.4	52.2	53.1
	P65	56.4	55.4	54.9	54.4	54.5	55.9

CON- control group; DYS- dyskinesis group

Middle Trapezius – Elevation and Lowering Phases (grid %)

Outlier values are bolded

Group	Participant	30° - 60°	60° - 90°	90° - 120°	120° - 90°	90° - 60°	60° - 30°
CON	**P04**	47.4	44.5	39.9	45.1	47.0	45.0
	P05	44.1	43.4	42.1	38.2	38.0	35.7
	P06	58.4	66.1	61.4	58.4	67.8	66.8
	P08	41.1	41.0	41.2	39.2	43.5	44.3
	P09	60.8	62.9	60.1	50.6	51.5	48.9
	P12	44.6	50.2	49.8	52.2	53.4	47.4
	P13	68.0	69.7	71.3	69.1	66.4	62.0
	P16	63.8	63.9	65.2	64.4	61.6	57.1
	P17	63.9	61.8	56.8	55.4	52.0	42.2
	P18	45.2	48.0	46.0	45.8	46.4	45.7
	P19	64.9	67.2	66.4	68.0	67.3	67.1
	P20	64.3	62.3	58.0	47.8	54.5	51.8
	P21	74.1	74.9	73.1	62.3	64.9	60.8
	P27	51.7	57.2	58.5	67.0	66.3	62.2
	P28	65.3	65.5	66.4	66.7	65.0	60.0
	P29	67.2	68.0	68.3	63.5	61.2	61.8
	P31	53.4	56.5	54.7	54.1	53.0	49.8
	P33	70.0	67.6	61.4	58.5	60.7	64.3
	P35	66.5	62.9	53.5	58.0	66.4	63.1
	P36	71.9	70.7	67.6	68.0	67.9	66.3
	P37	69.6	70.1	72.6	69.6	68.2	65.6
	P38	57.2	60.2	61.8	62.5	60.8	57.5
	P39	66.9	67.5	65.3	61.6	64.5	58.6
	P41	43.0	40.5	36.5	36.2	37.1	36.3
	P42	58.4	60.8	59.8	60.5	60.0	54.7
	P43	58.4	59.6	62.5	61.3	58.9	55.2
	P44	69.1	70.7	71.3	69.3	68.3	65.5
	P45	59.5	55.0	54.1	60.1	62.0	55.1
DYS	**P02**	44.7	44.4	40.8	42.6	40.6	40.9
	P10	65.1	65.8	59.7	59.1	56.8	54.8
	P11	67.0	66.1	65.4	65.1	60.9	54.3
	P14	50.6	57.5	53.5	52.9	52.4	42.6
	P15	64.2	65.3	67.4	64.1	61.8	55.0
	P22	61.7	58.7	53.1	54.4	54.9	50.3
	P23	65.9	67.2	67.9	68.6	65.5	60.2
	P24	51.1	51.8	47.1	44.9	50.2	49.7
	P30	70.0	68.4	64.9	57.6	63.7	65.8
	P40	71.4	71.9	70.4	67.9	70.2	68.1
	P46	58.7	60.9	62.4	62.8	60.9	55.8

	P47	64.1	67.2	70.3	70.9	67.5	64.0
	P49	57.6	56.9	53.1	52.6	53.2	48.1
	P51	63.7	67.4	69.4	72.2	70.7	65.2
	P52	56.8	57.5	55.0	56.7	54.3	51.9
	P53	71.9	74.3	76.0	63.4	65.7	63.1
	P54	70.5	70.5	71.0	71.9	69.7	56.2
	P55	61.5	61.6	58.0	61.4	60.8	57.0
	P56	59.5	60.6	58.6	53.9	50.0	47.4
	P57	37.3	37.6	38.8	38.9	37.2	36.9
	P58	60.1	58.6	57.4	56.4	57.7	58.9
	P59	60.0	58.7	56.5	57.2	56.4	52.2
	P60	59.4	61.0	61.2	59.5	60.3	59.5
	P61	71.3	71.4	69.7	70.0	70.0	68.7
	P62	65.9	67.9	65.5	65.6	67.2	61.6
	P63	70.8	66.2	59.8	62.7	61.7	63.8
	P64	56.1	56.0	57.0	55.8	54.8	52.1
	P65	64.5	66.0	66.3	67.3	66.4	62.1

CON- control group; DYS- dyskinesis group

Lower Trapezius – Elevation and Lowering Phases (grid %)

Outlier values are bolded

Group	**Participant**	**30° - 60°**	**60° - 90°**	**90° - 120°**	**120° - 90°**	**90° - 60°**	**60° - 30°**
CON	**P04**	38.2	38.0	39.0	38.2	39.5	42.7
	P05	46.5	44.9	45.8	46.5	46.5	46.2
	P06	26.3	25.5	26.0	25.6	25.3	26.4
	P08	46.4	45.3	46.4	42.8	41.9	42.0
	P09	37.8	39.8	41.2	42.5	40.5	38.9
	P12	37.0	36.7	38.6	40.0	38.4	38.1
	P13	35.5	35.0	36.2	38.8	37.1	39.1
	P16	38.6	39.3	39.9	39.1	37.5	35.4
	P17	30.7	30.8	30.4	30.4	31.9	39.1
	P18	43.4	43.3	45.6	45.5	44.4	44.1
	P19	33.4	33.6	33.9	34.0	33.5	33.7
	P20	37.6	35.8	36.3	37.3	37.3	35.8
	P21	27.4	27.8	29.1	30.8	29.5	34.0
	P27	35.1	34.5	35.2	35.9	35.3	36.3
	P28	39.0	41.8	41.2	41.4	41.0	38.3
	P29	40.5	43.6	47.0	49.9	46.0	42.7
	P31	45.1	43.6	45.2	45.5	45.7	47.2
	P33	22.6	22.8	22.9	23.9	25.0	30.4
	P35	37.0	35.4	34.6	34.2	33.4	38.7
	P36	30.3	30.4	29.9	29.8	30.2	29.3
	P37	28.7	29.1	29.4	30.9	30.9	32.5

	P38	33.5	33.0	33.4	34.1	34.5	36.5
	P39	32.5	30.6	28.8	31.8	34.4	44.8
	P41	27.4	28.0	30.8	31.7	35.0	40.0
	P42	33.3	33.8	35.6	34.4	34.5	37.9
	P43	45.8	46.7	45.6	45.1	45.1	44.4
	P44	30.5	28.5	28.5	30.3	31.8	35.0
	P45	25.9	25.7	22.8	25.6	26.0	31.8
DYS	**P02**	38.9	38.7	39.1	38.9	39.2	43.3
	P10	39.1	38.7	40.4	40.0	38.9	37.4
	P11	37.4	38.9	39.5	37.7	36.9	35.1
	P14	42.1	41.2	41.6	42.7	43.6	44.4
	P15	28.3	28.8	28.3	28.7	29.5	34.6
	P22	32.1	30.4	32.5	31.1	33.9	38.6
	P23	40.5	41.7	41.6	42.4	40.1	39.8
	P24	51.1	**52.5**	52.5	**54.1**	51.3	47.2
	P30	25.2	25.0	24.2	25.1	25.5	27.7
	P40	25.7	26.2	25.9	27.4	27.7	27.1
	P46	35.6	32.6	31.5	34.0	37.0	43.4
	P47	24.7	24.1	23.6	22.9	24.2	26.9
	P49	21.6	21.4	21.9	22.2	24.3	33.1
	P51	27.1	24.6	24.1	24.7	26.4	34.6
	P52	23.5	22.4	21.1	21.9	24.4	29.5
	P53	24.7	24.4	23.9	25.3	25.6	28.9
	P54	37.4	35.5	33.2	34.6	40.0	44.5
	P55	35.6	35.2	35.2	33.9	37.8	42.9
	P56	29.6	28.3	27.6	31.1	34.3	41.2
	P57	38.6	40.2	40.0	40.3	39.8	39.4
	P58	21.4	20.8	20.8	20.7	21.8	21.5
	P59	21.8	22.3	22.0	21.1	22.7	22.4
	P60	21.7	21.2	21.4	21.0	21.9	23.7
	P61	28.5	27.9	27.3	29.2	31.0	38.7
	P62	32.5	28.7	27.5	29.4	31.5	37.7
	P63	18.7	19.4	20.4	19.3	19.2	20.1
	P64	27.1	26.6	25.6	26.1	27.2	29.6
	P65	27.0	24.8	24.0	25.4	29.8	43.9

CON- control group; DYS- dyskinesis group

***Appendix E – Distribution Scores* (*N* = 56)**

Grid/Task	Angle	Group	Shapiro-Wilk *p* -value	Skewness	Skewness SE	Skewness z-score	Kurtosis	Kurtosis SE	Kurtosis z-score
UT_FE	30° - 60°	CON	0.227	0.466	0.441	1.057	-0.147	0.858	-0.171
		DYS	0.502	0.616	0.441	1.398	1.608	0.858	1.874
	60° - 90°	CON	0.338	0.579	0.441	1.315	-0.016	0.858	-0.019
		DYS	0.048	0.400	0.441	0.908	3.656	0.858	4.259
	90° - 120°	CON	0.801	0.328	0.441	0.744	-0.325	0.858	-0.378
		DYS	0.022	0.285	0.441	0.647	3.979	0.858	4.636
UT_FL	120° - 90°	CON	0.298	0.389	0.441	0.884	-0.723	0.858	-0.842
		DYS	0.074	-0.099	0.441	-0.225	3.174	0.858	3.698
	90° - 60°	CON	0.334	0.325	0.441	0.738	-0.822	0.858	-0.958
		DYS	0.290	-0.055	0.441	-0.124	1.979	0.858	2.305
	60° - 30°	CON	0.310	0.342	0.441	0.775	-0.924	0.858	-1.077
		DYS	0.943	-0.198	0.441	-0.448	0.313	0.858	0.364
MT_FE	30° - 60°	CON	0.035	-0.575	0.441	-1.305	-0.866	0.858	-1.009
		DYS	0.023	-1.132	0.441	-2.570	1.642	0.858	1.912
	60° - 90°	CON	0.025	-0.782	0.441	-1.776	-0.350	0.858	-0.408
		DYS	0.020	-1.211	0.441	-2.749	2.079	0.858	2.422
	90° - 120°	CON	0.088	-0.668	0.441	-1.516	-0.369	0.858	-0.430
		DYS	0.218	-0.698	0.441	-1.583	0.349	0.858	0.407
MT_FL	120° - 90°	CON	0.022	-0.789	0.441	-1.791	-0.328	0.858	-0.382
		DYS	0.203	-0.679	0.441	-1.541	0.198	0.858	0.231
	90° - 60°	CON	0.005	-0.927	0.441	-2.105	-0.093	0.858	-0.108
		DYS	0.086	-0.843	0.441	-1.913	0.675	0.858	0.786
	60° - 30°	CON	0.057	-0.616	0.441	-1.399	-0.612	0.858	-0.713
		DYS	0.576	-0.494	0.441	-1.123	-0.199	0.858	-0.231
LT_FE	30° - 60°	CON	0.518	0.097	0.441	0.221	-0.734	0.858	-0.856
		DYS	0.130	0.628	0.441	1.426	-0.101	0.858	-0.117
	60° - 90°	CON	0.430	0.089	0.441	0.203	-1.014	0.858	-1.182
		DYS	0.026	0.881	0.441	2.001	0.310	0.858	0.361
	90° - 120°	CON	0.194	0.011	0.441	0.024	-0.998	0.858	-1.163
		DYS	0.006	0.929	0.441	2.109	0.180	0.858	0.210
LT_FL	120° - 90°	CON	0.617	0.123	0.441	0.279	-0.798	0.858	-0.929
		DYS	0.050	0.893	0.441	2.028	0.552	0.858	0.644
	90° - 60°	CON	0.404	0.016	0.441	0.036	-0.719	0.858	-0.837
		DYS	0.228	0.485	0.441	1.100	-0.342	0.858	-0.399
	60° - 30°	CON	0.925	-0.179	0.441	-0.405	-0.405	0.858	-0.472
		DYS	0.137	-0.339	0.441	-0.769	-1.069	0.858	-1.245

UT_FE- upper trapezius shoulder flexion (elevation); UT_FL- upper trapezius shoulder flexion (lowering); MT_FE- middle trapezius shoulder flexion (elevation); MT_FL- middle trapezius shoulder flexion (lowering); LT_FE- lower trapezius shoulder flexion (elevation); LT_FL- lower trapezius shoulder flexion (lowering); SE- standard error.

***Appendix F – Results of Two-Way Between-Within ANOVAs* (*N* = 56)**

	df (a*g)	df (error)	*f*	*p*-value	η_p^2	Power
UT_FE	1.230	66.432	.077	.833	.001	.059
UT_FL	1.370	73.998	2.887	.081	.051	.454
MT_FE	1.269	68.515	.015	.942	<.001	.052
MT_FL	1.337	72.189	1.576	.217	.028	.268
LT_FE	1.336	72.136	4.635	.025*	.079	.646
LT_FL	1.069	57.740	4.272	.041*	.073	.546

UT_FE- upper trapezius shoulder flexion (elevation); UT_FL- upper trapezius shoulder flexion (lowering); MT_FE- middle trapezius shoulder flexion (elevation); MT_FL- middle trapezius shoulder flexion (lowering); LT_FE- lower trapezius shoulder flexion (elevation); LT_FL- lower trapezius shoulder flexion (lowering); df (a*g)- degrees of freedom of interaction term (angle by group); df (error)- degrees of freedom of error term; *f*- value of *f*-statistic; η_p^2- effect size (partial η^2)

* indicates a significant group by angle interaction on barycentre coordinate between groups ($p < .05$)

***Appendix G – Mean and Standard Deviations of $BARY_Y$ (% of grid from origin)* [*N* = 56]**

	Group	30°-60° (SD)	60°-90° (SD)	90°-120° (SD)
UT_FE	CON	56.5 (4.6)	55.2 (5.1)	54.5 (5.4)
	DYS	57.5 (5.5)	56.1 (5.7)	55.6 (5.6)
MT_FE	CON	59.6 (9.7)	60.3 (9.7)	58.8 (10.3)
	DYS	61.5 (8.2)	62.0 (8.0)	60.6 (9.0)
LT_FE	CON	35.2 (6.6)	35.1 (6.7)	35.7 (7.3)
	DYS	30.6 (7.9)	30.1 (8.2)	29.9 (8.4)
		120°-90° (SD)	**90°-60° (SD)**	**60°-30° (SD)**
UT_FL	CON	55.0 (5.4)	55.1 (5.3)	55.3 (5.1)
	DYS	55.9 (5.7)	56.3 (5.9)	57.4 (5.5)
MT_FL	CON	57.6 (9.9)	58.4 (9.3)	55.4 (9.3)
	DYS	59.9 (8.6)	59.3 (8.5)	55.9 (8.2)
LT_FL	CON	36.3 (6.9)	36.1 (6.2)	37.9 (5.3)
	DYS	30.4 (8.5)	31.6 (7.9)	34.9 (7.9)

$BARY_Y$- barycentre y-coordinate; UT_FE- upper trapezius shoulder flexion (elevation); UT_FL- upper trapezius shoulder flexion (lowering); MT_FE- middle trapezius shoulder flexion (elevation); MT_FL- middle trapezius shoulder flexion (lowering); LT_FE- lower trapezius shoulder flexion (elevation); LT_FL- lower trapezius shoulder flexion (lowering).

***Appendix H – Between-Group Differences in $BARY_Y$ (% of grid from origin)* [*N* = 56]**

	30°-60°	**60°-90°**	**90°-120°**
UT_FE	.9	.9	1.1
MT_FE	1.9	1.7	1.8
LT_FE	-4.6*	-5.0*	-5.8*
	120°-90°	**90°-60°**	**60°-30°**
UT_FL	.9	1.1	2.1
MT_FL	2.2	1.0	.6
LT_FL	-5.9*	-4.5*	-3.0

$BARY_Y$- barycentre y-coordinate; UT_FE- upper trapezius shoulder flexion (elevation); UT_FL- upper trapezius shoulder flexion (lowering); MT_FE- middle trapezius shoulder flexion (elevation); MT_FL- middle trapezius shoulder flexion (lowering); LT_FE- lower trapezius shoulder flexion (elevation); LT_FL- lower trapezius shoulder flexion (lowering).

Group Difference = DYS barycentre – CON barycentre

Positive value indicates DYS barycentre located caudally relative to CON; negative value indicates DYS barycentre located cranially relative to CON

* indicates a significant difference in mean barycentre coordinate between groups ($p < .05$)

***Appendix I – Mean and Standard Deviations of $BARY_Y$ (% of grid from origin) – Groups Combined* [*N* = 56]**

	30°-60° (SD)	**60°-90° (SD)**	**90°-120° (SD)**
UT_FE	57.0 (5.1)	55.7 (5.4)	55.1 (5.5)
MT_FE	60.5 (9.0)	61.2 (8.9)	59.7 (9.6)
LT_FE	32.9 (7.6)	32.6 (7.9)	32.8 (8.3)
	120°-90° (SD)	**90°-60° (SD)**	**60°-30° (SD)**
UT_FL	55.5 (5.5)	55.7 (5.6)	56.4 (5.4)
MT_FL	58.8 (9.3)	58.9 (8.8)	55.7 (8.7)
LT_FL	33.3 (8.2)	33.9 (7.4)	36.4 (6.8)

$BARY_Y$- barycentre y-coordinate; UT_FE- upper trapezius shoulder flexion (elevation); UT_FL- upper trapezius shoulder flexion (lowering); MT_FE- middle trapezius shoulder flexion (elevation); MT_FL- middle trapezius shoulder flexion (lowering); LT_FE- lower trapezius shoulder flexion (elevation); LT_FL- lower trapezius shoulder flexion (lowering).