Contralateral Effects of Unilateral Pectoral Stretching

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Abstract

Context: Pathological conditions limiting range of motion (ROM) or requiring immobilization can result in muscle atrophy, decreases in ROM and decreased strength. Unilateral static stretching (SS) is often used in conjunction with strength training to mitigate the ROM and strength losses related to these conditions. Adaptations are believed to result from cross-over of the sensory and mechanical effects of SS, though this has solely been examined in the lower limb. No studies have examined the effects of unilateral SS in the upper limb. More specifically, there has been no investigation into how unilateral SS can reduce the risk of pectoral shortening, resulting in forward shoulder posture (FSP) and altered scapular kinematics, increasing injury risk. One study has demonstrated high-intensity SS of the pectorals can decrease pectoralis minor muscle stiffness (PMin SWV) of the stretched limb, but these effects have not been compared to stretching at lower intensities nor have they been examined in the contralateral (CL) limb or on pectoralis major stiffness (PMaj SWV). To fully understand the effects of unilateral pectoral SS, it is important to examine its effects on horizontal abduction ROM (HaROM), FSP, pectoralis minor length (PML), PMin SWV and PMaj SWV. As lower intensity stretching is more tolerable for the individual, it is also important to understand whether its effects on these variables mimic those of higher intensity stretching. **Objective**: The proposed project will examine the CL effects of unilateral pectoral stretching at two intensities on PMaj SWV, PMin SWV, FSP, PML and HaROM. Additionally, we examined the effect of changes in intensity and HaROM in the intervention limb on the CL limb. **Methods**: A convenience sample of 30 participants completed two sessions of unilateral pectoral SS on their dominant limb at two stretching intensities (moderate (MOD) and maximal (MAX)) in addition to a control condition (CON). The CON consisted of a 150s rest period to mimic the length of the stretch conditions. Each participant performed three sets of 30-seconds with a 30-second rest between each rep at each intensity with PMaj SWV, PMin SWV, FSP, PML and HaROM being measured on both limbs prior to and immediately following each intervention. **Results:** Patient-administered SS at a MOD and MAX intensity increased HaROM, with MAX exhibiting a greater change than MOD. For FSP, PMaj and PML showed no significant change for each condition but there was a significant difference between sexes at baseline for each of these variables, demonstrating structural differences between males and females. PMin SWV increased during the CON and MOD conditions but remained unchanged in the MAX condition. There were also significant baseline differences in PMin SWV between all three conditions. ANOVAs were repeated on the percent change scores for each variable to eliminate the effect of baseline differences. Differences between all conditions were found for HaROM and PMin SWV. Stretch intensity decreased from the start of the first repetition to then end of the last repetition in both conditions with a greater decrease in the MAX condition than MOD. The HaROM and intensity change score of the intervention limb was effective at predicting change in the MAX condition but not the MOD condition.
Conclusions: These findings demonstrate that unilateral pectoral SS is effective at improving HaROM in the CL limb at a MOD and MAX intensity, though MAX intensity may be more favourable as it elicits greater improvements in HaROM. Acutely, this method of stretching is not effective in reducing PMaj or PMin SWV, PML and FSP, though the chronic adaptations remain unknown. Future research should seek to further examine PMin SWV profiles to better understand the stiffness adaptations observed in this study. The application of this research to a clinical population to further examine the effectiveness of unilateral pectoral SS in preserving ROM in a pathological limb. Collectively, the findings of the current study support the used of unilateral pectoral SS at a high intensity as part of a holistic rehabilitation protocol.

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Dedication

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List of Abbreviations

ACSM – American College of Sports Medicine
CL – Contralateral
CON – Control Condition
FSP – Forward Shoulder Posture
HaROM – Horizontal Abduction Range of Motion
PMaj – Pectoralis Major
PMin – Pectoralis Minor
PML – Pectoralis Minor Length
ROI – Region of Interest
ROM – Range of Motion
SS – Static Stretching
SWE – Shear Wave Elastography
SWV – Shear Wave Velocity

1.Introduction

Stretching is a common modality used in rehabilitation and physical performance settings to prepare tissues for movement, increase ROM (ROM), decrease muscle stiffness and assist in tissue healing. Though there are many modes of stretching, static stretching (SS) is recommended by the American College of Sports Medicine (ACSM) as a key component of the flexibility dimension of fitness [1]. SS can be performed either actively or passively and involves bringing a muscle or group of muscles into a lengthened position and then maintaining that position for a fixed amount of time [2,3]. Significant research has been conducted on the effects of SS, with the most well-documented outcome being increases in ROM [4-7]. The literature does not clearly identify the underlying mechanisms behind the effects of SS, though they do propose two main theories explaining the effects: the sensory and mechanical theories.

The mechanical theory examines how proposed changes in the physiological properties of muscles may increase muscle length, including adaptations in viscoelasticity, neuromuscular relaxation, and decreased stiffness [8, 9]. In this theory, the increase in muscle length is the main cause of increased ROM following stretching due to adaptations in stiffness [9-11]. The sensory theory examines how an increase in ROM may be due to increased stretch tolerance [12-15]. As ROM increases have been documented in both the presence and absence of mechanical alterations, the sensory theory may provide a more holistic understanding of how SS increases ROM. The adaptations resulting from these theories are typically dependent on the dosage of SS.

Numerous modifiable factors influence SS dosage, including duration, number of repetitions, and intensity [4-7]. The most recognized SS dosage is two to four sets of 10-30-second holds, for a cumulative total of 60 seconds per muscle group [1]. Though this recommendation is from the ACSM, stretching is often prescribed in a wide variety of combinations of time,

repetitions, and intensity. The most common factors that are modified are duration and repetitions (objective measures) and intensity (subjective measure) [4-7]. The subjective nature of intensity makes it very difficult to compare individuals and populations in research [16]. Additionally, a discrepancy exists regarding the measures used to define intensity, as there is no validated tool or scale used globally to evaluate an individual's perceived stretch intensity [17].

Of the available research, there is no single combination of dosing factors that consistently increases ROM over another. The limited research on stretch-intensity to date suggests that both high- and low-intensity SS elicit the same degree of ROM increase [3,12,18,19]. Both high- and low-intensity stretching of the lower limb have been shown to increase passive torque, decrease muscle stiffness, and increase ROM [3,12,18,19]. Higher-intensity stretching generally results in greater discomfort for the individual, increases musculotendinous unit stiffness, and triggers an inflammatory response [18, 19]. Clinically, this leads to lower-intensity stretching being prescribed more broadly, as high-intensity stretching appears to provide no additional benefit(s) for most populations [15]. To the author's knowledge, there are no studies to date that examine the effects of SS in the upper limb even though it is structurally and functionally different than the lower limb. The anatomical differences should be considered when evaluating the effectiveness of a SS protocol.

The shoulder girdle is a complex structure comprising four joints and various soft tissues that can affect its structure and function. Of the four joints, three are synovial (sternoclavicular, acromioclavicular, and glenohumeral) and one is "false" (scapulothoracic) [20]. The joints, muscles, and connective tissues within the complex allow for extensive multi-directional movement of the upper limb and thorax. The high degree of freedom of movement at the joint makes it structurally unstable and requires significant dynamic stability from the surrounding

musculature [21,22]. The reliance on dynamic stabilization leaves the complex susceptible to various pathologies [21,22]. Furthermore, the connection of both shoulder complexes to the trunk from the joints and soft tissues can increase the risk of pathology in the unaffected shoulder following an injury [23]. The interconnection also results in postural abnormalities that generally affect both shoulders, such as forward shoulder posture (FSP) [24,25].

The most well-documented postural abnormality is FSP [26]. FSP is generally defined as anterior deviation and abduction of the scapula resulting from shortened pectoral muscles. The pectoral muscles consist of the pectoralis major (PMaj), which has three regions (clavicular, sternal and costal) that connect the proximal humerus to the trunk and the pectorals minor (PMin) that extends from the coracoid process to the 3rd-5th ribs [20]. The direct attachments of these muscles from the trunk to the humerus and scapula allow for anterior displacement of the humerus and protraction of the scapula when the muscles are shortened [26]. The altered position of the scapula results in changes in scapular kinematics that may predispose an individual to shoulder pathologies [25]. Interventions aimed at lengthening the pectoral musculature to reduce the injury risk can assist in shoulder rehabilitation protocols and address postural abnormalities.

A common modality used in a clinical setting to increase pectoral length is SS. The purpose of this modality is to elongate the pectoral musculature, reduce FSP, reduce injury risk or rehabilitate shoulder injuries [27-29]. Though many studies have examined long-term SS and therapeutic interventions that utilize a combination of modalities, there are no studies that have examined the acute response to SS or the CL response to unilateral SS. One study examined the effect of pectoral SS at different stretching positions on pectoralis minor length (PML) [30]. The same research group has also examined the effect of pectoral stretching on PMin stiffness and scapular kinematics [31, 32]. From these projects, the research group determined that positioning the

shoulder at 90° and 150° of horizontal abduction and stretching to a point of discomfort [31] effectively lengthens the PMin [30] in as little as one 30-second repetition. Additionally, stretching for a cumulative 5 minutes increased scapular external rotation and posterior tilt [32]. No other variables, such as ROM, FSP, PMaj stiffness, or CL effects, were examined in these studies, leaving knowledge gaps that warrant further investigation.

In a clinical setting, unilateral pectoral stretching is commonly prescribed to help preserve ROM in a limb affected by pathology. Given the anatomical implications of how the pectorals attach to the thorax and the connective tissue that expands the chest and shoulder girdle, it is important to understand how unilateral stretching may impact the CL joint and musculature. Researchers have reported that unilateral SS increases ROM and decreases muscle stiffness [33, 34]. The resulting adaptations are believed to be closely related to sensory adaptations [33, 34] rather than to mechanical adaptations [35-38]. The sensory adaptations are theorized to result from the spillover effect, with adaptations occurring at the cortical, subcortical, and spinal network levels [33, 34]. Additionally, a global increase in stretch tolerance is believed to contribute to increased ROM in the CL musculature [39]. As in SS research, all studies examining CL SS have been conducted on the lower-limb musculature [33, 40, 41]. Though the research is very limited, two studies examining the CL increases in ROM found that unilateral stretching can increase the ROM of the CL musculature by 4-8%[33, 40]. As no studies have examined the effectiveness of unilateral stretching protocols in the upper limb, it is unknown whether these findings apply to this region. Thus, the purpose of this work is to examine the effects of unilateral SS on the pectoral musculature by evaluating the CL effects on FSP, PML, HaROM, PMaj shear wave velocity (SWV) and pectoralis minor (PMin) SWV.

1. Literature Review

The purpose of the master's thesis project is to examine the acute response of the CL pectoral muscles in response to two unilateral SS intensities. To understand the relevance of the current project, it is important to review the relevant literature on the local and non-local effects of SS, the sensory and mechanical effects of stretching, and the role of stretching intensity in tissue response. The following chapters will discuss the structure and function of skeletal muscle and connective tissue, and how these tissues contribute to adaptations resulting from SS. The effects and recommended dosing parameters, will be discussed alongside the CL effects of unilateral strengthening and SS.

Muscle Structure

Muscle tissue is a highly organized system in which microscopic and macroscopic structures come together to enable force generation and transmission. The muscle involvement in movement and its structure make it a key target tissue for both performance enhancement and rehabilitation. Muscular tissue begins at the cellular level, with each cell (myofiber) containing repeating subunits called sarcomeres [42]. The sarcomeres are defined by Z-discs at each terminus, with interlacing actin (thin) and myosin (thick) filaments, creating a lattice that gives a striated appearance and allows for contraction [42]. The sarcolemma, which is the cellular membrane of the myofiber, is enveloped by a connective tissue layer called the endomysium[42,43]. The endomysium provides a connection to the extracellular matrix and adjacent fibres via the endomysial network, which supports lateral force transmission between neighbouring fibres and to the tendon-bone unit [42,43]. Groupings of myofibers are called fascicles and are enveloped by connective tissue called the perimysium, creating muscle subunits [42,43]. The subunits are further encased by an

outer fibrous sheath called the epimysium, which assists in blending the muscle fibres with the tendons at the proximal and distal ends of a muscle [43,44].

Both actin and myosin play key roles in muscle tone through cross-bridge cycling and the modulatory roles of the M-Bands and Z-Discs[45, 46]. The cycling of cross-bridges produces muscle shortening which contributes to a muscle's tone and force production leading to changes in the stiffness of a muscle during activity (dynamic stiffness). M-Bands and Z-Discs act to stabilize actin and myosin at rest, creating passive stiffness. The stiffness changes that occur are explained by the concept of thixotropy, which explains that muscle stiffness decreases with movement and increases at rest [45, 46]. In pathological conditions that significantly limit ROM, such as immobilization, the thixotropy is disrupted as passive stiffness increases significantly in the absence of cross-bridge cycling [47] and muscle atrophy occurs [48]. Muscle length will also decrease as the muscle loses sarcomeres to adjust to the shortened position [49]. Therapeutic interventions, such as SS, are used to mitigate these effects on muscle length and stiffness caused by reductions of ROM associated with pathology.

Muscle Tone

Understanding the modifiable factors that contribute to muscle tone assist in understanding how SS may be used to mitigate increases in tone that result from disuse. Muscle tone is muscle tension occurring in the absence of muscle activity and is affected by active and passive muscle characteristics [50, 51]. The active characteristics are generally related to neuromuscular factors, and the passive characteristics relate to the viscoelastic properties of the muscle [50]. The neurological factors that dictate muscle tone are quite complex and include both spinal and supraspinal stimulation, which are generally quantified using electromyography

while the muscle is relaxed [60]. Passively, the viscoelastic properties of the muscle, primarily the muscle's stiffness, contribute significantly to the muscle's resting tone [50, 51].

Muscle tone is essential for the proper functioning of the joints. Abnormally high resting muscle tone causes shorter length (hypertonicity) and relates to dysfunction within the muscles and joints [52]. Decreased length can place abnormal tension on muscle's attachments and surrounding tissues (e.g., tendons, synergistic, agonistic, and antagonistic muscles), resulting in changes in joint position and movement [52]. Muscles involved in maintaining posture are predisposed to increased tone, including the PMaj and PMin [52]. For example, shortened pectoral muscles create FSP altering shoulder kinematics [25] and increasing the risk of shoulder pathology [24,30]. Rehabilitative strategies aimed at reducing FSP are focused on reducing muscle tone and passive stiffness by lengthening the postural muscles such as, PMaj and PMin [53.]

Viscoelasticity is a structure's ability to return to its original length following the application of tensile force, with the change in length directly proportional to the force applied [51,54,55]. Viscosity refers to the muscle's ability to dissipate energy and resist deformation over time when lengthened, and can be characterized by creep, hysteresis, and stress relaxation [55, 56]. Creep describes a time-dependent increase in strain (i.e. deformation) when load/stress is applied to a muscle or musculotendinous unit [55, 56]. Hysteresis involves the dissipation of energy within a muscle as it moves from a loaded to an unloaded state [57]. Stress relaxation refers to the progressive decrease in resistance to an applied force (relaxation) of a muscle held at a constant length during passive loading. In SS research, each of these properties is examined using a variety of techniques to understand how SS effects the viscoelastic properties of the muscle. Both creep and hysteresis are commonly evaluated using dynamometry [56,58,59] while ultrasound

evaluates stress relaxation [57]. Dynamometry is most used to measure creep and stress relaxation. In SS research, creep is measured by holding a limb at a constant torque for a fixed period, maintaining the resistive force applied by the muscle to the dynamometer [58]. Alternatively, stress relaxation is measured by examining the gradual decrease in resistive force while a joint is held at a constant angle [56]. Ultrasound is preferred for hysteresis as the displacement of the muscle's musculotendinous junction can be measured to graph the stress and strain during loading and unloading. The difference between the two demonstrating the energy lost during deformation [57].

Length-Tension/Stress-Strain Relationship

Understanding the length-tension relationship of a muscle allows for a more comprehensive understanding of how the structural SS effects contribute to adaptations in muscle function. The relationship between a muscle's length and tension can either be active, where the capacity for force generation is dependent on muscle length, or passive, where the relationship exists between the length and viscoelastic properties of a muscle [60]. A key factor in the passive relationship is the muscle's slack length; the length of a musculotendinous unit at which passive tension starts to develop [61, 62]. Slack length is muscle-dependent and can shift with muscular training, activation and stretching [61]. Generally, slack length can be shortened temporarily by brief contractions at short lengths [63] or lengthened by stretching [64]. Changes to a muscle's slack length shift its operating position along the force-length curve during movement, altering the functional capacity across the joint's ROM [61,66]. In the PMaj, the slack length differs based on the region of the muscle due to the variability in fibre orientation. For example, during shoulder abduction the slack length occurs at 43° of shoulder abduction for the sternocostal region of the PMaj and 62° of shoulder abduction for the abdominal region [66].

Fascial Anatomy

The muscular tissues of the shoulders are generally the primary target for SS protocols though consideration of the surrounding connective tissues is warranted as they contribute to stiffness and force transmission [43,67,68]. The fascial system is a network of interconnected tissues that envelops musculoskeletal, visceral, and neural tissues and is also found superficially beneath the cutaneous tissues and subcutaneous fat [69-71]. The fascia surrounding each system can vary in tissue composition, with some rich in collagen and others in hyaluronan [71]. The fascial network of the chest and shoulders is a stratified system that connects the skin, pectoral region, axilla, thoracic wall, clavicle/pectoral girdle, and upper limb [69].

Conceptually, the fascia, muscles and accessory tissues (e.g., nerves and vessels) form the myofascial unit which transmits force and contributes to kinesthetic awareness of surrounding tissues [43,67,68]. Multiple reviews support the myofascial unit framework, highlighting that muscles transmit force through the endomysium and fascia to neighbouring muscles, neurovascular tracts and periosteum[68,72,73]. The myofascial unit framework acts as a general concept that can be directly applied to the shoulder girdle by considering the fascial, retinacular, and epimysial connections within the area [68,72,73,74]. Considering the vast fascial networks in the upper limb and chest, it is important to understand how these tissues contribute to force transmission and stiffness changes resulting from SS.

Deep fascial networks surround the primary muscles of the chest and shoulder connect to epimysium, perimysium and endomysium within the PMin and PMaj and the intermuscular septa [43,67,68]. The deep fascial layers interact with the pectorals, allowing load transmission from the muscle fibres to the surrounding fasciae and the neurovascular network [43,67,68]. Additionally, the thoracic wall and axillary fasciae connect with the pectoral and brachial fascia, allowing force

transmission between the chest wall and upper limb [73, 75]. The fascial networks are densely innervated and provide sensory feedback, proprioception, and motor control, thereby influencing shoulder mechanics and pain syndromes [67, 68, 72, 76]. To the author's knowledge, no studies have examined the fascial involvement in HaROM gains and stiffness alterations caused by SS. One study on the lower limb found that the stretch tolerance of knee extension is limited primarily by the fascia lata rather than muscular tissues [77]. Considering this, it is important to consider the potential of fascial involvement in SS outcomes of the stretched limb, and how the altered perception and force transmission may contribute to the CL effects of stretching.

Passive Tension Measurement Technique

Accurate measurement of passive tension component of muscle tone allows for the examination of muscle structure and assists in evaluating the effectiveness of SS on muscle tension. [78]. The passive tension is caused by the structural components of the muscle (e.g. fascial sheaths, titin and cross-bridges) in complete absence of neural activity [78]. In SS research, the quantification of passive muscle tension allows for the differentiation between mechanical and sensory changes following an intervention, as this tension occurs without the influence of neurological factors [79]. A non-linear increase in passive tension occurs as muscle length increases during stretching, with tension rising more drastically at near maximal length [78,80]. The steep increase in tension at end ROM serves as a protective mechanism to prevent excessive tissue lengthening [78]. In pathological states, the steep increase in tension can be altered by atrophy and muscle shortening causing elevated passive resistance and decreased ROM [78].

The observed increase in tension as the muscle nears end ROM, can be examined using four techniques: ultrasonography, dynamometry, elastography and myotonometry [78, 81, 82]. Ultrasonography typically involves the use of B-Mode ultrasound which allows for structural

examination of the muscle including fascicle length[78]. As previously discussed, dynamometry allows for the evaluation of passive torque and stiffness of joints while elastography allows for the localized evaluation of tissue stiffness [78]. Myotonometry involves applying a mechanical force to the skin to evaluate the deformation of the tissues and quantify stiffness [81,82].

In SS research, these evaluative tools are generally used in combination to provide a broader profile of tissue stiffness. Combining dynamometry and B-Mode ultrasound enables quantification of joint torques at various angles or fascicle lengths, allowing for examination of length-tension curves of specific muscles or muscle groups [78]. Though this technique is very useful for examining length-tension curves, dynamometry can pose some limitations in its use, including the joints for which it can be used [9, 10].

B-Mode ultrasound can be combined with two types of elastography: strain or shear-wave elastography (SWE). Elastography quantifies muscle stiffness by projecting sound waves into the tissue and evaluating how they return to the transducer [83]. Strain elastography, combined with B-Mode ultrasound, enables muscle tension examination by applying manual compression to a tissue via a soundhead/transducer and measuring the resulting deformation, also known as tissue stiffness [83]. Unfortunately, strain elastography is extremely operator- and technique-dependent, resulting in poor repeatability [83]. The resulting measurement provides information on the relative stiffness or strain ratio of a tissue [83].

SWE with B-Mode ultrasound allows for the quantification of the muscle's shear modulus as an index of stiffness [83]. There is a strong linear relationship between the shear modulus and active and passive muscle forces, thereby allowing quantification of changes in passive tension in response to stretching [78,83-85]. Although it is generally more reliable than strain

elastography, SWE remains operator-dependent, with variables such as muscle activity, probe alignment, probe placement, and joint angles requiring strict standardization [78,86].

Myotonometry is a technique more commonly used clinically to quantify stiffness in muscles and other tissues by applying a brief mechanical oscillation that induces tissue deformation [81, 82]. For clinicians, myotonometry devices, such as the MyotonPro, are non-invasive tools that can provide objective tissue stiffness values that can be tracked over the course of a rehabilitation program. The devices are handheld and measurement acquisition and processing is completed within seconds making its use very conducive to a clinical environment. The stiffness measurements obtained using myotonometry should be interpreted with caution, as the device measures the stiffness of all superficial structures between the target muscle and the probe and should not be used for deep tissues [82]. Due to this limitation, myotonometry is best suited for a clinical environment where it can be used as an objective measure used to evaluate patient progress rather than in a research setting where the goal is to evaluate specific tissue stiffness. In SS research, B-Mode ultrasound combined with SWE is the preferred measurement technique, as it allows for the most tissue-specific stiffness measurements [78, 86] and does not pose the same joint limitations as the dynamometer [9,10]. Further examination of these methods occurs in later sections.

Static Stretching

SS is tension applied to a muscle to increase its resting length and decrease the perception of stiffness and pain [2, 3]. It is primarily used in rehabilitation and sports settings for muscle recovery and injury prevention [1]. Performed either actively or passively, SS is the most common type of stretching, in which the individual being stretched performs a continuous motion to progressively lengthen a muscle until a stretch is perceived [2, 3, 79, 87, 88]. When performed

passively, another person guides the individual's limb into the stretch position; when performed actively, the individual guides their own limb into the appropriate position. There are many modifiable factors that impact the effectiveness of a SS bout including sets, intensity and time [4-7].

Athlough research on the effectiveness of SS for recovery and injury prevention is mixed, it is widely implemented across a variety of athletic and rehabilitative settings [2,3,87]. The most frequently reported outcome of SS improvements in ROM [4-7]. Alternatively, some research has also documented acute performance impairments following SS [89-92] while others show no functional impairments [89, 97]. Though positive ROM change exists, there is mixed evidence supporting the long-term efficacy of SS [98,99] and acute functional impairments following SS [92, 95-97]. The lack of definitive evidence leads researchers and practitioners to question the practical implications and effectiveness of stretching. The ROM increases following stretching are thought to decrease injury risk and incidence, as reduced flexibility may predispose an individual to injury [95]. The evidence supporting a reduction in injury risk is limited; some researchers have shown a decrease when SS is added to a pre-exercise warm-up lasting less than 60 seconds [3,89,95]. It is also important to note that research consistently indicates that stretching should be combined with other interventions to create a comprehensive injury-prevention program [89, 95, 96].

Despite mixed evidence regarding its long-term effects [98,99] the ACSM considers SS as an essential part of a post-exercise recovery tool and to reduce muscle soreness [1]. Conversely, a recent review found that SS is no more effective than other modalities in restoring strength or reducing delayed-onset muscle soreness [97]. It should also be noted that the authors of the review stated that the diversity of protocols across the limited number of available articles led to insufficient data to conduct a proper systematic review or meta-analysis of their effects [97]. Additionally, several prior reviews found no effect of SS on delayed-onset muscle soreness [98, 99].

The majority of the available SS research has focused on the lower limbs, with few authors examining its effects on the upper limbs. There are two theories that explain the increase in ROM resulting from SS: the sensory and mechanical theories.

Mechanical Stretching Theory

Mechanical theories explaining the adaptations of skeletal musculature in response to SS involve examining changes in tissue properties that have been discussed previously. Acutely, SS reduces musculotendinous unit stiffness and viscoelastic resistance [9, 10]. The mechanical changes that influence increases in ROM are primarily due to muscle length and viscoelastic properties [9, 10]. The properties that determine muscle length can include muscle extensibility, tension, cross-sectional area and time [9-11]. Viscoelastic changes that occur at a muscular level are quantified by changes in passive torque, neuromuscular characteristics and musculoskeletal stiffness [9, 10]. Passive torque and neuromuscular adaptations are often used to describe changes in muscle tone. Passive torque quantifies the passive tension in a singular muscle or muscle group at a given angle, with changes in torque indicating adaptations in the viscoelastic properties of a muscle [9, 10]. Neuromuscular characteristics involve the level of muscle excitation and are commonly quantified using electromyography. Electromyography defines the contractile mechanism that influences changes in muscle tone. This can also determine the level of excitation occurring in a given muscle in response to the stretch [9, 10].

The neuromuscular responses to SS have been widely examined, with proposed alterations being theorized to affect several neural mechanisms. Motoneuron excitability is influenced by SS and is measured by examining Hoffman reflex (H-Reflex) amplitude and Ia afferents [100]. There is also contentious literature that examines the effect of SS on corticospinal excitability and inhibition [101, 102]. Recent meta-analyses suggest that no significant neural

changes occur acutely in response to SS [19] though another review found that SS can likely alter the amplitude and duration of the muscular action potential wave (M-Wave) and exteroceptive (E) Reflexes [100]. There is some disagreement in the literature about the magnitude and persistence of neural changes acutely after stretching [102,103]. During the stretch, sensory receptors in the skin initiate E-Reflex inhibition, which contributes to sympathetic inhibition and promotes muscle relaxation[100]. Evidence suggests E-reflex inhibition may be dose-dependent, occurring only during large-amplitude stretching and ceasing upon termination of the stretch [104].

Torque and ROM are generally measured simultaneously by using a dynamometer as a participant moves through a given range of motion [56-58]. From this measurement, passive torque can be quantified. In stretching research, ROM is stopped when a participant first reports a stretch sensation, at the point of discomfort, or at maximal stretch without pain [56-58]. Peak/Passive torque is measured at the predetermined point, and this value is used to calculate musculotendinous unit stiffness. Additionally, this measurement has also been used in some studies to examine the impact of stretching on passive muscular properties and to quantify stretch tolerance [106,108].

Musculotendinous unit stiffness is defined as the relationship between passive torque and passive ROM, which represents the viscoelasticity of the musculotendinous unit. Passive torque and ROM are plotted to create a curve, and musculotendinous unit stiffness is considered the sloped portion of the curve, with authors utilizing a variety of methods to identify which portion of the curve represents the musculotendinous unit stiffness [56-58]. Some authors have used the final third of the curve, 50% of the maximal ROM angle achieved or a measurement

between certain angles [106,108]. Using this method, SS has been shown to decrease musculotendinous unit stiffness and passive torque, though there are some limitations to its use with the dynamometer [9, 10]. Primarily, the use of the dynamometer limits which specific muscles and muscle groups can be tested due to the device's size and setup. There are also tissue-specificity limitations, as the dynamometer assesses torque at a specific joint rather than at specific tissues, which ignores the contributions of tendons, connective tissue, and additional musculature that contribute to a specific ROM. As previously mentioned, torque is not specific to muscle tissue; it can also result from changes in surrounding tissues. To examine these more specific tissue changes, researchers have begun using shear-wave elastography [9, 10].

Shear-wave elastography is a novel technique that is coupled with B-Mode ultrasound to accurately quantify tension of specific soft tissues [83]. B-Mode ultrasound is used to locate the specific tissue, and then the shear-wave elastogram allows for an adjustable ROI to be identified in the target tissue. Once the ROI is determined, pulses are applied to the target tissues, creating a ripple effect of shear waves through the tissues, and the propagation speed is measured by collecting the shear-wave velocity[78,83-85]. The output of this measurement is a colour-coded map overlaid on the ROI, with each colour representing different levels of tissue stiffness and an average stiffness measurement for the given region.

Stretching research has begun incorporating SWE to quantify tissue-specific stiffness changes, and compared with measures of musculotendinous unit stiffness, it allows for greater specificity because a single structure is measured at a time [78, 83-85]. Elastography also does not pose the same barriers as dynamometry, allowing a broader range of mechanical responses and tissues to be examined. One study examined passive torque and SWE in response to resistance

training and found that velocity increased post-exercise, but passive torque did not, with no correlation between the two measurements. The authors concluded that the two methods demonstrated limited agreement, which may be angle-specific, and that SWE may be more sensitive to change. Though shear-wave elastography may be more beneficial than dynamometry, it still has limitations in musculoskeletal tissues [10, 105].

Due to the anisotropic nature of muscular architecture, the shear waves can travel faster and more efficiently parallel to muscle fibres rather than perpendicular [10,105]. As such, velocity measurements are sensitive to the probe's location and orientation, and changes in these factors can affect the measurements. The anatomy of each muscle examined should be heavily considered prior to measurement acquisition, as multipennate or fusiform muscles may not allow for parallel alignment of fibre orientation with the ultrasound probe. Significant subcutaneous fat or tissue defects (scar tissue, ossifications) can severely affect tissue visualization and the propagation and measurement of subcutaneous tissues. Shear waves attenuate more strongly with depth, which may cause superficial tissues to affect their effectiveness in measuring deeper tissues. Finally, elastograms can only measure a specific ROI of the muscle at a time, limiting their use in generalizing effects to the entire muscle outside the ROI [10, 105].

Sensory Stretching Theory

The sensory theory of stretching posits that changes in ROM following SS are influenced by increases in stretch tolerance [12-15]. From a mechanical perspective, increases in passive torque at maximal ROM angles indicate greater stretch changes, but this overlooks instances in which stretch tolerance increases despite a lack of measurable viscoelastic

modification [13, 106]. Decades ago, researchers discovered that increases in ROM induced by SS may not be due to increases in muscle length but to adaptations of sensory pathways [33,107]. When examining muscle changes following stretching, ROM and passive torque were used to produce a length-tension curve, which is used to calculate musculotendinous unit stiffness [106,108]. Theoretically, if SS increased muscle length, the entire length tension curve would shift to the right, indicating longer muscle lengths for a given torque. However, it was found that, instead of shifting to the right, ROM and passive torque increased only at the end ranges. This indicates that muscle length did not change, and the increase in ROM is attributed to an alteration in participants' perception of sensation at the end of ROM. The rightward shift of the torque-angle curve has been a contentious topic in the literature, with some authors reporting that SS does not shift the torque-angle curve. Others have found that the shift does occur after SS, indicating that mechanical changes may result from induced muscle length. As stated previously, the applicability of the exploration of the right shift theory is limited to only joints that are conducive to the use of dynamometry [106,108].

Alternatively, increases in stretch tolerance can be more widely explored at various joints and within a wider range of musculature, as it is subjectively reported rather than objectively measured. The mechanisms underlying increased stretch tolerance are theorized to be primarily attributable to endogenous pain modulation [14] and peripheral afferent input from musculotendinous units [109-111]. Endogenous pain modulation plays a central role in stretch tolerance, demonstrating that endogenous analgesic systems can modulate stretch tolerance and that increases in pain modulation correlate to more significant increases in ROM[14]. Hypoalgesia induced by exercise and conditioned pain modulation can also increase stretch tolerance, suggesting a shared central pain-processing mechanism that limits ROM during stretching [14].

Peripheral afferent input can influence stretch perception and reflex pathways. Multiple reviews have recognized that modulation of afferent activity from muscle spindles, Golgi tendon organs and cutaneous receptors can alter perceived stretch and reflex responses [13, 112, 113]. The modulation of these receptors can affect stretch tolerance, and, in turn, ROM. Spinal and supraspinal pathways modulate the sensitivity of afferents and the subsequent perception of stretch, which may also contribute to shifts in the torque-angle relationship during stretch [13, 112]. Generally, the literature agrees that neural pathways together contribute significantly to increases in stretch tolerance after SS [13, 14, 107, 112, 114].

When examining the literature, some authors have noted that CL stretching effects in homologous muscles may be related to mechanoreceptors and nociceptors [115, 116, 117]. More specifically, activation of mechanoreceptors and nociceptors in one limb can influence CL muscle performance and fatigue via central mechanisms that dampen voluntary drive. This literature does not specifically relate to increases in ROM on the CL side but may assist in demonstrating how CL increases in stretch tolerance may occur [115, 117]. The possible neural changes occurring contralaterally as a result of unilateral SS are further discussed in later sections of this work.

Static Stretching Dosage

Multiple dosing factors can be manipulated to try and alter the benefits derived from a SS bout. Of the modifiable parameters, duration and repetitions are the most frequently manipulated in the literature as they are easily modifiable and not subjective. Researchers have shown that a minimum of 5-seconds of SS is required to increase ROM [17]. The ACSM recommends that 2-4 sets of 10-30 seconds of stretching be performed at the point of discomfort for a minimum of 60 seconds per muscle group [1]. More subjective factors, such as intensity, are

often neglected in the literature, with a recent review noting that 79 out of 152 articles examining SS mentioned the stretch intensity[3].

Static stretch intensity is the perceived intensity or tolerance to stretch that an individual experiences as the degree of stretch increases. Although intensity can vary among participants, there is evidence supporting its effectiveness in modifying the tissue response to SS. SS at an intensity that is too low may not elicit tissue responses that increase ROM, but may help reduce the risk of sprains and strains [3, 12]. High-intensity SS may cause the participant significant discomfort by placing stress on the musculotendinous unit and inducing an acute inflammatory response [18, 19]. Although both force and torque can be objectively measured during a stretch, neither is used to prescribe intensity to patients. Alternatively, intensity is more commonly prescribed using a stretch intensity or visual analog scale with descriptive terms and numerical ratings to quantify perceived intensity [12, 16]. Common descriptive terms used in research include the point of discomfort, maximal tolerable stretch or point of pain [16,19]. Further, intensity is generally prescribed as a percentage of the individual's reported intensity at the numerical rating that the individual associates with a given term [16,19]. As intensity is very dependent on individual perception, this results in challenges with comparing outcomes between participants and studies [16].

Many studies have examined the acute effects of different SS intensities on the hamstrings, quadriceps, and calves. Increases in ROM related to static stretch intensities have primarily been examined in the lower body, including the hamstrings, quadriceps and calves [19, 34]. Collectively, the results of these studies are varied and nearly half of the studies to date have found no difference among intensities [19, 92, 118]. Additionally, the literature reports that high-

intensity SS increased passive torque and decreased stiffness, while lower-intensity stretching increased passive torque and stiffness compared with higher intensities [19, 34, 92, 118]. One study found that performing a stretch at 120% of the point of discomfort on a visual analog scale was more effective at increasing ROM and decreasing musculotendinous unit stiffness than 110% [19]. The group also examined the effect of time on the two variables and reported that higher-intensity stretching evoked larger changes than longer-duration stretching [19]. Alternatively, another group examined SS at three intensities (80%, 100%, and 120%) on quadriceps ROM and stiffness and reported that SS at 100% resulted in sufficient increases in ROM and decreases in muscle stiffness [16]. This may indicate that higher-intensity SS may yield greater effects, but there may be limited benefit from stretching beyond 100% of an individual's tolerance. All the literature to date has examined only the effects on the lower limbs, not on the upper-limb musculature. As findings in one area do not always translate to effects in the other, it is important to conduct additional research to examine how the findings translate to the upper limb.

Pectoral Static Stretching

PMin and PMaj significantly influence the position and movement of the shoulder complex, acting anteriorly on the shoulder girdle [119]. The attachments from the thorax to the humerus (major) and coracoid processes (minor) allow for internal rotation and abduction of the shoulder. Though these movements occur smoothly in a normal shoulder, shortening of the PMin has been associated with FSP [26]. Clinically, FSP is quantified using the double square method, which measures the anterior displacement of the shoulder [26]. In research settings, motion capture is more commonly used to evaluate FSP and scapular kinematics [25]. The anterior positioning of the shoulder alters scapular movement and can predispose an individual to shoulder pathologies

[25]. Although multiple studies evaluate the effect of the PMin on FSP, few include the PMaj in their evaluations. As mentioned, the PMaj attaches directly to the humerus, which can contribute to anterior displacement of the humerus, therefore altering the movement of the shoulder girdle. There is little research examining the effects of SS on muscle stiffness in conjunction with horizontal abduction range of motion (HaROM), FSP, and PML. Given the impact of both pectoral muscles on FSP, interventions aimed at reducing FSP and improving scapular movement should target both muscles.

SS is a common method used to lengthen the pectoral muscles in a variety of settings, including during warm-ups/cool-downs in sporting pursuits, to reduce FSP, and during mastectomy rehabilitation. When used to treat FSP, a clinician will typically administer a stretch to the pectorals by passively placing the shoulder at 90° of HaROM and then externally rotating so that the forearm is approximately parallel to the floor. Alternatively, a patient-administered stretch can also be prescribed by having the patient place their forearm on a wall and then horizontally abducting until they perceive a stretch [28, 30,120]. The preferred position for the PMin allows for optimal stretching of the sternal and clavicular regions, but not the abdominal region [31]. Though this is a commonly used intervention, the acute effects of SS on the pectorals have been minimally investigated, as most acute studies have focused on the lower limb or involve multi-week protocols that often include multiple modalities. Two studies examining pectoral stretching as a stand-alone method for treating FSP showed postural improvement at 2 and 4 weeks [27,28]. One study examined the effects of a 4-week stretching protocol on shoulder balance and strength in individuals with FSP [29]. It was found that horizontal abduction strength, extension strength and static balance of the shoulder improved following the 4-week protocol [29]. When PMin stretching was combined with upper trapezius strengthening over a 3-week period,

there was a significant reduction in FSP and PML [120]. These studies demonstrate that pectoralis stretching can result in lengthening of the PMin and a decrease in FSP.

Given the impact of muscle stiffness on length and ROM, it is important to investigate whether changes in muscle stiffness correspond to improvements in FSP. As research on SWE in the musculoskeletal system is emerging, only one group has assessed the effects of pectoral stretching on muscle stiffness. The use of SWE to assess the effects of SS can assist in differentiating whether they result from structural (e.g., changes in stiffness) or neural adaptations. The researchers have examined the acute and prolonged effects of PMin stretching on muscle stiffness as well as the effect of dose on muscle stiffness [30, 32]. In their first study, participants performed 10 repetitions of a 30-second pectoral stretch, with researchers collecting the muscle stiffness before the first repetition, between each repetition and following the final repetition [30]. Stiffness was determined to decrease after the first repetition and subsequently after the remaining nine repetitions[30]. In a subsequent study, the group examined the difference in stiffness changes between short duration (1 set of 30 seconds) and long duration (10 sets of 30 seconds) stretching[32]. Muscle stiffness was measured pre- and post-stretching, as well as at 5-minute intervals following each intervention, and both interventions decreased stiffness, but the long-duration stretch resulted in a longer-term decrease [32].

Though this study examined the effects of maximal intensity stretching on the PMin, it did not examine the effects on PMaj. Additionally, the CL muscles were not examined, and there are no current studies examining the effects of unilateral pectoral stretching on the opposing side. Moreover, there are no studies to date that examine the effects of unilateral stretching on CL FSP, PML or HaROM. Further research into the effects of unilateral pectoral stretching on the

PMaj and PMin, as well as FSP, PML, and HaROM of the CL limb, will help clinicians better understand how to address these tissues.

Cross-Over Effects of Strength Training

Strength training of the CL limb is a commonly used tool in rehabilitation settings to increase and maintain strength in both limbs during times of injury [121,122]. Typically used in situations when a limb is immobilized or ROM is reduced, the opposing limb can undergo training to elicit a crossover effect resulting in a decrease of muscle strength and ROM losses of the injured limb [123-125]. One meta-analysis showed strength gains in the untrained limb of 7.6%, representing approximately 35.1% of those seen in the trained limb [126]. Although these training methods are commonly used, unilateral training can also lead to non-local muscular fatigue, which should be considered when training for strength and performance [127].

The two main theories used to explain the neurological causes of the cross-over effect include the bilateral access and cross-activation hypotheses [128]. The bilateral access hypothesis posits that movement blueprints are stored in a central location accessible for control of both limbs [124,] and that the amount of transfer depends on the difficulty of the task [125]. The cross-activation hypothesis holds that unilateral execution of a movement results in a bilateral increase in corticospinal excitability [128]. While both theories provide explanations for the cross-over effects, when they are applied to strength training, it is believed that CL effects driven by cross-activation occur during maximal strength training (e.g., training at 90-100% 1RM), whereas bilateral access is better applied to more complex tasks (e.g., sport-specific skills) [128,129]. The addition of visual stimuli has also been examined to increase the resultant effects of cross-education by activating mirror neurons [130]. One study found that strength gains increased by 34% with the addition of visual stimuli compared with unilateral training [131].

Although research on the neurological causes of the cross-over effect is robust, the effects of training on muscle composition and function resulting from the cross-over effect are seldom examined. Early research examining changes in muscle size in the untrained limb showed that there may be increases in muscle cross-sectional area following unilateral training [132] but this finding has not been replicated in subsequent studies [133]. Although training the CL limb may not increase muscle size in the untrained limb, some research has shown that training may help attenuate muscle loss associated with immobilization. Performing isometric ulnar deviation on an uninjured limb five times a week for three weeks has been shown to prevent muscle loss in the injured limb [125]. A similar result was shown in the biceps and triceps following maximal isometric elbow flexion and extension three times a week for 4 weeks, when performed on the uninjured limb [134]. The findings were further reproduced in a study examining the effects of a single-arm biceps curl performed on the uninjured limb 3 times a week for 3 weeks [135]. More recent studies have shown that the frequency, intensity, and type of training significantly influence observed strength gains, with eccentric contractions showing an additive effect and isotonic and isokinetic contractions yielding similar gains when matched for volume and intensity [136]. High-intensity, high-volume training has been shown to produce greater CL gains than lower-intensity training [136]. Shorter-duration training lasting 1-2 weeks has shown inconsistent strength gains compared with longer interventions lasting more than 3 weeks [136]. There is no research examining pectoral muscle cross-education following unilateral strength training, though research on the cross-over effects of the upper limbs is extensive. Further research should be conducted to examine the cross-over effects of strength training on the pectoral muscles. Understanding the extrapolation of the current literature can help clarify the

breadth of cross-over effects and how they may affect those of other modalities, such as stretching.

Non-Local Effects of Stretching

Similar to unilateral strength training, unilateral stretching has been shown to elicit local, CL and global effects. The global effects of stretching refer to areas of the body aside from those CL to the target tissue. Clinicians will generally consider the global effects when prescribing stretching to address individual patient goals. The observed effects can include increases in ROM and decreases in force output [95]. The observed benefits have been theorized to be caused by psycho-physiological influences such as increases in stretch or pain tolerance [37,38,137] and the downregulation of sympathetic excitatory nerve stimulation [5]. Prolonged SS can also affect the nervous system by reducing motor neuron spindle reflex activity [97,138]. The afferent innervation of the type I and II muscle spindles via spinal neurons [139] projects to the somatosensory and primary motor cortices [140,141,] which could also contribute to global changes. Additionally, fascial chains have also been theorized to be related to the global effects of SS due to the fascia's ability to modify its stiffness and transfer stress to nearby structures[142-144]. Fascial involvement has been observed in two studies examining increases in cervical ROM in response to gastrocnemius and hamstring stretching [145,146].

Decreases in sympathetic nervous system stimulation and neural inhibition are theorized to reduce the force output of the target muscles [5-6]. The negative impact of SS on performance is dose-dependent [95]. A recent scoping review reported that typical research protocols average six 36-second repetitions, accumulating 216 seconds of total stretch duration [95]. Since single stretch durations over 60 seconds are known to impair force output, these extensive total volumes likely trigger a global, systemic reduction in strength [5-7]. Few studies have reported ROM increases

following unilateral SS in a variety of areas outside of the same musculature on the opposite side. The current literature examines a variety of tissues, including the effects of stretching on opposing limbs (e.g., upper limb vs. lower limb), opposing CL limbs (e.g., left upper limb vs. right lower limb), and ipsilateral agonist vs. antagonist musculature (e.g., hip flexor vs. hamstring). Globally, hamstring SS increases in hip, back, and shoulder ROM have been observed [147]. One study reported an 8.2-9% increase in shoulder extension following lower body SS [16]. In another study by the same group, a 5.2% increase in hip flexor ROM was observed following upper limb SS [148]. Unilateral SS of the hamstrings has been shown to increase ROM of the CL hip flexor [33].

Finally, population variables such as age, training status and limb dominance can also contribute to the observed effects of unilateral stretching [95,122,149]. Younger populations tend to show more rapid and pronounced central neural adaptations that can assist in improved performance and mapping of unilateral tasks [122]. Untrained or sedentary individuals may experience greater non-local adaptations due to a lower threshold for neuromuscular stress, which increases systemic sympathetic responses and central drive [95,149]. An individual's dominant hemisphere possesses a more complex network of motor pathways, creating more highly organized blueprints that may be accessed by the non-dominant limb [122].

Collectively, the current literature indicates positive full-body effects on ROM following unilateral stretching, supporting the clinical use of this modality. To further examine these effects, it is important to examine the effects of SS on the target tissue's homologous CL musculature to examine the specificity of the cross-over effects. Knowing the extent of the documented effects may assist clinicians in choosing the most appropriate dosing parameters when prescribing stretching for global and CL benefits.

Cross-Over Effects of Stretching

Most used in rehabilitation; unilateral stretching protocols are typically prescribed to preserve the ROM of an immobilized limb. As with the global effects of unilateral SS, the effects on the CL musculature include changes in ROM, stiffness, and neural excitability. There are many theories that contribute to the cause of the effects, with an emphasis on neural adaptations [122] and some conflicting reports of morphological changes [150,151]. The spillover hypothesis posits that motor blueprints occurring in the cortical, subcortical, and spinal networks of the stretched limb may be accessed by the CL tissues to increase ROM [33, 34, 152]. Prolonged SS of a limb can reduce muscle electromyographic [35], H-reflex activity [152] and type I and II muscle spindle afferent output [100]. Additionally, group III and IV muscle afferent spindles can inhibit corticospinal motor pathways [39], thereby diminishing central drive to the stretched limb and potentially to the non-stretched limb. The decrease in central drive can relax the non-stretched muscle, increasing ROM [149]. The widespread cortical interconnectivity may also relate to a global increase in stretch tolerance that extends beyond the target tissue and may contribute to CL increases in ROM [39].

Morphologically, the findings that increases in ROM are attributed to adaptations in muscle structure are inconsistent. Many authors note that non-local ROM changes cannot be attributed to muscle elasticity [35,36] or to changes in viscoelastic properties [37, 38, 154] as the muscles are not directly undergoing the intervention.

Few studies have examined whether unilateral stretching increases ROM in the CL limbs, and those that have are predominantly in response to lower-limb stretching. For example, unilateral hip flexor SS increased CL hip extension by 6-8% [33]. In a recent study, participants underwent a single session of unilateral SS of the quadriceps and hamstrings, performing 8 sets

of 30-second repetitions, with increases in hip flexion ROM observed in both limbs, with the unstretched limb demonstrating a 4.3% increase from pre- to post-measures [40]. A similar study also demonstrated an increase in hip flexion ROM following unilateral hamstring stretching [41]. While existing studies indicate that ROM gains are driven by the central nervous system, additional studies should examine the effects of unilateral stretching on CL muscles in the upper limb. It is important to explore extrapolation from the current literature, as the joints, specifically those in the shoulder girdle, allow for greater free movement than those in the lower limb and therefore may respond differently than those in the lower limb or surrounding fewer mobile joints.

2. Purpose

Significant gaps remain in the current literature, limiting clinicians' access to research-based clinical recommendations for care. The purpose of this master's thesis is to evaluate the acute neurophysiological and mechanical responses to unilateral pectoral SS of the dominant limb. The proposed study will aim to isolate how varying prescriptive factors, primarily stretch intensity, acutely impact tissue viscoelasticity, postural alignment, stretch tolerance, and range of motion. By conducting this study on a healthy, active population, this study will assist in creating a foundation for further investigation into how more targeted rehabilitation protocols can be created for a variety of pathological conditions impacting the upper limb. To address the current limitations in the available literature, this study will focus on three central objectives:

3. Objectives

The objectives of the proposed study are to determine:

1) Whether cross-over effects of unilateral pectoral stretching occur in CL PMaj and PMin stiffness, FSP, PML and HaROM; and
2) If the cross-over effect will elicit change in the variables related to the muscle structure (SWV, FSP and PML) along with increases in HaROM; and,
3) If stretch intensity impacts the extent of the effects observed in the CL PMaj and PMin stiffness, FSP, PML and HaROM, and
4) If the extent of HaROM and intensity changes on the intervention limb influence the degree of change in HaROM on the CL limb,

4. Hypotheses

Considering no current literature has examined the CL effects of unilateral pectoral SS, I hypothesize that:

1) Effects will be seen in FSP and HaROM. Given that FSP and HaROM can be heavily influenced by the neuromuscular adaptations of stretching, I hypothesize that FSP will decrease and HaROM will increase following stretching
2) No effects will be seen in PMaj stiffness, PMin stiffness and PML. As these variables are significantly related to the viscoelastic properties of the muscle, I hypothesize that no changes in these variables will occur, as they are not affected by the acute crossover effects of SS.
3) The effects seen in FSP and HaROM will be greater at higher intensities. Given that the neuromuscular cross-over of movement is greater at higher intensities and with transfer from the dominant to non-dominant limb, I hypothesize that greater differences will occur in the maximal stretch condition than in the moderate condition.

4) The pre-post differences in intensity and ROM on the intervention limb will have an influence on the degree of change occurring in the CL limb. I hypothesize that the maximal intensity condition will have a greater effect than the moderate condition

5. Experimental Methods

The current study was part of a larger study [160] that explored the effect of static stretching at two different intensities on HaROM, PML, FSP and PMaj and PMin stiffness. The current study examined the effect of these interventions on the unstretched limb (the CL limb) and presents data from the stretched limb (i.e. the intervention limb) to characterize the CL effects in the context of what effects were observed on the intervention limb .

5.1 Experimental Overview

Participants attended two experimental sessions at least 72 hours apart in this repeated-measures crossover design study. During each session, participants were randomly assigned to one of two stretching intensities (moderate or maximal) for a static, self-administered pectoral stretch. A control condition (CON) was conducted at the beginning of the first experimental session. The non-dominant limb was assessed for any CL effects on the stretching protocol. The stretch was performed at two intensities (MOD and MAX) on the dominant limb. PMaj and PMin stiffness, FSP, PML and HaROM of the dominant and non-dominant limbs were collected before and immediately after each stretch intervention and the CON. All experimental procedures were approved by the University of Manitoba Research Ethics Board and adhered to the guidelines set forth in the Declaration of Helsinki[155].

5.2 Participants

An a priori sample size was determined utilizing G*Power (V3.1) with an α of .05 and a β of .8; a total of 24 participants were required to detect a large effect size (ηp^2=0.14). Therefore, 32 participants (16 males and 16 females) were recruited to accommodate potential attrition and data errors. Data from 2 participants (1 female, 1 male) were dropped due to an injury unrelated to the study and poor ultrasound image quality.

Prospective participants underwent evaluation to ascertain their eligibility based on the following criteria: between 18-40 years of age; absence of recent history of pain, injury or orthopedic conditions (within 6 months) in either arm/upper thoracic region; no diagnosed neurological or musculoskeletal disorders; no contraindications to ultrasound use (e.g. open wounds, ultrasound gel allergies, pacemaker); no engagement or history of engagement in sports or activities that necessitated extreme upper body ranges of motion (e.g., gymnastics, yoga, baseball pitching) within the last 5 years; not pregnant or breastfeeding; and a Beighton's score of ≤ 5 for joint hypermobility[156]. Handedness was determined using the Modified Edinburgh Handedness Inventory[157]. Participants were instructed to abstain from upper body resistance training for 48 hours, cannabis and alcohol consumption for 24 hours and caffeine intake for 6 hours in preparation for each session.

5.3 Experimental Sessions

During the first session, after obtaining informed consent, stretching intensity (moderate or maximal) was randomly assigned according to a predetermined schedule. Participant characteristics such as height, weight, age and sex assigned at birth were self-reported. Handedness was determined using the Modified Edinburgh Handedness Inventory[157].

PMaj and PMin stiffness, FSP, PML and HaROM were measured before (PRE) and immediately after (POST) the CON and each stretch intervention (Figure 1). All measurements were taken bilaterally, with the dominant arm measured first to mitigate potential differences related to measurement order and increased rest.

At least 72-hours later, participants engaged in identical procedures except that there was no CON prior to the stretch intervention and the intensity of the stretch intervention was different

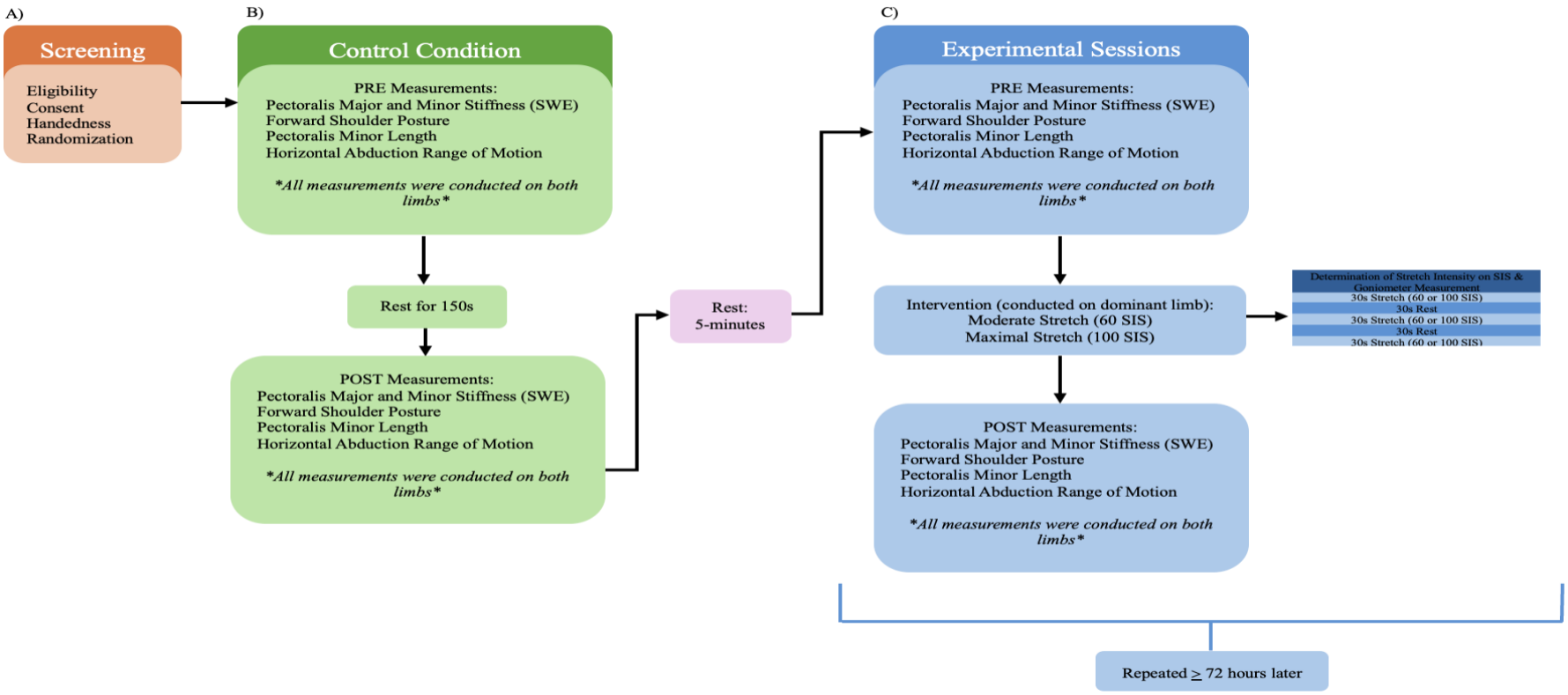

Figure 1. Experimental Overview. *A) Potential participants were screened for eligibility. Once deemed eligible, informed consent was obtained, and handedness was assessed. Participants were then randomized to a stretch intervention; B) A CON was completed at the beginning of the first session only. PRE measurements (PMaj and PMin stiffness, FSP, PML and HaROM) were taken on the dominant and non-dominant limb. The participant then rested for 150s, which mimicked the length of the stretch intervention. All PRE measurements were reassessed following the rest period. Prior to the intervention, the participant rested for an additional 5 minutes; C) PRE measurements were taken prior to the intervention. Following these measurements, the participant performed three sets of a 30-second moderate-to-maximal pectoral stretch on their dominant limb, with 30 seconds of rest between repetitions. PRE measurements were then reassessed. The participant attended a second visit at least 72 hours later where the second stretch intensity was performed.*

5.3.1. Shear Wave Elastography

PMaj and PMin stiffness were assessed using SWE while participants were seated on a plinth reclined at 30°. B-mode ultrasound was performed using a 2-10 MHz transducer (XDclear linear probe, GE, Mississauga, Ontario, Canada) on a Logiq E10s ultrasound machine (GE, Mississauga, Ontario, Canada). A single researcher applied ultrasound gel to the participant's skin to facilitate image acquisition and placed the probe approximately 1 inch below the clavicle and lateral to the sternal edge. The PMaj and PMin were then identified, and the probe was aligned parallel to the muscle fibres, and the probe placement for each muscle was marked with a permanent marker (Figure 2).

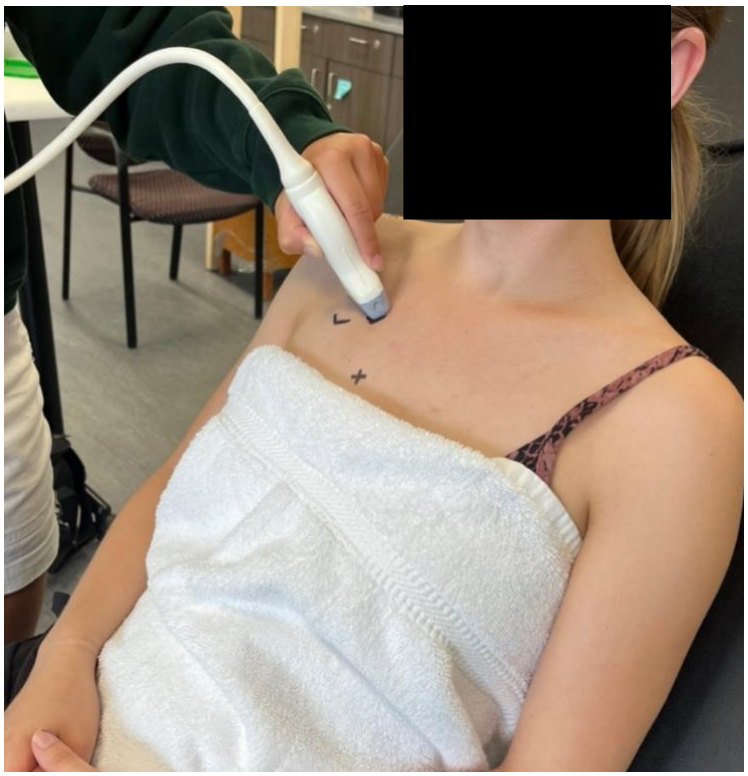

Figure 2. Ultrasound probe alignment. *The ultrasound probe was aligned with the PMaj or PMin muscle fibers (the PMaj in this photo), and the corner of the probe was outlined to ensure a repeatable measurement location for the pre- and post-measurements.*

Once each muscle was identified, Elasto mode generated a colour-coded map of shear-wave velocity across the largest ROI attainable for each muscle, excluding blood vessels and connective tissue. A 10-second elastogram was captured for each muscle, bilaterally. During image acquisition, the participants were instructed to inhale deeply, exhale for 3 seconds, and then hold the bottom of their breath for a maximum of 10 seconds to minimize motion artifacts from breathing [32]. From each elastogram, three images with minimal artifacts (identified as regions with abnormal colouring (e.g. large portions of opacity/darkening in the ultrasound image that isn't visible on other images or areas of the elastogram that are a significantly different colour than the other images) or devoid of colour) were selected and retained for subsequent analysis (Figure 3).

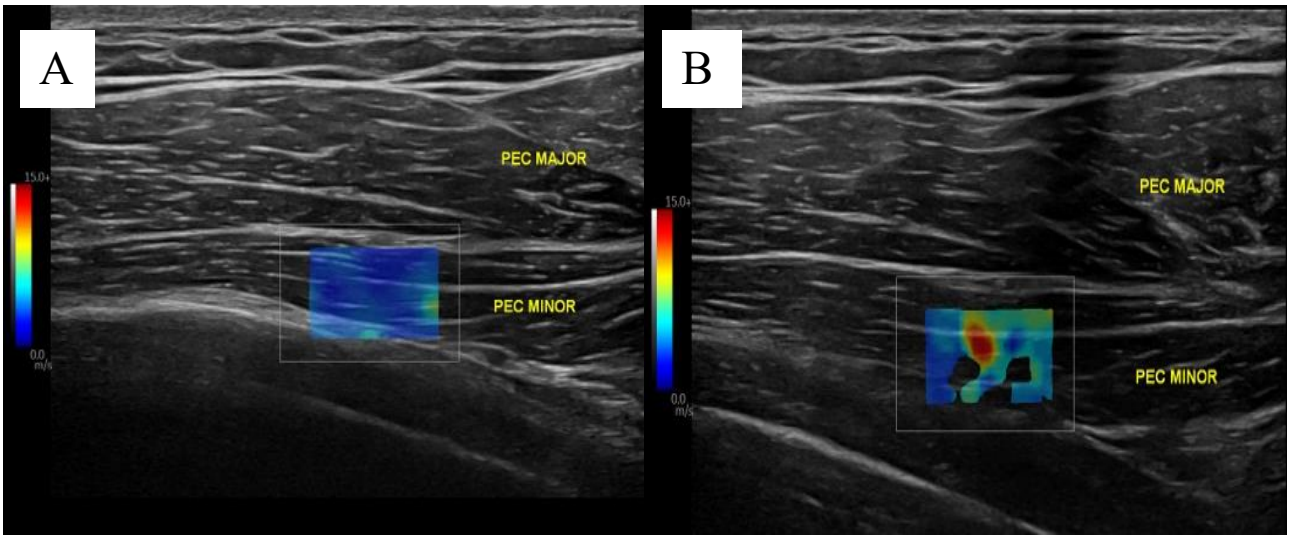

Figure 3. Elastogram examples. *A) An elastogram of the PMin with adequate coloring and free of artifacts. B) An elastogram of the PMin demonstrating areas devoid of color and abnormal coloring*

5.3.2 Forward Shoulder Posture

FSP was assessed using the double-square method [26]. First, the acromion process was palpated and marked with a permanent marker. The participant was then instructed to stand with their back against a wall, not correcting their posture. The distance between the wall and the

participant's acromion process was measured using a modified combination square (Figure 4) three times on each side. Between each measurement, the participant was instructed to step forward and shake out their arms before returning to the wall. The average of the three measurements were used for analysis.

Figure 4. Double square measurement. *The acromion process was marked using a permanent marker and the distance was measured using a modified combination square*

5.3.3 Pectoralis minor length

PML was measured with the participants lying supine on a plinth [158]. A researcher palpated and marked the anterior-inferior edge of the 4th rib, positioned 1 finger-width lateral to the sternum and the coracoid process. Subsequently, a soft measuring tape was used to measure the distance between the two landmarks (Figure 5). The distance was measured three times, and the mean used for analysis.

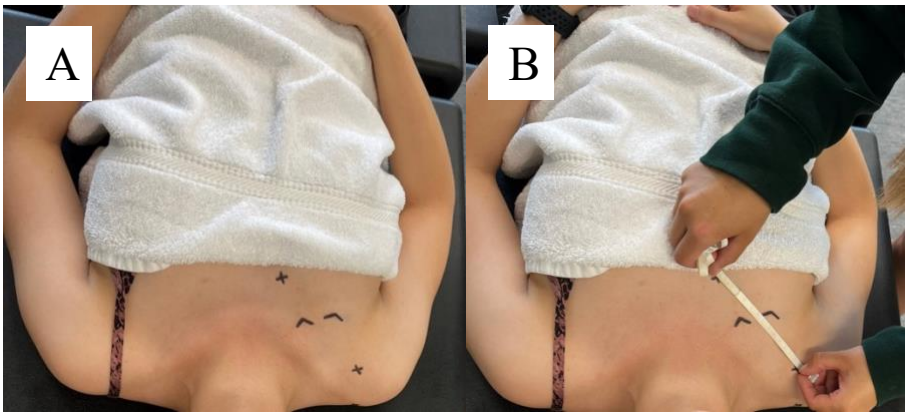

Figure 5. PML measurement. *A) The coracoid process and anterior inferior aspect of the 4th rib were marked using a permanent marker. B) The distance between the two marks was measured using a soft measuring tape.*

5.3.4 Stretch intensity

Perceived stretch intensity was assessed while performing HaROM, utilizing a validated stretch intensity scale [159] to determine the joint angle for the two stretch intensities (moderate = 60; maximal = 100). The scale, ranging from 0 (indicating no stretch) to 150 (representing the maximum attainable ROM with pain) and 100 being the maximum achievable ROM without pain (Figure 6).

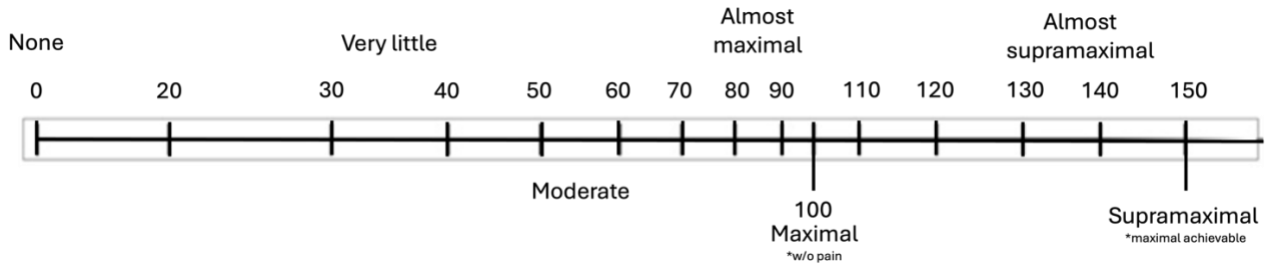

Figure 6. Stretch Intensity Scale

Adapted from Freitas et al. (2015) [157]

5.3.5 Horizontal Abduction Range of Motion

Participants stood beside a pole anchored to weighted platform and flexed their elbow 90°. From this position, they were instructed to abduct their shoulder to 90°, so that their forearm and palm rested on the pole (Figure 7A).

Figure 7. Stretch position.*A) Participant was instructed to place the dorsal aspect of their hand and forearm on the post with their shoulder abducted to 90° and their elbow flexed to 90°. B) From this position, the participant walked forward to increase horizontal abduction [160]. C) With the participant in the starting position, and researcher placed the goniometer on the participant's shoulder, aligning the center of the device with the participant's acromion process [160]. The arm was then aligned with the center of the humerus, with a starting point of 90°. Photographs B & C were used with permission from © 2026 Sarah A. Bohunicky*

Participants were then instructed to walk forward (resulting in shoulder horizontal abduction) until they felt a stretch in their pectorals equivalent to 100 on the stretch intensity scale. Once 100 was attained, a researcher measured the horizontal abduction angle using a goniometer (Figure 7B & 7C). The procedure was repeated three times with the average angle used to determine the participants position for the maximal stretch condition.

5.3.5 Control Condition

Following baseline measurements, participants rested for a 150-seconds seated on a plinth angled at 30° at the start of the first session only as a CON. Immediately after, PMaj and PMin stiffness, FSP, PML and HaROM were measured (i.e. POST for the CON). Following 5-minutes rest, PMaj and PMin stiffness, FSP, PML and HaROM were measured again to serve as baseline measurements (PRE) for the stretch intervention randomly assigned for that visit.

5.3.6 Stretch intervention

Participants performed three sets of self-administered static pectoral stretching lasting 30 seconds each, with the initial intensity at moderate (60 on the stretch intensity scale) or maximal (100 on the stretch intensity scale). The stretch intervention used the same positioning as the HaROM assessment (see Figure 7a). The moderate intensity stretch involved having the participant step forward until they reported a stretch intensity of 60. During the first repetition, the participant's horizontal abduction angle was measured using a goniometer and their arm was returned to this angle for all subsequent repetitions. For the maximal intensity stretch, the participant stepped forward until their shoulder achieved the same horizontal abduction angle that was collected at baseline, which was measured as 100 on the stretch intensity scale. The asked to rate their intensity again to examine any differences in HaROM that may be influenced by stretch tolerance. Once the desired joint angle was attained, the position was maintained for the first 30-second stretch interval. Subsequently, the participant returned their arm to their side for a 30-second rest period. The stretch interval was repeated two more times with an additional rest interval between repetitions two and three.

During each 30-second stretching interval, participants were prompted at the 15-second and 30-second mark to indicate their perceived intensity of the stretch. These values were used to monitor variations in perceived intensity during the three repetitions at each stretch intensity. The averages for each time point and stretch intensity were used for analysis.

5.4 Data Processing

The velocity (m/s) of shear wave propagation calculated by the system quantified stiffness. A blinded research assistant assessed each elastogram for artifacts and determined the largest area possible within the ROI of each elastogram within the same timepoint and ensured that the diameter of the ROI and location remained constant (Figure 8). The mean velocity of three separate elastograms were determined with the mean used for analysis.

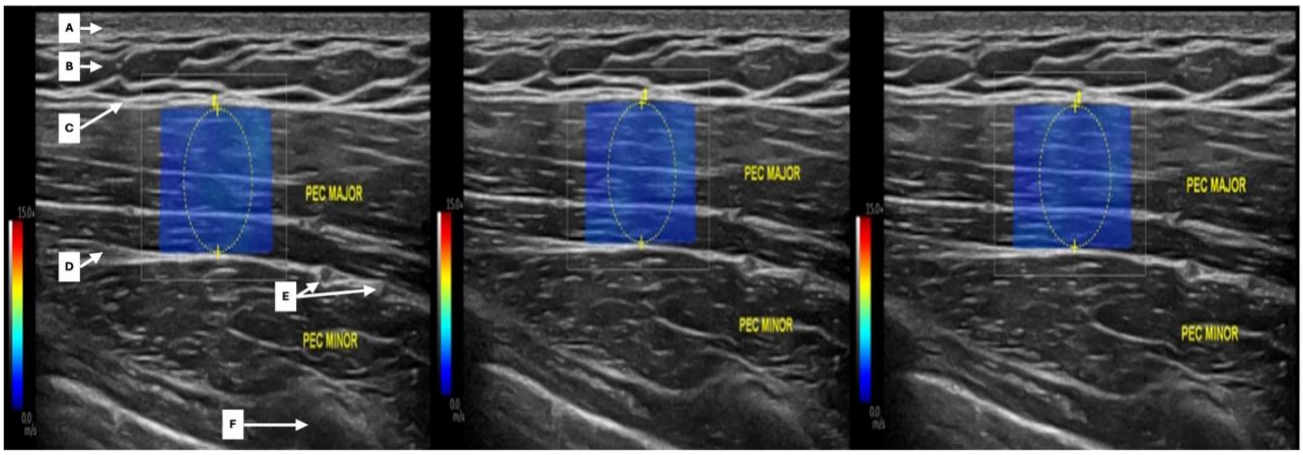

Figure 8. ROI Measurement. *Three elastogram images collected from the PMaj. The ROI was set at a diameter of 0.88cm and placed in approximately the same location on each image. A) Cutaneous (Skin Layers), B) Subcutaneous Fat Layer, C) Subcutaneous Fascia (Pectoral Fascia), D) Intermuscular Fascia (Clavipectoral Fascia), E) Large Vessels, F) Coracoid Process*

5.5 Statistical Analysis

Statistical analyses were conducted using jamovi (The jamovi project, Version 2.7, Sydney. Australia)[161]. Normality was assessed using the Shapiro-Wilk test for normality with

p<0.05 indicating a violation of normality. Outliers were considered standardized residuals with values greater than 3 standard deviations from the regression line [162]. Data that deviated from normality were transformed using a log10 transformation to address positive skewness and outliers were evaluated to identify their source. This was done by reviewing the raw data (e.g. images, all measurement values) to determine if data processing errors occurred and consideration of if the values were physiologically possible.

Sphericity was assessed using Mauchly's where a violation of sphericity was indicated by $p < 0.05$. Where sphericity was violated, estimates of epsilon were used to adjust the degrees of freedom used to calculate the p-value. Two-way repeated measures ANOVAs were conducted to examine the interaction between condition (CON, MOD, and MAX) and time (PRE and POST) on PMaj and PMin stiffness, PML, FSP and ROM in the CL limb. Sex was used as a between subjects' factor to examine if the observed effects unilateral SS on the dependent variables were different between males and females.

Additionally, multiple linear regression analyses were conducted for the MOD and MAX conditions to evaluate the predictive relationship between the intervention and CL limbs. For each model, the ROM change score of the CL limb served as the dependent variable. The predictors included the stretched limb ROM change score and stretching intensity change score, alongside sex as a categorical factor. The overall model fit was evaluated using the coefficient of determination (R^2), and individual predictor impacts were examined using unstandardized coefficients (β) and associated t-tests.

Statistical significance was set at $p < .05$. In cases where significant interactions were observed, post-hoc Bonferroni corrected t-tests were conducted to reduce the chance of Type 1 errors and to identify main effects. Bonferroni was selected over other methods of correction

because it allowed for logical comparisons to occur between time, condition and sex (e.g. the PRE and POST for each variable and condition vs. unnecessary comparisons such as comparing PRE of CON to POST of MAX). Effect size was calculated to provide a measure of the magnitude of the observed interactions. Small, medium, and large two-way interaction effect sizes (η_p^2) were interpreted as 0.01, 0.06, and 0.14, respectively [163].

6. Results

6.1 Intervention Limb Results

As this study was conducted as part of a larger study examining the intervention limb, the following intervention limb results are reported in order to provide a comparison between the intervention and CL limb[160].

Data was processed and analyzed in a similar manner with all assumption checks and effect size thresholds remaining consistent between the intervention and CL limb[160]. The only difference being that influence sex was not examined for its potential effects on the PRE-POST measurements across the three conditions. Two-way (*Intervention*Time*) ANOVAs were used to examine the interaction between time and condition for all dependent variables[160].

Table 1. Intervention Limb ANOVA

	CON		MOD		MAX		*f*	*p*	η_p^2	β
	PRE	**POST**	**PRE**	**POST**	**PRE**	**POST**				
FSP (cm)	11.1 (1.4)	10.8 (1.6)	10.9 (1.6)	10.7 (1.6)	10.9 (1.6)	10.8 (1.6)	1.809	.173	.059	.363
PML (cm)	14.36 (1.35)	14.37 (1.34)	14.28 (1.28)	14.68 (1.31)	14.25 (1.33)	14.35 (1.3)	1.241	.297	.041	.260
HaROM (degrees)	105 (5)	103 (5)	104 (5)	107 (6)	103 (5)	108 (5)	58.195	<.001*	.667	1
PMaj SWV (m/s)	2.87 (.69)	2.96 (.85)	2.99 (.62)	3.06 (.80)	2.80 (.48)	2.96 (.51)	.197	.821	.007	.079
PMin SWV (m/s)	2.28 (.65)	2.17 (.47)	2.21 (.65)	1.99 (.49)	1.99 (.63)	2.09 (.74)	1.3	.280	.043	.271

Mean and standard deviation M(SD) are reported for the three conditions
n = 30, FSP = Forward shoulder posture, PML = Pectoralis minor length, HaROM = Horizontal abduction range of motion, PMaj = Pectoralis major, PMin = Pectoralis minor, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition
** Denotes significant effects (p<.05). Values as reported in Bohunicky, 2026[160]*

In the intervention limb, data for the HaROM, PMaj SWV and PMin SWV were not normally distributed and applying transformations did not correct the distribution. Thus, all analyses were conducted on the raw data. HaROM was the only variable that showed a significant change from PRE-POST across conditions (Table 1). Both condition (p=.029) and time (p<.001) showed to have a significant effect on HaROM. Post hoc testing revealed that HaROM decreased significantly in the CON (p<.001, mean difference -2.6°, 95% CI -1.6° to -3.6°) and increased following MOD (p<.001, mean difference 2.7°, 95% CI 4.0° to 1.4°) and MAX (p<.001, mean difference 2.7°, 95% CI 4.0° to 1.4°).

For the patient reported stretch intensity, mean participant ratings for their initial intensity were 60 for MOD and 98 for MAX (Table 2). At the end of the final repetition, participant's mean intensity ratings were 52 for MOD and 81 for MAX with the mean change from PRE to POST being -9 for MOD and -17 for MAX.

Table 2. Intervention Limb Intensity Descriptives

	PRE				POST				Mean Change
	Mean	**SD**	**MIN**	**MAX**	**Mean**	**SD**	**MIN**	**MAX**	
MOD (*n = 30*)	60	0	60	60	52	15	20	80	-9
MAX (*n =30*)	98	6	70	100	81	17	30	100	-17

Descriptive statistics on the subjective intensity rating on the stretch intensity scale[15] [7]*(Figure 6). MOD = Moderate condition, MAX = Maximal condition*

6.2 Contralateral Limb Results

There were two outliers as assessed by standardized residuals. Both occurred for the same participant during the pre-measurements during CON for the PMaj and PMin. These data points were reviewed to ensure there were no data processing errors. The values were determined to be correct and were physiologically possible, so they were kept in the data set.

Data was not normally distributed for PMaj and PMin shear wave velocity (SWV) as assessed by Shairo-wilk test for normality (p<.05) and thus a log10 transformation was applied. The distribution for all SWV variables was corrected except for the PRE CON for PMaj and PMin. Removing the outliers would have improved the fit of the model in both instances but their presence/removal did not impact the outcome of the analysis. As such, the repeated-measures ANOVA for these variables were carried out on the log10 transformed data, maintaining the outliers. Though the violation of normality occurred, the ANOVA was still run as it is robust to violations of normality and to maintain the same statistical analysis for all dependent variables. Mauchly's test confirmed that the assumption of sphericity was met for all variables regarding condition and the time × condition interaction (p>.05), except for PML. For PML, sphericity was violated for the condition factor consequently, Greenhouse-Geisser corrections were applied (Table 6).

6.2.1 Repeated Measures ANOVAs

Biological sex (referred to a sex for the remainder of this document) did not influence the outcome of the HaROM, and time, condition and sex did not have a combined effect (Table 3). Both time and condition had a significant effect on HaROM with a statistically significant interaction also occurring between the PRE-POST measurements and conditions (CON, MOD & MAX) (Table 3).

Table 3. Repeated Measures ANOVA for CL HaROM

	PRE	***POST***		***dF***	***F***	***p***	η_p^2
CON	*108 (6)*	*108 (6)*	***Sex***	*1,28*	*3.55*	*.070*	*.112*
MOD	*107 (6)*	*110 (7)*	***Time***	*1,28*	*40.74*	*<.001**	*.593*
MAX	*109 (5)*	*112 (5)*	***Condition***	*2,56*	*8.26*	*<.001**	*.228*
			Time * Condition	*2,56*	*8.81*	*<.001**	*.239*
			Time * Condition * Sex	*2,56*	*0.91*	*.409*	*.031*

Mean and standard deviation M(SD) are reported for the three conditions
n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition
** Denotes significant effects (p<.05).*

Post-hoc testing indicated no differences in ROM between conditions at the PRE. There was no change in ROM from PRE to POST in CON. However, ROM increased significantly at POST following MOD (M_{diff}= -2.67, $p_{Bonferroni}$=.004) and MAX (M_{diff}= -3.03, $p_{Bonferroni}$<.001) SS (Figure 9). At POST, ROM was significantly greater following MAX SS than CON (M_{diff}= -3.70, $p_{Bonferroni}$<.001), while MOD did not differ from CON or MAX SS.

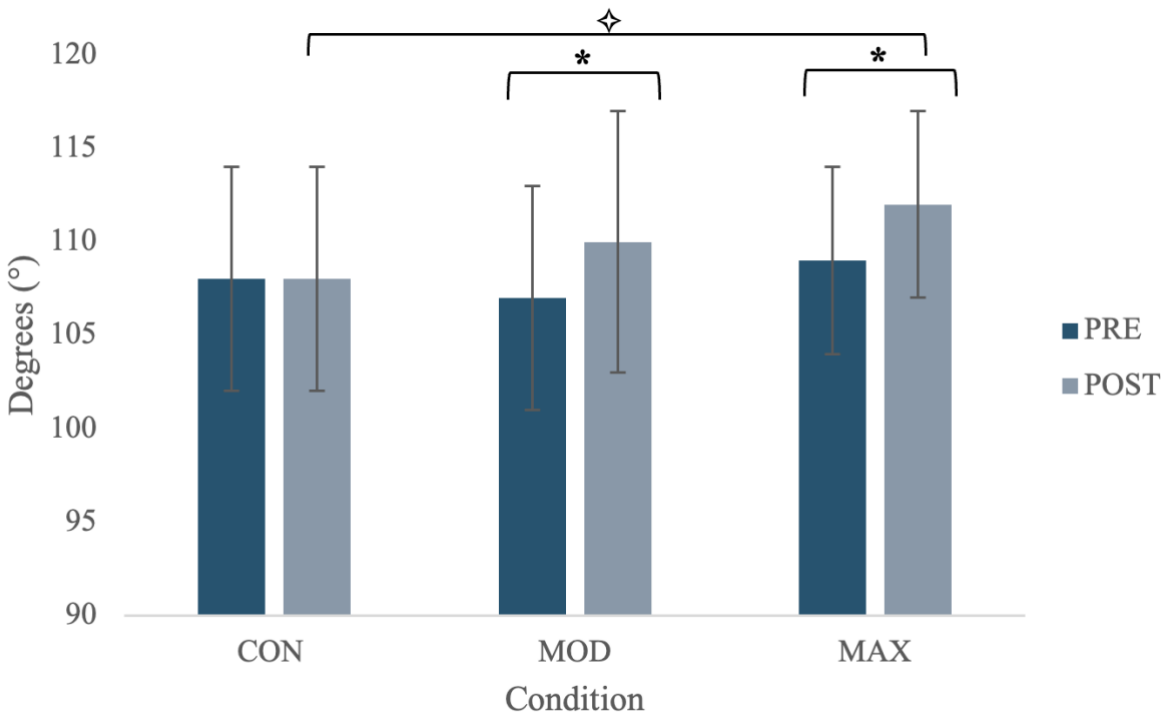

Figure 9. Mean CL ROM PRE and POST intervention. *Error bars represent ± 1 standard deviation*

n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition
** denotes significant within-condition change from PRE to POST (p<.05; MOD: p = .004; MAX: p<.001) ✧ denotes significant POST differences between the CON and MAX condition (p<.001)*

Sex did not influence PMin SWV, and no combined influence of time, condition and sex existed (Table 4). A significant change occurred between PRE and POST measurements for PMin SWV, but the individual conditions did not have an impact on the outcome. Additionally, the PRE to POST change between was different between the three conditions (Table 4).

Table 4. Repeated Measures ANOVA for CL PMin SWV

	PRE	***POST***		***dF***	***F***	***p***	η_p^2
CON	*1.41 (0.37)*	*2.11 (0.58)*	***Sex***	*1,28*	*1.52*	*.228*	*.051*
MOD	*1.75 (0.24)*	*2.00 (0.41)*	***Time***	*1,28*	*32.63*	*<.001**	*.538*
MAX	*2.10 (0.38)*	*1.98 (0.41)*	***Condition***	*2,56*	*3.55*	*.070*	*.112*
			Time * Condition	*2,56*	*24.42*	*<.001**	*.466*
			Time * Condition * Sex	*2,56*	*0.75*	*.478*	*.026*

Mean and standard deviation M(SD) are reported for the three conditions
*n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

Post-hoc testing revealed significant baseline (PRE) differences between all three conditions with CON differing from MOD ($p_{Bonferroni}$<.001) and MAX ($p_{Bonferroni}$<.001) and MOD differing from MAX ($p_{Bonferroni}$<.001) (Figure 10). Between the three conditions, significant stiffness increase occurred from PRE to POST in the CON (M_{diff}= -0.18, $p_{Bonferroni}$<.001) and MOD conditions (M_{diff}= -0.06, $p_{Bonferroni}$=.032), while no change occurred within the MAX condition. PMin SWV did not differ among the POST values for all three conditions.

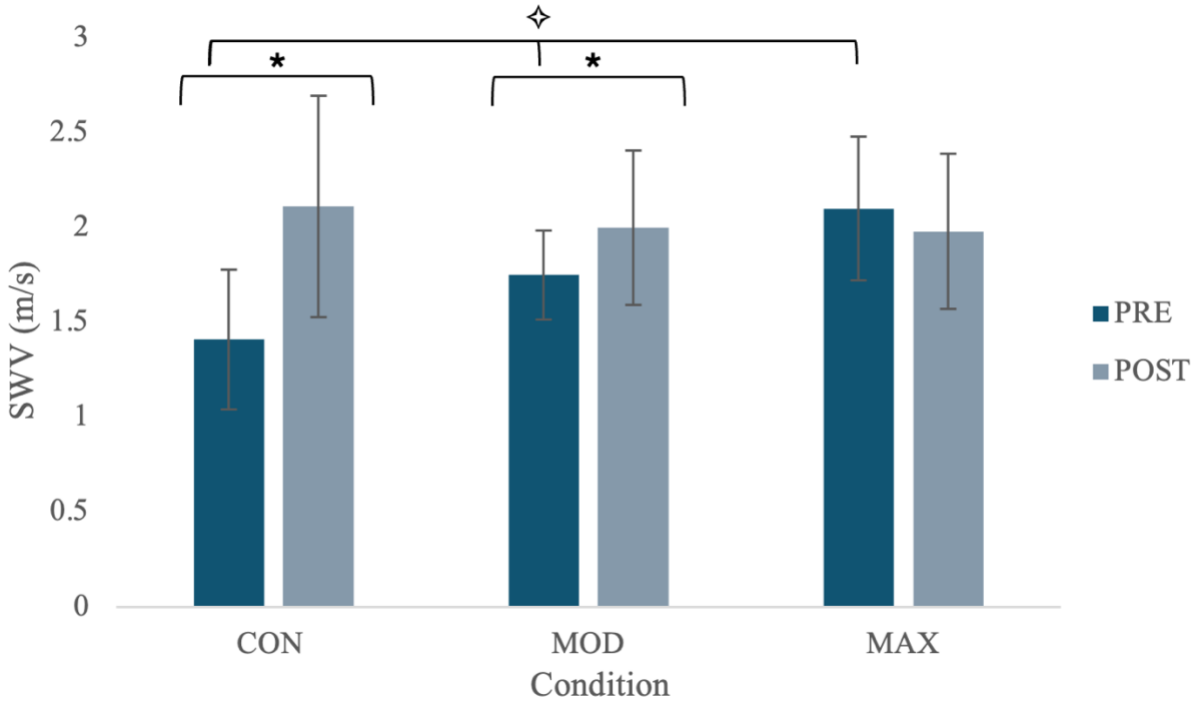

Figure 10. Mean CON PMin SWV PRE and POST conditions. *Error bars represent ± 1 standard deviation*

n = 30, m/s = meters per second, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition, denotes statistically significant within-condition changes from PRE to POST (p<.05), ✧ denotes significant baseline (PRE) differences across the three conditions (CON-MOD: p<.001; CON-MAX: p<.001; MOD-MAX: P<.001)*

A significant main effect of sex existed for FSP, PML and PMaj SWV (Table 5-7), demonstrating structural baseline differences between biological males and females for FSP, PML and PMaj SWV. For all three variables, time and condition did not influence the outcome and there were no combined effects of sex, time and condition observed. Additionally, there were no significant changes from PRE to POST between all conditions. This demonstrates a lack of CL changes in FSP, PML and PMaj SWV following unilateral pectoral stretching.

Table 5. Repeated Measures ANOVA for CL FSP

	PRE	***POST***		***dF***	***F***	***p***	η_p^2
CON	*11.2 (1.41)*	*11.1 (1.43)*	***Sex***	*1,28*	*8.80*	*.006**	*.239*
MOD	*11.2 (1.50)*	*11.2 (1.60)*	***Time***	*1,28*	*3.94*	*.057*	*.123*
MAX	*11.0 (1.35)*	*10.9 (1.42)*	***Condition***	*2,56*	*3.03*	*.056*	*.098*
			Time * Condition	*2,56*	*0.71*	*.497*	*.025*
			Time * Condition * Sex	*2,56*	*0.17*	*.846*	*.006*

*Mean and standard deviation M(SD) are reported for the three conditions n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

Table 6. Repeated Measures ANOVA for CL PML

	PRE	***POST***		***dF***	***F***	***p***	η_p^2
CON	*14.6 (1.35)*	*14.7 (1.38)*	***Sex***	*1,28*	*14.50*	*<.001**	*.341*
MOD	*14.6 (1.49)*	*14.6 (1.53)*	***Time***	*1,28*	*0.30*	*.590*	*.010*
MAX	*14.7 (1.33)*	*14.8 (1.36)*	***Condition***	*1.53, 42.85[a]*	*0.62*	*.503*	*.022*
			Time * Condition	*2,56*	*1.66*	*.200*	*.056*
			Time * Condition * Sex	*2,56*	*0.94*	*.396*	*.033*

*Mean and standard deviation M(SD) are reported for the three conditions n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition; * denotes significant effects (p<.05), a Greenhouse-Geisser correction applied due to violation of the assumption of sphericity (W=0.693, p = .007,ϵ = 0.765)*

Table 7. Repeated Measures ANOVA for CL PMaj SWV

	PRE	***POST***		***dF***	***F***	***p***	η_p^2
CON	*2.71 (0.58)*	*2.76 (0.55)*	***Sex***	*1,28*	*4.54*	*.042**	*.139*
MOD	*2.70 (0.46)*	*2.72 (0.51)*	***Time***	*1,28*	*0.15*	*.701*	*.005*
MAX	*2.66 (0.38)*	*2.65 (0.50)*	***Condition***	*1.53, 42.85[a]*	*0.09*	*.845*	*.003*
			Time * Condition	*2,56*	*0.14*	*.869*	*.005*
			Time * Condition * Sex	*2,56*	*0.76*	*.473*	*.026*

*Mean and standard deviation M(SD) are reported for the three conditions n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05), a Greenhouse-Geisser correction applied due to violation of the assumption of sphericity (W=0.488, p <.001,ϵ = 0.661)*

6.2.2 Percent Change Score Repeated Measures ANOVAs

To further examine the impact of the stretch intervention on all variables with the elimination of baseline differences. A repeated measures ANOVA was conducted on the percent change score in PMin SWV , PMaj SWV, PML, FSP and HaROM for the CON, MOD and MAX conditions. No significant effects were found in FSP, PMaj SWV and PML (Appendix A-C).

Significant differences were found between conditions for HaROM (Table 8). Post-hoc testing revealed significant differences between CON and the two interventions with CON differing from MOD ($p_{Bonferroni}$=.022) and MAX ($p_{Bonferroni}$=.002) and no significant difference occurring between MOD and MAX ($p_{Bonferroni}$= 1.000) (Figure 11).

Table 8. Repeated Measures ANOVA for CL HaROM Percent Change Scores

	M(SD)		***dF***	***F***	***p***	η_p^2
CON	*0.13 (2.67)*	***Condition***	*2, 56*	*8.28*	*<.001**	*.228*
MOD	*2.49 (3.21)*	***Condition*Sex***	*2, 56*	*0.99*	*.380*	*.034*
MAX	*2.83 (2.41)*					

*n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

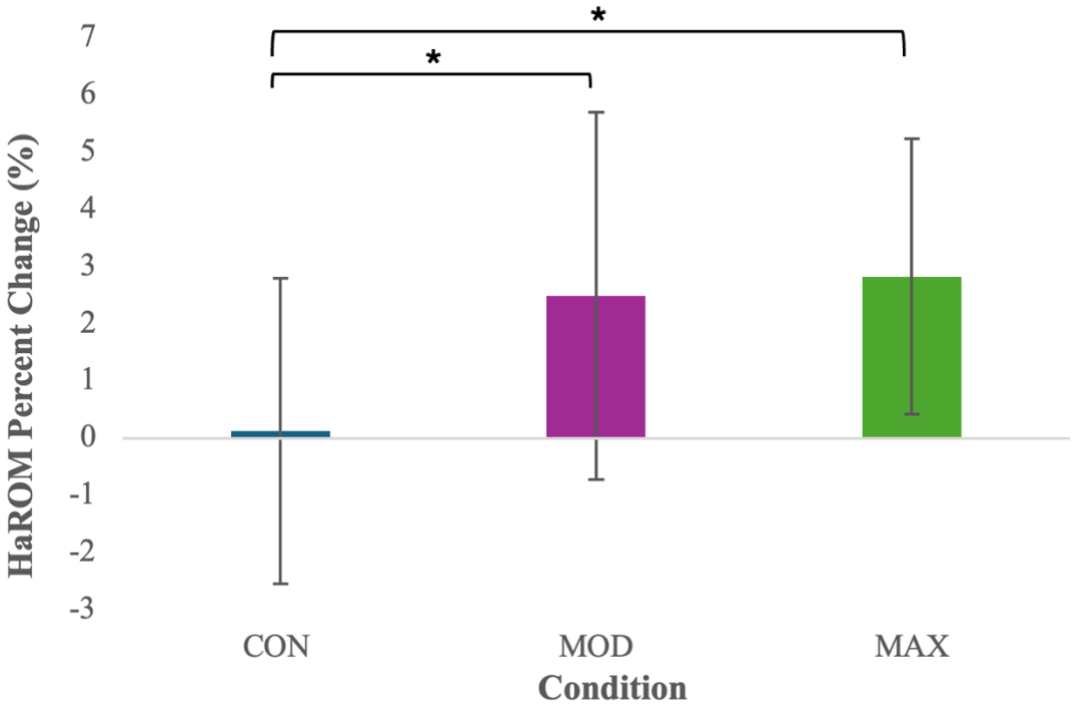

Figure 11. Mean CL HaROM Percent Change Scores. *Error bars represent ± 1 standard deviation*

n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition, denotes statistically significant between condition differences (p<.05)*

Significant differences were found between conditions for PMin SWV (Table 9). Post-hoc testing revealed significant differences between all three conditions with CON differing from MOD ($p_{Bonferroni}$=.001) and MAX ($p_{Bonferroni}$<.001) and MOD differing from MAX ($p_{Bonferroni}$= .011) (Figure 12).

Table 9. Repeated Measures ANOVA for CL PMin SWV Percent Change Scores

	M(SD)		***dF***	***F***	***p***	η_p^2
CON	*58.3 (58.3)*	***Condition***	*2,56*	*24.23*	*<.001**	*.464*
MOD	*14.8 (24.1)*	***Condition*Sex***	*2,56*	*1.68*	*.196*	*.057*
MAX	*-4.80 (18.7)*					

*n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

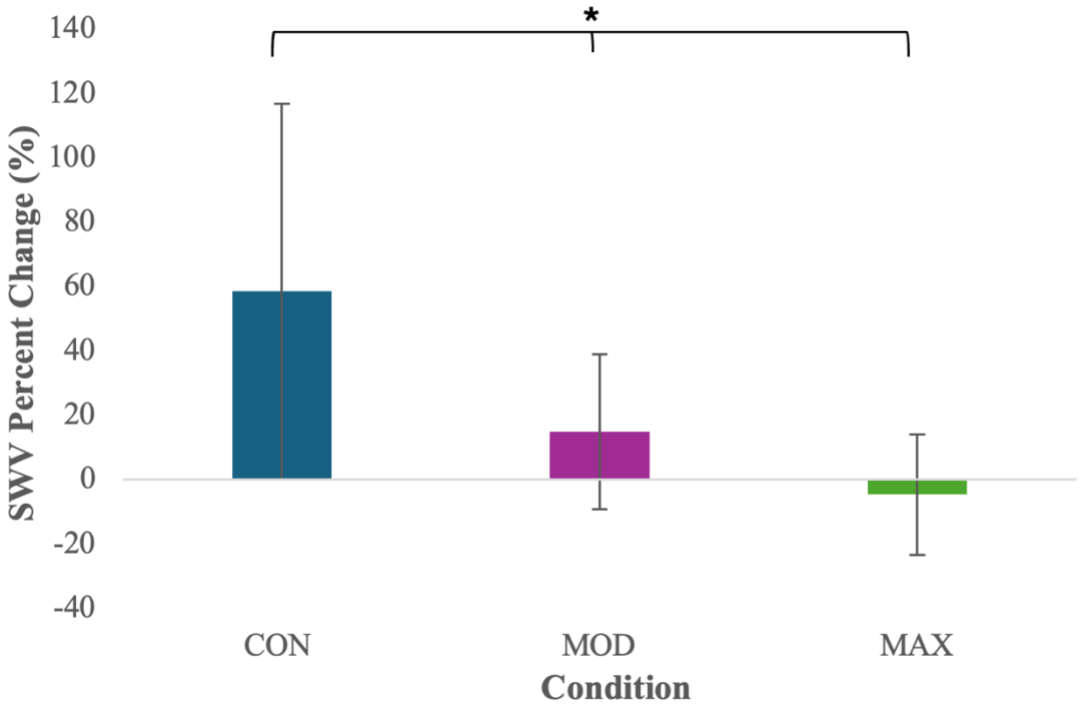

Figure 12. Mean CL PMin SWV Percent Change Scores. *Error bars represent ± 1 standard deviation*

n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition, denotes statistically significant between condition differences (p<.05)*

6.2.3 Multiple Linear Regression Models

Two separate multiple linear regression analyses were conducted to determine if changes in perceived intensity, changes in ROM of the stretched limb, and sex predicted ROM change of the CL limb during both stretching conditions (Table 10).

Table 10. Multiple linear regression models predicting CL ROM Change

	B	*SE*	*t*	*p*	R^2
Model 1: MAX					*.224*
Intercept	*2.479*	*0.923*	*2.686*	*.012*	
Intensity Change	*0.069*	*0.029*	*2.356*	*.026**	
INT HaROM Change	*0.398*	*0.169*	*2.349*	*.027**	
Sex	*-0.332*	*0.879*	*-0.378*	*.709*	
Model 2: MOD					*.097*
Intercept	*1.676*	*1.185*	*1.414*	*.169*	
Intensity Change	*-0.056*	*0.045*	*-1.247*	*.224*	
INT HaROM Change	*0.187*	*0.184*	*1.020*	*.317*	
Sex	*0.011*	*1.311*	*0.009*	*.993*	

n = 30, MOD = Moderate condition, MAX = Maximal condition, HaROM = Horizontal abduction range of motion
** denotes significant effects (p<.05)*

For the MAX condition, the model explained approximately 22.4% of the variance in the CL limb's ROM change. Both intensity change and stretched limb ROM change were statistically significant positive predictors of non-stretched ROM adaptations (Table 10). Sex was not a significant predictor of ROM in either condition. In contrast, the regression model of the MOD stretching condition was not statistically significant and accounted for only 9.7% of the variance in the CL ROM change. Intensity change and ROM change of the stretched limb did not predict HaROM change in the CL limb.

7. Discussion

The current study explored the impact of two unilateral static stretching intensities on the CL limb, examining their effects on HaROM, FSP, PML, PMin SWV and PMaj SWV. We sought to observe whether the extent of change occurring in these variables were affected by the intensity at which the stretch was performed. It was also important to understand whether the stretch intervention elicited change in the structural variables such as FSP, PML and SWV in

conjunction with and increase in HaROM. We also aimed to understand whether the intensity change score and HaROM change score in the dominant limb influenced the degree of HaROM change in the CL limb. Using these objectives, the project aimed to address four main hypotheses (Table 11).

Table 11. Summary table of study hypotheses and relevant results

Hypothesis	Accepted/Rejected	Explanation
1) Effects will be seen in FSP and HaROM. Given that FSP and HaROM can be heavily influenced by the neuromuscular adaptations of stretching, I hypothesize that FSP will decrease and HaROM will increase following stretching	**Partially Accepted** - Increases in HaROM occurred from PRE to POST for MOD and MAX (*Accepted*) - No change in FSP was observed in all three conditions (*Rejected*)	- A significant interaction between time and condition (p<.001, η_p^2 = .239) existed for HaROM, with post-hoc testing indicating that unilateral stretching induced increases in the CL limb for the MOD and MAX conditions - No statistically significant change occurred from PRE to POST in FSP across all conditions (p = .497)
2) No effects will be seen in PMaj stiffness, PMin stiffness and PML. As these variables are significantly related to the viscoelastic properties of the muscle, I hypothesize that no changes in these variables will occur, as they are not affected by the acute crossover effects of SS.	**Partially Accepted** - No change in PMaj and PML were observed for all three conditions (*Accepted*) - Increases in PMaj SWV were seen from PRE to POST for the CON and MOD conditions (*Rejected*)	- No statistically significant interactions occurred between time and condition for PML (p = .200) and PMaj (p = .869) - A statistically significant change was seen between condition and time (p<.001, η_p^2 = .466) for PMin with post-hoc testing indicating increases in PMin from PRE to POST for the CON and MOD conditions
3) The effects seen in FSP and HaROM will be greater at higher intensities. Given that the neuromuscular cross-over of movement is greater at higher intensities and with transfer from the dominant to non-dominant limb, I hypothesize that greater differences will occur in the maximal stretch condition than in the moderate condition.	**Partially Accepted** - MAX condition resulted in greater increases in CL HaROM (*Accepted*) - FSP showed no change for the MOD and MAX conditions (*Rejected*)	- A greater statistically significant change in HaROM was observed for the MAX condition (p<.001) - No statistically significant change was observed in FSP for the MOD and MAX conditions (p = .497) indicating the stretch had no effects on FSP
4) The pre-post differences in intensity and ROM on the intervention limb will have an influence on the degree of change occurring in the CL limb. I hypothesize that the maximal intensity condition will have a greater effect than the moderate condition	**Accepted**	- The dominant limb intensity and HaROM change score had a direct influence on the HaROM increase observed in the CL limb. With the change scores predicting 22.4% of the increase for the MAX condition

In line with our first hypothesis (Table 11), the most significant finding in this study was significant interaction between time and condition for CL HaROM. This finding confirms that acute unilateral stretching of the dominant limb pectorals results in CL ROM gains at both a MOD and MAX stretch intensity compared to the CON condition. The observed improvements in HaROM for both conditions occurred primarily without structural changes in FSP, PML and PMaj SWV. The only structural changes observed occurred in the PMin SWV with increases in stiffness occurring for the CON and MOD condition following the stretch and the MAX condition exhibiting no change and baseline differences occurring between the three conditions. The acute increase in HaROM without structural change closely aligns with the sensory theory of stretching that was discussed previously [12-15]. According to this framework, acute improvements in ROM can occur independently of mechanical tissue deformation or adjustment in musculotendinous unit resting length. Considering this, the acute HaROM increase in the CL limb was likely to have occurred primarily from cross-over of the central nervous system input [35-38, 154].

The main sensory theories that can assist in explaining the cross-over are the spillover hypothesis and endogenous pain modulation theory [14]. Spillover was likely to have occurred from activation of the mechanoreceptors (Type I and II muscle spindles) and nociceptors (Group III and IV unmyelinated afferents) in the dominant limb that projected directly to the somatosensory and primary motor cortices on the CL side[139-141]. The carry-over of nociceptor activation likely resulted in exercise induced hypoalgesia on the CL side causing endogenous pain modulation [14]. Communication between hemispheres can cause acute, global attenuation of the reflexes and a systematic increase of stretch tolerance to the matching pathway of the CL limb [139-141]. This means that when the CL limb was moved into horizontal abduction following

the stretch of the other limb, the alpha-motoneuron pool likely experienced less resistance from the reflex receptors, allowing participants to achieve greater ROM before hitting the stretch threshold of 100 on the SIS. In addition, the spill over hypothesis can also assist in addressing the intensity dependent carry-over seen in the regression modeling between the dominant and CL limb.

The multiple linear regression data (Table 10) demonstrates an intensity-dependent effect in the cross-education pathways. For the MAX stretching condition, the prediction model was highly effective, explaining 22.4% of the CL limb HaROM increase ($R^2 = .224$). Both changes in perceived stretching intensity and ROM on the intervention limb were powerful positive predictors of the CL HaROM gains ($p = .026$ and $p = .027$, respectively). Alternatively, the model evaluating the MOD condition was insignificant, indicating that the same variables were not effective at predicting improvements in the CL limb ($R^2 = .097$). The difference in prediction highlights that the upper-limb flexibility cross-education is not a linear phenomenon and requires greater neuromuscular stimuli to activate central sensory pathways. In the MOD stretching condition, the afferent input entering the dorsal horn of the spinal cord was likely insufficient to trigger cross-over of the dominant motor pathway or analgesic release as described by the bilateral access hypothesis [128]. In this hypothesis, it is described that the level of movement transfer is intensity-dependent, with higher intensity movement resulting in larger degrees of corticospinal excitability [125, 128].

Therefore, when a participant completed the MAX condition it is likely that a more significant recruitment of high-threshold mechanoreceptors occurred changing the central drive and CL outcome. The close association between the intervention ROM gains and the CL gains supports the third and fourth hypotheses that the degree of transfer would be intensity dependent.

This means that the more intense the stretch is on the intervention limb, the greater the central nervous system response resulting in a greater stretch tolerance and ROM gains in the CL limb. This supports the belief that higher intensity stretching may be more effective than lower intensity stretching when the goal is to increase or maintain the HaROM of the CL limb during unilateral stretching.

The sensory theory explanation of the HaROM gains is further supported by the lack of change observed in FSP, PML and PMaj SWV. As reported in Table 4-6, there were no significant main effects of time or condition nor any multi-factor interactions for FSP, PML and PMaj SWV in the CL limb. This supports the second hypothesis (Table 11) as the variables that were meant to examine structural adaptations such as stiffness (PMaj SWV), muscle length (PML) and FSP exhibited no change in conjunction with the increase in HaROM. Though this supports the acute effects of unilateral SS being primarily sensory, it is unknown whether changes in the structural variables would exhibit more change with a longer duration protocol.

Though the other structural variables exhibited no change, the PMin SWV presented a significant deviation from the hypothesis that no structural adaptations would be observed. A significant interaction between time and condition was detected with a large effect size (η_p^2 = .466). The post-hoc testing also exhibited interesting results as statistically significant changes from PRE to POST were only observed in the CON and MOD conditions (Figure 10). There were also significant baseline data imbalances that existed across all three conditions even with the random allocation of intervention and testing days. Instead undergoing localized tissue relaxation or no change, muscle stiffness values actually increased significantly over time within both the CON (1.41m/s to 2.11m/s) and MOD (1.75m/s to 2.00m/s) conditions, while MAX (2.10m/s to 1.98m/s) showed minimal change. The observations could potentially be explained

by the concept of thixotropy. Thixotropy states that resting tissue stiffness operates as a time-dependent variable where stiffness decreases with movement and increases with immobilization [45,46]. When individuals remain static or immobilized for extended periods, the cross-bridges between actin and myosin filaments gradually settle which can allow the surrounding hyaluronan in the deep pectoral fascia to thicken, causing a progressive rise in passive resistance and passive shear modulus stiffness [43,45,46].

During all conditions, participants sat resting on the plinth during data collection. As such, the CL PMin experienced minimal movement or activation during these periods which may have allowed thixotropic settling, causing muscle stiffness values to steadily climb from PRE to POST for all conditions and it is possible that the difference seen in the MAX condition was related to the difference in intensity. PMin SWV during the MAX condition remained largely unchanged from PRE to POST which suggests that though the stretch did not actively reduce the SWV, the cross-over of mechanoreceptor downregulation could have generated sufficient active relaxation to reduce PMin SWV during the MAX condition. The resulting relaxation could have mitigated the passive stiffening that occurs during prolonged physical rest.

Another possibility that could have related to the lack of observed structural modification could be a lack of stimulus sufficient to elicit structural changes. The HaROM utilized for each stretch intervention was determined from the PRE HaROM measurement for the MAX condition and the patient reported 60 on the SIS for the MOD condition. This means that the participant returned to the same angle for each subsequent stretch rather than stretching at the 60 or 100 stretch intensity. Few participants (MOD: n=13; MAX: n=3) consistently reported their perceived stretch as being 60 or 100 at the PRE measurement of all three repetitions meaning that most participants were stretching below the intended intensity for each repetition.

Considering PMin SWV changes have been reported in the intervention limb at both moderate and high intensity stretch it is unusual to observe such a substantial increase in SWV rather than a decrease or lack of change. Future studies could consider having the participant stretch to the prescribed intensity for each repetition and recording the HaROM achieved. This could allow for further examination of how stretch tolerance can change during a prescribed stretching program and may result in different outcomes in the structural dependent variables.

A significant main effect of sex was observed across FSP, PML and PMaj SWV which may be explained by structural and mechanical differences that occur between males and females. These differences include thorax width, scapular size and muscles mass without these differences affecting the outcome of the interventions. This is reflected by the lack of statistical interactions between time and condition for FSP, PML and PMaj SWV that occurred in conjunction with the main effect of sex. Furthermore, the structural differences occurring in conjunction with the increases in HaROM without a main effect of sex highlights that though males and females may differ structurally, their spinal and supraspinal networks respond similarly to unilateral stretching.

7.1 Limitations

There are many factors that may cause limitations in the current study. First, the PMin baseline SWV discrepancies (Figure 10) appeared to be the most substantial limitation. There were statistically significant differences observed in the PRE values across all three conditions, regardless of sex. It is unknown whether these differences occurred because of normal stiffness fluctuations or were an effect of the testing protocol. Baseline PMin SWV should be further examined to better understand the normal fluctuations in PMin SWV over time to more accurately examine how SS may impact these tissues.

Second, SWE allows for the collection of one tissue's SWV at a time which can limit the potential of directly observing the effects of the stretch across all tissues immediately following the stretch. Though the researchers attempted to collect the data as soon as possible following the intervention, each measurement takes time and participant movement, which could have contributed to the observed changes as well as the lack of change that occurred. Additionally, the ROI set by the device only allows for one area of the muscle to be observed at a time. In multi-directional muscles, like the pectoralis major, this can pose a limitation as it is possible that the lack of SWV change observed may not be true for the whole muscle.

Finally, the subjective nature of stretch intensity also poses a limitation in the current study. Participants reported their stretch intensity based on a validated SIS scale but day-to-day variability in pain tolerance and other individual characteristics could have influenced their stretch perception. Tolerance to discomfort is highly variable and can be skewed by prior athletic training, psychological state and pain tolerance. As training age and stretch experience were not controlled for, it is unknown if their presence or absence affected the results. Additionally, the use of NSAIDs and other pain medications was not controlled for during the data collection, which could have also altered pain tolerance.

7.2 Future Directions

The results observed in the current study warrant further investigation into the reliability of the results and applicability across populations. Primarily, further investigation should evaluate the potential of chronic adaptations. As the current project is isolated to purely acute, post-intervention responses, future studies should evaluate multi-week, progressive unilateral pectoral stretching protocols to determine if chronic cross-education can induce lasting structural changes in CL resting posture or resting tissue morphology [27-29].

Further, the inclusion of a clinical group could allow for a broader understanding of how the results of the study may translate to a clinical setting. The testing population was limited to young, asymptomatic, healthy individuals so the direct application to immobilized clinical populations and those with significant FSP would require further investigation [123-125]. Future studies should include this population to provide useful clinical insights into determining if unilateral stretching can assist in mitigating the effects of immobilization.

Considering it has been hypothesized that the current study results have largely resulted from sensory adaptations, further investigations should more closely examine this. Future projects should integrate a method of evaluating the sensory adaptations to unilateral static stretching. Neurophysiological monitoring, such as surface electromyography or transcranial magnetic stimulation, could be used to evaluate muscle reflex responses, muscle guarding and quantify changes in corticospinal excitability and intracortical inhibition within the interhemispheric network. One challenge that limited the use of surface electromyography in the current study was the lack of space on the participant's chest to allow for the placement of electrodes in conjunction with the SWE probe.

Finally, standardization of the pre-test physical activity and controlling for pain mitigating medications should also be considered for future studies. The addition of these controls will allow for more comparisons between subjects and limit the variability of stretch tolerance across visits. Additionally, to reduce the potential thixotropic variations in muscle stiffness, future methods could add a structured, active upper-body warm-up protocol immediately preceding the PRE measurements of all conditions and collect data for each condition on separate days. These additions would assist in ensuring all baseline cross-bridges are mechanically disrupted and standardized prior to data collection.

7.3 Conclusions

In conclusion this master's thesis contributes to evidence that acute unilateral static stretching of the pectoral girdle induces an acute, intensity dependent cross-education effect, increasing HaROM in the CL limb. As the flexibility gains occurred in the absence of modifications in FSP, PML and PMaj SWV mechanisms causing the increase in ROM are likely related to increased stretch tolerance. Additionally, the current study suggests that a maximal stretch intensity may better predict increase range of motion compared to the MOD condition. Clinically, these findings highlight the use of high-intensity unilateral stretching results in acute increases in HaROM in a healthy population.

8. Appendices

Appendix A:

Table 12. Repeated Measures ANOVA for CL FSP Percent Change Scores

	M(SD)		***dF***	***F***	***p***	η_p^2
CON	*-1.04 (3.27)*	***Condition***	*2, 56*	*0.70*	*.502*	*.024*
MOD	*-0.20 (4.18)*	***Condition*Sex***	*2, 56*	*0.14*	*.872*	*.005*
MAX	*-1.19 (3.07)*					

*n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

Appendix B:

Table 13. Repeated Measures ANOVA for CL PML Percent Change Scores

	M(SD)		***dF***	***F***	***p***	η_p^2
CON	*58.3 (58.3)*	***Condition***	*2,56*	*1.71*	*.190*	*.058*
MOD	*14.8 (24.1)*	***Condition*Sex***	*2,56*	*1.15*	*.324*	*.039*
MAX	*-4.80 (18.7)*					

*n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

Appendix C:

Table 14. Repeated Measures ANOVA for CL PMaj SWV Percent Change Scores

	M(SD)		***dF***	***F***	***p***	η_p^2
CON	*58.3 (58.3)*	***Condition***	*2,56*	*0.560*	*.575*	*.020*
MOD	*14.8 (24.1)*	***Condition*Sex***	*2,56*	*0.640*	*.531*	*.022*
MAX	*-4.80 (18.7)*					

*n = 30, CON = Control condition, MOD = Moderate condition, MAX = Maximal condition * denotes significant effects (p<.05)*

9. References

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