

Impact of Stretch Intensity on Group and Individual Responses to Pectoral Static Stretching

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Abstract

Context: Static stretching (SS) is a foundational element of many preventative and rehabilitative injury protocols that increases range of motion (ROM), likely through improved stretch tolerance and reduced muscle stiffness. Yet, the specific role of intensity, a modifiable dose parameter, remains unclear as different intensities appear to improve ROM similarly, but their effects on stiffness are inconsistent. Moreover, studies examining differing SS intensities have exclusively been conducted on lower limb musculature, leaving gaps in our understanding of intensity-specific effects in the upper limb. In the shoulder, shortened pectoral muscles contribute to forward shoulder posture (FSP), a factor implicated in the development of shoulder pathology, for which SS is commonly prescribed. However, little is known about how varying SS intensities influence pectoral muscle stiffness, length, ROM, or FSP, both at a group and individual level, as existing work rarely considers individual variability in responsiveness.

Objective: To address these gaps, this dissertation examined the group and individual responses to acute pectoral static stretching at different intensities on pectoralis major and minor stiffness, horizontal abduction ROM (haROM), pectoral muscle length (PML), and FSP in asymptomatic individuals. **Methods:** In Study One, pectoral stiffness (measured via shear wave elastography), PML, FSP, and haROM were measured before and after a 3x30 second bout of a therapist-administered passive pectoral SS at low and moderate intensities (110% and 120% of a baseline ROM). Study Two examined the impact of self-administered moderate and maximal pectoral SS intensities on the same outcomes compared to a control condition (CON). Using this same protocol, Study Three evaluated the individual response variability to moderate and maximal pectoral SS, exploring predictors of response through baseline characteristics and rate-dependent changes. **Results:** Therapist-administered low and moderate pectoral SS decreased FSP and pectoralis minor stiffness. Patient-administered moderate and maximal intensity pectoral SS similarly increased haROM compared to CON. There were transient changes in both PML and FSP. PML increased over time with considerable differences between moderate and maximal SS intensities compared to CON. FSP decreased across time, but differences between interventions were negligible. Despite significant group-level improvements in haROM and PML, a substantial portion of individuals were non-responders, with more participants responding to maximal intensity. Response patterns varied across outcomes, though every participant responded in at least one measure to one intensity. Baseline characteristics did not differ between responders and non-responders, and a moderate rate-dependent effect was observed for haROM at moderate intensity only. **Conclusion:** These findings demonstrate that SS intensity influences the likelihood of eliciting meaningful adaptations, with higher intensity producing higher response rates despite similar group-level gains. Importantly, responsiveness varied across ROM, stiffness, muscle length, and posture, indicating that ROM improvements may arise from diverse, individual-specific mechanisms rather than uniform pathways. The absence of distinct baseline characteristics further underscores that stretching benefits cannot be assumed from initial flexibility or muscle length alone. Collectively, this work challenges the assumption of uniform SS efficacy, highlights the need for individualized monitoring of outcomes, and suggests that tailoring SS intensity to individual responsiveness may optimize both preventative and rehabilitative practice.

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Dedication

To all the special people who have dedicated their energy to me in one way or another to help me reach this point. I would not be here without my village. And to my Grandpa, Gus, who instilled in me the value of education. *Knowledge is power.* I hope I've made you proud.

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List of Terms

FSP- forward shoulder posture
haROM- horizontal abduction range of motion
MTU- muscle-tendon unit
PMI- pectoralis minor index
PML- pectoralis minor length
POD- point of discomfort
ROM- range of motion
SS- static stretch/stretching
SWE- shear wave elastography
SWV- shear wave velocity

Chapter 1: Introduction

Static stretching (SS) is a therapeutic tool widely implemented in clinical settings to prevent or rehabilitate injury, or in athletic and fitness environments to aide athletic performance and recovery^{1,2}. SS requires a muscle or group of muscles to be held in a lengthened position for a prescribed period of time. An abundance of literature has examined the impact of SS, consistently demonstrating its ability to enhance range of motion (ROM)³⁻⁵, yet the precise mechanisms underlying the increase in ROM are not entirely understood⁴. Two main theories have been presented in the literature: the mechanical and sensory theories⁶.

The mechanical theory purports that changes in various physiological properties of muscle contributes to increasing muscle length and thus ROM, including viscoelastic deformation, neuromuscular relaxation, and decreased stiffness⁶. To-date most literature defines the passive mechanical changes that occur in response to stretching through passive torque measurements, calculations of passive muscle-tendon unit (MTU) stiffness, and more recently, muscle stiffness via shear wave elastography (SWE). Changes in the viscoelastic behaviour of a muscle or group of muscles is often characterized by the passive torque exerted by the muscle during a stretch⁷⁻¹⁰. Passive torque is then used to plot a length-tension curve, and passive MTU stiffness is calculated as the slope of a given portion of that curve^{11,12}. Specific stiffness of a muscle is more recently quantified via SWE, where the velocity of shear waves propagating through the tissue are measured and indicative of tension or stiffness¹³⁻¹⁵.

Alternatively, the sensory theory suggests that increases in ROM in response to stretching are the result of altered sensation leading to greater tolerance of stretch^{6,16,17}, especially in the absence of mechanical alterations¹⁸⁻²⁰. Given that increases in ROM occur both in the

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presence^{21–26} and absence^{18–20} of mechanical alterations, both theories may provide insight into the mechanisms that contribute to increased ROM from SS⁴.

Three components contribute an acute SS dose: duration (i.e., time under stretch), number of repetitions, and intensity the stretch is maintained at²⁷. While the American College of Sports medicine recommends that SS consists of two to four sets of 10-30 second (s) holds²⁸, various combinations of duration, repetitions, and intensities have been examined. Most often manipulated are duration and repetitions as they are objective elements of dose prescription²⁹, whereas intensity is more difficult to control between participants due to its subjectivity. Traditionally, SS intensity is prescribed based on the sensation of a stretch that may be mildly uncomfortable but does not cause pain; however, SS literature often does not report intensity²⁹ or inconsistently uses various methods to define intensity (e.g., point of discomfort, maximum ROM before pain, etc), making it difficult to compare between studies^{5,27}.

Evidence-based recommendations suggests there is no one combination of duration and repetitions of stretching that uniformly enhances ROM⁵. Similarly, current evidence suggests that high- or low- intensity SS enhances ROM equally^{3,5}. However, the existing body of literature that investigates the impact of SS intensity is limited and has exclusively been conducted in lower body musculature. To-date, between comparisons of high- and low-intensity SS, higher-intensity increased ROM^{12,25,30–34} and passive torque^{12,30,31}, and decreased stiffness^{12,34}, and lower-intensity increased passive torque³⁵ and stiffness^{33,36}, while both intensities equally increased ROM^{35–41} and passive torque^{32,40}, and decreased stiffness³². Considering that stretching at a higher intensity causes greater discomfort²⁹, increases stress on the MTU⁴² and triggers an inflammatory response^{43,44}, stretching at a reduced intensity may be more advantageous if the outcome effects are negligibly different than those of high-intensity SS.

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These gaps highlight the need to consider how SS intensity may influence regions beyond the lower limb. Particularly, SS is commonly applied in the shoulder, yet its impact remains largely unexplored. The shoulder complex consists of four joints: three synovial (sternoclavicular, acromioclavicular, and glenohumeral) and one “false” (scapulothoracic) that together connect the upper extremity to the trunk via bony articulations or muscular attachments, respectively⁴⁵. Together, these joints allow for more freedom of movement than any other joint, which is imperative to performing a variety of activities and functions required for daily living. This flexibility means that the shoulder relies primarily on dynamic stabilization of the surrounding musculature, which unfortunately sacrifices its stability, leaving it highly susceptible to pathology^{46,47}. The delicate balance and relative contributions of the various muscle groups surrounding the shoulder influences its position and movement, and aberrant shoulder posture and motion increases the risk of developing shoulder pathology⁴⁸⁻⁵³.

One such posture is forward shoulder posture (FSP), which is characterized by shortened pectorals causing anterior deviation and abduction of the scapula^{54,55}. The pectoral muscle group consists of pectoralis major, a large fan-shaped muscle attaching from the clavicle and sternum to the proximal humerus, and pectoralis minor, a smaller triangular-shaped muscle attaching from the sternocostal cartilages of ribs 3-5 to the coracoid process on the scapula. Given these attachments, when adaptively shortened, the pectorals protract the scapula and humerus anteriorly, contributing to a FSP⁵⁶. Specifically, FSP is strongly negatively correlated to pectoralis minor resting length⁵⁴. This forward shoulder position alters the movement or kinematics of the scapula^{55,56} and may lead to the development of shoulder pathologies⁴⁸⁻⁵³. As such, strategies aimed at lengthening the pectorals to optimize FSP are vital to rehabilitating or preventing shoulder pathology.

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Pectoral SS is a commonly implemented strategy to elongate the pectorals, reduce FSP, and prevent or rehabilitate shoulder injuries. Interestingly, little research has evaluated the impact of a single acute pectoral SS, as literature to-date has employed combination therapies (e.g., SS and exercise or massage)^{57,58} or multi-week protocols^{48,59–61}. To-date, one research group has examined the effectiveness of various pectoral stretch positions on pectoralis minor elongation⁵⁰, and the impact pectoral SS has on pectoralis minor stiffness and scapular kinematics^{62,63}. Umehara and colleagues determined that horizontal abduction at 90° and 150° of arm elevation effectively lengthens pectoralis minor⁵⁰, and as little as one set of 30s of pectoral SS at maximum tolerable range without pain effectively decreased pectoralis minor stiffness⁶³. Pectoral SS for a cumulative 5-minutes increased scapular external rotation and posterior tilt⁶². Despite these findings, Umehara et al. did not measure changes in any other variable such as ROM or pectoralis major stiffness. As such, the impact that acute pectoral SS has on FSP, pectoralis minor length, range of motion, and pectoralis major stiffness remains unknown, and if stretching the pectorals at different intensities would impact these outcomes differently. Thus, one of the primary objectives of this work was to examine the group effects of pectoral SS at differing intensities.

Effective clinical practice is guided by research evidence and tailored to the individual; however, traditional analyses typically focus on group-level effects. Although these analyses indicate that SS generally increases ROM, evidence shows that a considerable proportion of individuals do not respond to SS⁶⁴. Advancing toward individualized exercise prescription therefore requires examining how SS influences response rates, whether intensity affects the likelihood of response, and what factors influence responsiveness. Accordingly, a secondary

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purpose of this work was to investigate inter-individual variability in response to pectoral SS at differing intensities.

This dissertation is organized into several chapters that progress from a review of the literature, through three manuscripts examining the effects of pectoral SS intensity, to a final discussion and conclusion. Chapter 2 reviews the relevant literature. Chapter 3 presents the first manuscript, which examines the effects of low and moderate intensity therapist-administered pectoral static stretch on changes in pectoral stiffness, pectoral muscle length, horizontal abduction range of motion, and forward shoulder posture. Chapter 4 presents the second manuscript, which investigates the effects of a moderate and maximal intensity self-administered pectoral static stretch on the same outcome variables as Chapter 3, relative to a control condition. This chapter also explores the relationship between changes in participant-reported stretch intensities during stretching and pre-post changes in the same variables, as well as the magnitude of elongation of pectoralis major and minor during moderate and maximal stretch intensities. Chapter 5 presents the third manuscript, which examines individual responses to moderate and maximal pectoral stretching, including baseline differences between responders and non-responders, and the influence of baseline values on the magnitude and direction of change (i.e., rate-dependent effects). Chapter 6 discusses the findings of the dissertation, including limitations, and Chapter 7 provides a general conclusion.

Chapter 2: Review of the Literature

This doctoral dissertation examined the group- and individual-level responses to different intensities of acute pectoral static stretching (SS). As such, it is necessary to review the relevant literature pertaining to the effects of SS, including the mechanisms that lead to increased range of motion, and current evidence for implementing various intensities of SS. The following chapter will discuss skeletal muscle structure and the properties it contains that contribute to altered length, what SS is, the documented effects both broadly and in terms of varying SS intensities, and the current literature for pectoral SS. The chapters following the literature review contain manuscripts with methods aimed at delineating group and individual responses to different intensities of pectoral SS.

Muscle Tone

Resting muscle tone is a muscle's state of mechanical tension in the absence of contractile activity^{65,66}. Both active and passive factors contribute to a muscle's tone. The active factor is considered neurological stimuli, and its contribution to resting muscle tone is frequently quantified by electromyography (EMG) while relaxed (i.e., baseline EMG)⁶⁷. While the physiology responsible for the neurological input to a muscle that dictates its tone is complex and includes spinal and supraspinal (i.e., central nervous system) mechanisms⁶⁷, review of these mechanisms is beyond the scope of this work (for more information, see Ganguly et al., 2021⁶⁷). On the other hand, passive factors involve the viscoelastic properties of skeletal muscle, including stiffness (i.e., the passive resistance to deformation), which contribute to resting muscle tone⁶⁸. Stiffness is the relationship between stress (i.e., force) applied and strain (i.e., deformation), and is thus the passive resistance to deformation¹⁴. Viscoelasticity is the ability of a structure to resume its original length upon removal of a tensile force⁶; in muscles, it is the

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ability for the muscle to return to resting length upon termination of a stretch. It is these viscoelastic properties that stabilize posture when high levels of mechanical stability are not required (e.g., at rest or during low-energy activity such as computer work)⁶⁵.

A muscle that is hypertonic (i.e., has an increased resting muscle tone) is shorter in length and emits increased passive force on its bony attachments or surrounding joints, which may lead to decreased ROM, altered postures or movement patterns⁶⁹. Several muscles in the human body are predisposed to increased muscle tone due to their role in maintaining an upright posture, including the pectorals⁶⁹. When shortened, the pectorals increase forward shoulder posture (FSP)^{54,55}, which alters the position and kinematics of the scapula⁵⁵ that contribute to shoulder pathology^{48–53}. Thus, decreasing tonicity of the pectorals is crucial for preventing or rehabilitating shoulder injuries.

Skeletal Muscle Structure

Muscles contain a highly organized arrangement of muscle cells and connective tissue called the endo-, peri-, and epimysium, also known as fascia or myofascia⁷⁰. Endomysium surrounds individual muscle fibres that, when bundled together, are called fascicles, and are surrounded by perimysium. Several fascicles are encased in an outer connective tissue epimysium. The three levels of myofascia fuse together at either end of the muscle to form tendons that connect to bones to facilitate movement. Individual muscle fibres are composed of bundles of striated myofibrils that are the most basic functional contractile units of muscle contraction. Along each myofibril are serial sarcomeres that contain the myofilaments actin and myosin, which bind together to form a cross-bridge when stimulated by a motor nerve^{71,72}. Cyclical cross-bridge formation produces a mechanical shortening of the muscle, and these are considered active mechanisms that contribute to a muscle's tone.

Muscle Viscoelasticity

The ability for skeletal muscle to deform under force and return to its original shape once the force is removed is due to its viscoelastic structure with both elastic and viscous properties⁷³. Elastic properties of muscle pertain to its ability to return to its original length after deformation or stretch, and changes in length are directly proportional to the force applied. Alternatively, viscous properties reflect the energy dissipation or resistance to deformation over time when lengthened⁷³. Creep, stress relaxation, and hysteresis are all viscous properties of skeletal muscle^{73,74} (**Figure 1**).

Creep is the deformation that occurs under a fixed stress or load (**Figure 1A**). In SS research, creep is measured during a stretch using a dynamometer by measuring the amount of torque applied to the dynamometer by the limb being stretched (i.e., resistive torque). The dynamometer applies a constant torque to stretch the muscle for some period of time, and resistive torque of the stretched muscle is measured⁷⁵. For example, Ryan et al. (2010) found that applying constant torque stretch continually increased dorsiflexion ROM throughout a 30s stretch, with the majority of change happening in the first 15-20s⁷⁵.

Stress relaxation is the gradual decrease in stress or force of a muscle that is held at a constant length while stretching (**Figure 1B**)⁷⁴. In SS research, it is measured similarly to creep, except the angle applied to the stretch is maintained throughout, thus keeping the stretch at a constant angle rather than a constant torque. For example, while a constant torque stretch would continue altering the angle of the stretched limb to maintain the resistive force of the limb, constant angle would maintain the same limb angle throughout the stretch regardless of changes in resistive force. This concept was first demonstrated in human skeletal muscle by McHugh et

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al. (1992), where the application of a hamstring SS at maximal ROM for 45s elicited a decrease in the required force to keep the leg at the stretch angle⁷⁴.

Hysteresis is the dissipation of energy between loading and unloading a muscle and is measured via ultrasonography⁷⁶. From a relaxed state, participants will slowly increase and decrease torque development, and the displacement of the myotendinous junction is measured via ultrasound⁷⁶. Stress (i.e., force produced) and strain (i.e., displacement) are graphed (**Figure 1C**) during loading (i.e., increasing force) and unloading (i.e., decreasing force), and the difference between the two phases is the energy loss during mechanical deformation.

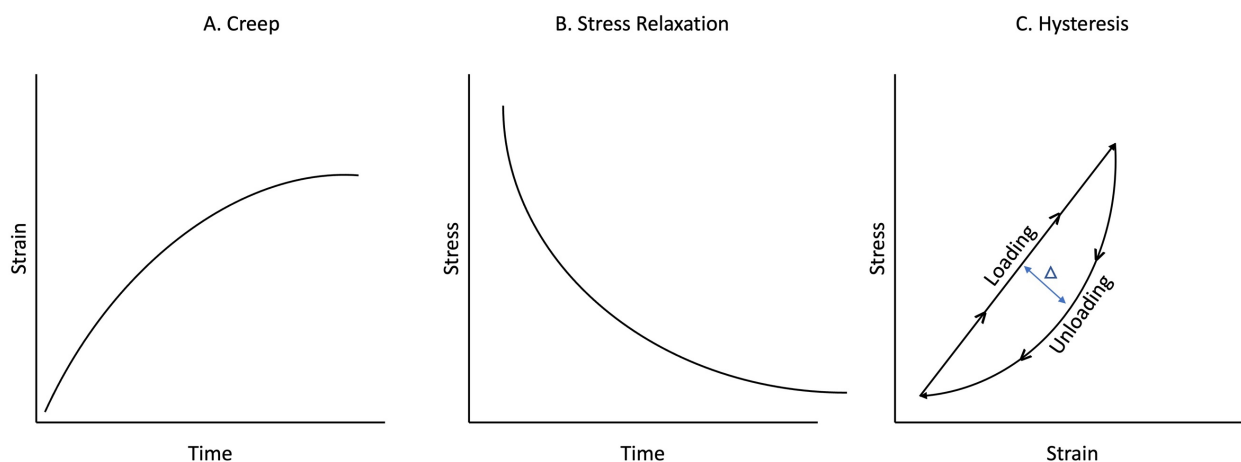


Figure 1: Graphical Representations of Muscle Viscoelastic Properties Creep, Stress Relaxation, and Hysteresis

The relationship between a muscle's length and the associated tension (also called the stress-strain relationship) can either be active, where a muscle's capacity for force production during contraction is dependent on its length, or passive⁷⁷. The passive length-tension relationship exists between the length and passive mechanical/viscoelastic properties of the muscle in the absence of muscle contraction⁷⁷. As a muscle is lengthened from a resting position, the shortest length at which passive tension is sustained and fascicles begin to lengthen is called

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slack length⁷⁷. The slack length for various muscles differs. For example, the gastrocnemius slack length is ~40% of its physiological ROM while vastus lateralis slack length is around 10-25% of its ROM⁷⁷. Alternatively, slack length may differ within muscles with varying fiber orientations. For example, the pectoralis major has several regions based on fiber orientation, and the mean slack length during shoulder abduction was around 43° for the sternocostal region and 62° for the abdominal region⁷⁸.

Passive Tension Measurement Techniques

As a stretch is applied and muscle length increases, passive tension nonlinearly increases¹⁵. The degree of passive tension at various muscle lengths can be determined by a few different methods, such as B-mode ultrasound combined with dynamometry, other ultrasonography elastography techniques such as strain or shear wave, and myotonometry¹⁵. Ultrasonography elastography techniques create stress to the soft tissue (i.e., physical compression or shear wave), which allows the resulting distortion and mechanical properties to be mapped⁷⁹.

The use of B-mode ultrasonography coupled with a dynamometer quantifies joint torques produced at various angles or fascicle lengths, thus allowing for the examination of length-tension curves of specific muscles or muscle groups¹⁵. Strain elastography requires application of manual compression to the tissue via the transducer/probe and stiffness is determined from the resulting deformation⁸⁰. However, significant challenges exist with strain elastography, such as poor repeatability and significant variation between operators and application techniques^{14,15}. Alternatively, shear wave elastography measures the propagation speed of shear waves through soft tissues that are generated by a probe, and is less operator-dependent than strain elastography¹⁴. Myotonometry, used most frequently in clinical environments, quantifies

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stiffness and other tissue properties by applying a short mechanical oscillation to the tissue and measures the resulting motion of the tissue¹⁵. However, myotonometry use is limited to superficial structures only, and increased levels of subcutaneous fat may impede its accuracy⁸¹.

Static Stretching

Static stretching (SS) is the most common type of stretching⁸² that is frequently used in athletic or fitness recovery environments and clinical or rehabilitative settings¹. SS deliberately elongates a muscle or group of muscles, thereby increasing the range of motion (ROM) of a joint⁸³. Typically, SS is performed as a controlled, continuous movement that lengthens the target muscle(s) past its resting length until a stretch is felt, and can be conducted passively or actively⁸². Passive SS uses external forces, where a joint is moved to the end range of motion by gravity, a partner, or stretching aids^{4,83}, where active SS requires the patient to apply the stretch⁸². This position is then held for some prescribed period of time^{4,84} and repeated as necessary⁸². This duration of stretch, the number of sets completed, and/or intensity of the stretch are all modifiable factors that are used to prescribe the dose of the SS.

While SS has long been a topic of debate in research and in clinical practice, it is frequently used as part of a warmup/cool down routine, in musculoskeletal rehabilitation, to improve physical performance or general physical function². The positive impact SS has on increasing ROM is well documented in the literature³⁻⁵. However, acute performance impairments following SS⁴², alongside queries of lasting effects^{85,86}, have left researchers and practitioners questioning the practical impact and effectiveness of SS. Increased ROM sought from SS is purported to decrease injury risk and incidence⁸⁷, as limitations in flexibility and ROM may predispose individuals to injury⁸⁸. Indeed, current evidence demonstrates that

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incorporating SS in pre-exercise warmup does have a moderate effect in mitigating injuries² when performed for under 60s.

Static Stretching Effects

Many acute and chronic effects of SS have been noted in the literature. In the remainder of the document, acute effects will refer to effects from a single bout or session of stretching, and chronic effects will refer to those observed following repeated SS sessions, usually taking place over the span of a few weeks. This review will focus on acute SS effects as the current work examined the acute effects of SS.

While the American College of Sports Medicine⁸⁹ considers SS an essential component of the cool-down phase to assist with recovery by increasing ROM, reducing stiffness and delayed onset muscle soreness (DOMS)⁹⁰, a recent systematic review found that stretching was no more effective at returning strength levels or reducing DOMS than passive rest or alternative recovery modalities⁹¹. Interestingly, the authors note that despite how commonly stretching is prescribed post-exercise to enhance recovery, the diversity of protocols within the limited sample of articles led to insufficient data to systematically review and meta-analyze for the effects on ROM⁹¹. Interestingly, previous systematic reviews indicate that post-exercise stretching does not affect DOMS^{92,93}.

One of the most documented acute effects of stretching is increasing ROM^{3,5,86}, regardless of stretch type (static, proprioceptive neuromuscular facilitation, dynamic, ballistic), dosing parameters (e.g., duration and intensity), activity level/trained state, age, or sex⁵. While SS for as little as 5s increases ROM, longer duration SS leads to more significant increases⁵. However, a recent systematic review revealed that no single SS duration provides a significant advantage to increasing ROM⁵. Interestingly, it is important to note that a majority of SS

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intervention research has been conducted on lower limb musculature (e.g., hamstrings, triceps surae, quadriceps, hip adductors)⁵, with comparably fewer investigations exploring the effect of SS on upper body muscles. Given that different muscles respond differently to SS⁹⁴, it is necessary to delineate the impact of SS on various muscle groups. A joint's passive ROM is dependent on several structures, such as muscle fascicles, tendons, aponeurosis, joint capsules, and ligaments⁹⁵. Additionally, a muscle's resistance to stretch is also caused by tonic reflex activity, or neural input^{96,97}. As such, two primary theories exist to account for changes in ROM from SS: mechanical and sensory theories⁶.

Mechanical Theory

According to Weppeler and Magnusson (2010)⁶, muscle length is multidimensional and changes in any one of the dimensions may lead to alterations in a given muscle's length. Muscle extensibility, tension, cross-sectional area, and time all have associated biomechanical properties that contribute to muscle length (**Figure 2**). In the SS literature, the viscoelastic adaptations that contribute to increased ROM from a given stretch are often quantified through alterations in passive torque, neuromuscular characteristics, muscle-tendon unit (MTU) stiffness, or shear wave elastography (SWE). Passive torque and neuromuscular characteristics are often used as surrogates for changes in muscle tone. Specifically, passive torque quantifies the passive tension (i.e., viscoelastic properties) of a muscle or group of muscles generated at a given angle or ROM¹⁵. Thus, changes in passive torque are indicative of changes in the viscoelastic properties of the muscle. Alternatively, neuromuscular characteristics such as the quantification of muscle excitation using EMG are used to define the contractile-related mechanisms that contribute to changes in muscle tone^{42,65}. Passive MTU stiffness and SWE measure changes in muscle stiffness following stretching. Given that stiffness is the relationship between stress and strain or

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length and tension¹⁴, passive MTU stiffness uses a plotted length-tension curve to calculate the slope of the relationship to reveal changes in viscoelastic stiffness¹¹. Whereas SWE applies a stress (i.e., shear waves) and measures the displacement or velocity of the shear waves (i.e., strain)⁷⁹, where faster velocity indicates higher stiffness⁹⁸.

Neuromuscular responses to SS have been widely studied in the SS literature. Tonic reflex activity is commonly measured via several reflexes: the Hoffman (H), tendon (T), or exteroceptive (E) reflexes. The H- and E-reflexes are differentiated by the electrical stimuli applied: the H-reflex to the proximal nerve (i.e., Ia afferent nerve) while the E-reflex is obtained by stimuli of the sensory receptors in the skin⁹⁹. The T-reflex is stimulated via percussion of the tendon, where Ia afferent neurons are stimulated in the muscle spindle⁹⁹. The H- and T reflexes examine the spinal reflex arc that includes both presynaptic (i.e., Ia afferent neuron) and postsynaptic (i.e., efferent neuron or motor excitability) pathways, while the E reflex examines only postsynaptic inhibitory mechanisms. The T reflex is additionally moderated by the sensitivity and contribution of the muscle spindles to any neurological changes in a stretch⁹⁵.

During SS, spinal reflex excitability is decreased, identified by a decrease in the H reflex amplitude^{95,100}. This presynaptic inhibition of Ia afferents weakens the release of neurotransmitters to the motor neurons, thus reducing the reflex arc through decreased excitability of the motor neurons⁹⁹. The decrease in T or E reflexes largely depends on the amplitude (i.e., intensity) of the stretch; small-amplitude stretches cause little to no change in T or E reflexes, while large-amplitude cause a decrease, demonstrating postsynaptic inhibitory mechanisms⁹⁵. As such, purposeful, slow stretching inhibits the stretch reflex, allowing for increased range of motion of the muscle. However, these reflexes return to baseline upon termination of the stretch⁹⁵.

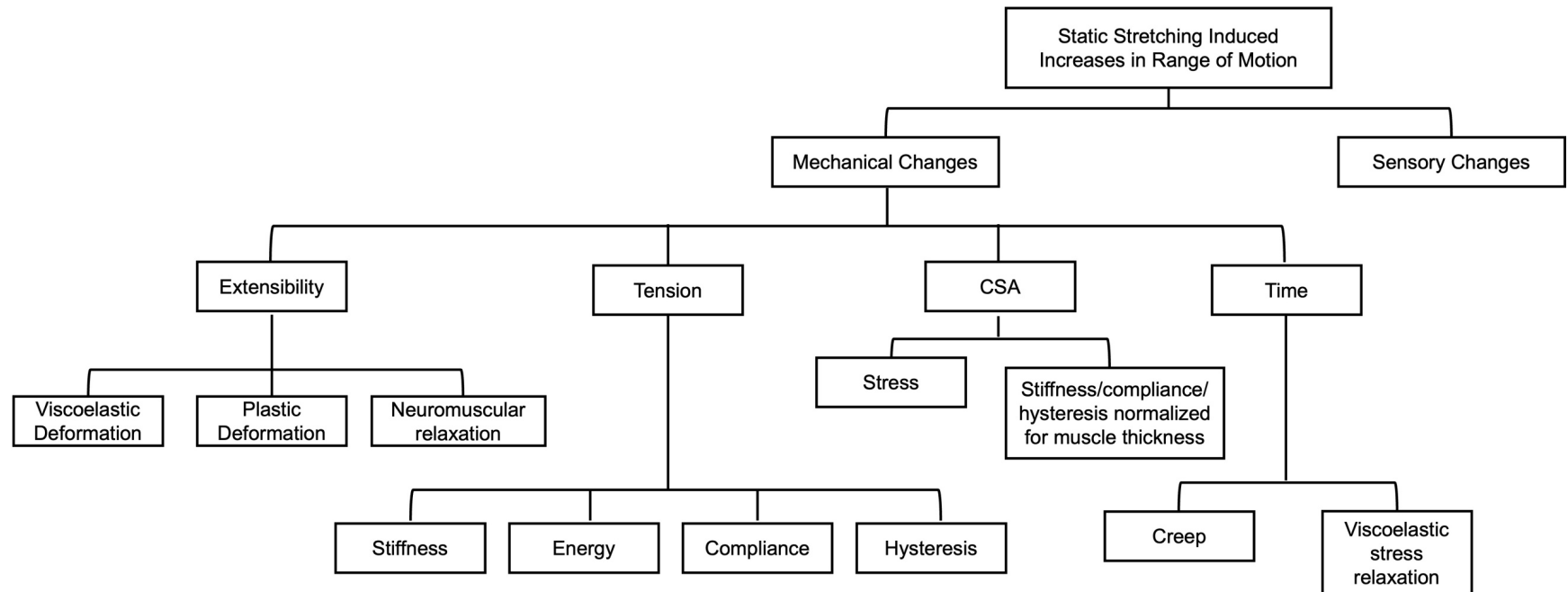


Figure 2. Biomechanical mechanisms leading to changes in range of motion of a muscle.

Adapted from Weppeler & Magnusson (2010)⁶

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SS is proposed to alter several neural mechanisms, such as muscle excitation amplitude and corticospinal excitability/inhibition; however, evidence is contentious⁴². While a recent meta-analysis suggests there are no significant acute neural changes resulting from SS⁸⁶, another review has found SS likely alters the amplitude and duration of the muscle action potential wave (M-wave) and E reflexes, though evidence is inconsistent⁴². The sensory receptors in the skin initiate E reflex inhibition, which contribute to sympathetic inhibition and promote muscle relaxation, though evidence suggests E-reflex inhibition may only occur during large-amplitude stretches and cease upon termination of the stretch⁴². Given that the current work is not evaluating neuromuscular changes, further review is outside the scope of this literature review.

Often called peak torque or peak passive torque, passive torque and ROM are measured simultaneously where a dynamometer moves participants through a ROM. Depending on the protocol, ROM is typically stopped when the participant first reports a stretch sensation¹¹, point of discomfort (POD)^{32,33,36–38,40,41}, or maximal stretch prior to onset of pain^{12,30,31}. At this point, the passive torque produced by the stretched muscle is measured by the dynamometer. While this measurement is used in-part to calculate MTU stiffness (discussed next), studies have often used it to evaluate the impact of stretching on passive muscle properties, and to quantify stretch tolerance^{8–10}.

MTU stiffness, sometimes referred to as passive stiffness, is the relationship between passive torque and passive ROM and represents the viscoelasticity of the MTU¹¹. Torque and ROM are plotted on a graph which creates a curve (N.m/°), and muscle-tendon stiffness is considered the slope of part of the curve^{11,12}. Previous methods calculate the final 1/3 of the curve^{11,39}, from 50% of the maximal ROM angle achieved^{12,17,40}, between certain angles (e.g., between 15 and 25° of dorsiflexion)³⁴.

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While SS effectively decreases MTU stiffness^{4,101,102} and passive torque^{103,104}, there are a couple limitations in using these methods to capture changes in passive muscle properties. First, the use of a dynamometer limits the muscle/muscle groups/range of motion that can be tested given its setup. Secondly, the contribution of various tissue including multiple muscles, tendons, and connective tissue (e.g., fascia) is difficult to delineate¹⁰⁵. As such, changes in torque may not be muscle specific and may be a result of changes in one or more muscles, tendon or connective tissue stiffness, or a combination of the three.

SWE is a relatively new technique that can accurately quantify tension of specific soft-tissues¹⁰⁶. An ultrasound coupled with a transducer is placed on the skin overlaying the soft tissue being measured. The SWE produces an adjustable region of interest (ROI) that can be placed over the target tissue that generates a colour-coded map of stiffness. The transducer generates a pulse into the targeted tissue, creating a ripple effect of shear waves through the tissue, where propagation speed of is subsequently measured to determine velocity of shear waves (SWV; m/s)¹³.

Stretching research has begun to incorporate SWE to quantify stiffness of specific muscles. Compared to MTU stiffness described above, SWE allows for greater specificity, as one structure can be measured at one time. Moreover, SWE does not pose the structural barriers that exist with dynamometry; as such, the mechanical response to SS of individual muscles or tissues anywhere in the body can be defined. One study has compared changes in passive torque and SWE in response to an eccentric training protocol, finding that SWV increased after exercise while passive torque did not, with no correlations between the two¹⁰⁷. The authors concluded that these two methods of stiffness quantification have limited agreement, may be angle specific, and that SWV may be more sensitive to changes in stiffness compared to passive torque/MTU

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stiffness. However, there are limitations to the use of SWE. First, the anisotropic nature of muscle architecture allows shear waves to travel faster when parallel to muscle fibres/fascicles as opposed to perpendicular to them¹⁴. Thus, SWV is sensitive to the orientation of the probe in reference to the muscle fibres, and slightly different probe locations may impact SWV. Secondly, the anatomy of specific muscles may impact the effectiveness of SWE, as “multi-orientation” fibers such as multipennate or fusiform fiber orientations may not allow for true parallel orientation of the fibers with the probe¹⁴. Third, shear waves attenuate greater in deeper structures, and thus superficial tissues such as other muscles or greater amounts of adipose tissue may impact its usability in deeper soft tissue¹⁴. Lastly, as elastograms can only be measured in one specific area of a muscle at one time, changes in stiffness are specific to that location and cannot infer to stiffness changes along the entire length and/or depth of the muscle.

To date, SWE has determined that one-minute of SS reduces iliacus stiffness¹⁰⁸, 20-seconds of SS reduces infraspinatus stiffness¹⁰⁹, 5x1-minute and 5x30-seconds of stretching (total 5-minutes and 2-minutes, respectively) decreases stiffness in the hamstrings^{110,111}, and 4x30-seconds (total 2-minutes) and 5x1-minute (total 5-minutes) of SS decreases stiffness in the gastrocnemius^{112,113}. Collectively, recent systematic reviews state acute SS effectively decreases passive stiffness of the MTU or individual muscles in younger and older populations^{86,102,114}.

Sensory Theory

It is theorized that changes in ROM from SS are due at least in part to increasing the individual's tolerance to stretch sensation^{4,6,7}, or the ability to tolerate increased stress. Traditionally, increased passive torque at maximal ROM angles would indicate increased stretch tolerance⁷, but it may also be indicated from increased ROM in the absence of viscoelastic changes. A few decades ago, researchers discovered that increased ROM induced by SS may not

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be due to increased muscle length^{18–20}. The plotting of ROM and the associated passive torque produces a length-tension curve, which is used to calculate MTU stiffness. Theoretically, if SS increased muscle length as predicted, the entire length-tension curve would shift to the right (**Figure 3**) indicating longer muscle lengths for a given passive torque or tension⁶. However, researchers found that instead of shifting the curve to the right, ROM and passive torque increased only at end-range^{18–20}. This indicated that the length of the muscle did not change, and the achievement of greater ROM was attributed to alterations in participant perception of sensation at end ROM.

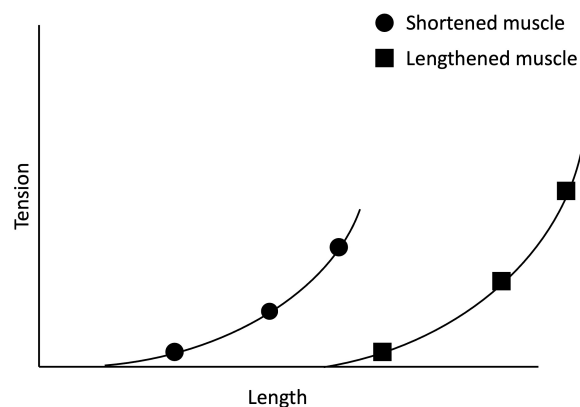


Figure 3. Shifting of length-tension curve with increased muscle length.
Adapted from Weppeler & Magnusson⁶

Indeed, the right shift of the torque-angle curve following SS has been contentious in the literature. While some literature has found SS does not shift the torque-angle curve^{18–20}, others have found that a shift does occur after SS^{21–26} demonstrating mechanical changes occurred to induce muscle lengthening. Moreover, the literature has demonstrated that these changes in neural and mechanical factors result in deficits in strength. For example, one of the most cited studies demonstrated a 28% decrease in strength following 30-minutes of passive stretching, and 9% of the decrements persisted at least 1-hour post-stretching¹¹⁵. Similar findings have been demonstrated in 10-²⁶ and 20-minute²² stretching protocols. Muscle excitation concomitantly

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decreased following 10- and 30-minutes of stretching, suggesting neural alterations contribute to decrements in strength¹¹⁵. In research implementing stretch protocols that are shorter and more representative of common stretching prescription, strength deficits are also reported, but are reversed after 10-minutes¹¹⁶. These deficits occur concurrently with increases in ROM, with ROM also returning to baseline within 10-minutes¹¹⁶. Interestingly, these changes in strength and ROM occurred without any changes in EMG amplitude¹¹⁶. Additionally, a systematic review demonstrated an acute SS increases ROM and maximal passive resisted torque (i.e., stretch tolerance) and decreases passive resisted torque, strength, and stiffness of the motor unit, muscle, and tendon, but does not change neural responses⁸⁶. The discrepancy in the literature demonstrates the complex, multifactorial contributions to changes in ROM following SS, and thus, SS research should consider both mechanical and sensory mechanisms that contribute to increasing ROM.

Static Stretch Dose

Single session SS prescription includes several dose parameters that, when altered, may alter the benefits or effects derived. These include duration (i.e., time under stretch), repetitions, and intensity of stretch. Duration and repetitions are most frequently manipulated in the literature as they are modifiable and quantitative²⁹. While as little as 5 seconds of SS increases in ROM⁵, the American College of Sports Medicine (ACSM) recommends SS be repeated for two to four sets of 10-30s held at the point of tightness or slight discomfort reaching a cumulative 60s per muscle group²⁸. Interestingly, SS intensity is often neglected, and a 2015 review found that only 79 of the 152 (51%) articles referenced SS intensity²⁹.

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Intensity

Intensity is a dose factor that may contribute to altered response to SS, although it is infrequently manipulated due to the inherent subjectivity of patient's perception of stretch intensity. Although this proves difficult to regulate between participants, intensity may influence tissue response. For example, SS intensity that is too low may not initiate a tissue response that will increase ROM, but may potentially decrease the risk of sprains and strains¹¹⁷. Moreover, higher intensity stretching may cause discomfort²⁹, places considerable stress on the myotendinous unit⁴², and induces an acute inflammatory response^{43,44}.

Objectively, SS intensity is the amount of force applied during stretching²⁹, while subjectively, it's the perceived intensity, or tolerance of stretch, by the individual. Though force or torque can be measured during stretching, it is not used to prescribe the intensity. Rather, intensity is moderated through various descriptive terms (e.g., discomfort or pain)²⁹ and methods such as point of discomfort (POD)^{32,33,36–38,40,41}, point of pain (POP)³⁷, maximum tolerable stretch/torque (MTS) immediately before pain^{12,30,31}, and numerical rating scales (NRS)³⁹. Variation in SS intensity is then prescribed as some percentage of these (e.g., 80% POD). However, the discrepancies in the way researchers prescribe intensity presents great challenges in comparing outcomes.

Several studies have examined the acute impact of different SS intensities in the hamstrings^{12,25,30–32,35,37–40} quadriceps^{33,36}, and calves^{32,34}. Collectively, all studies observed increases in ROM, though the effect of SS intensity is contentious as nearly half found higher intensity increased ROM greater than lower intensity^{12,25,30–34} and half found no difference between intensities^{35–41}. Additionally, high intensity SS increased passive torque^{12,30,31} and decreased stiffness^{12,34} compared to low intensity SS, while both high and low intensity increased

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passive torque^{32,40}, decreased stiffness³², and lower intensities increased passive torque³⁵ and stiffness^{33,36} compared to higher intensities (**Table 1**).

In the hamstrings, a high intensity SS held at maximum tolerable stretch (MTS) and incrementally increased every 90s until they were unable to increase anymore (mean duration of 244s) increased ROM and passive torque compared to a low-intensity (50% of MTS x 900s) which did not observe changes in either outcome³¹. A second study by the same research group compared a high intensity (100% MTS, 5x90s), moderate intensity (75% MTS, 5x135s), and low intensity hamstring SS (50% MTS, 5x180s), finding that the high-intensity increased ROM and passive torque, while low and moderate intensity observed no changes³⁰. Fukaya et al., (2020) compared a high (120% POD x100s) to a low intensity (50% POD x 240s) calf SS, finding that both SS intensities increased dorsiflexion ROM and passive torque and decreased stiffness, though greater changes in ROM and stiffness was observed with the high intensity SS³². Marchetti et al., (2022) found equal increases in hamstring ROM following a high (85% maximum imaginable POD 3x40s) and low intensity SS (50% maximum imaginable POD 6x40s)³⁸. However, it is important to note that the above mentioned studies^{30–32,38} alter both intensity and SS duration, making it challenging to delineate the effects of SS intensity alone.

Table 1. Summary of Studies Comparing Different Static Stretch Intensities.

Authors	Muscle/Muscle Group	SS Protocol	Results
Kataura et al. (2017) ¹²	Hamstrings	High intensity: 120% of maximum tolerable intensity Moderate intensity: 100% of maximum tolerable intensity Low intensity: 80% of maximum tolerable intensity x180s	High and moderate intensity SS increased ROM and passive torque, and decreased passive stiffness and isometric force Low intensity SS: increased static passive torque during SS.
Takeuchi and Nakamura (2020) ²⁵	Hamstrings	High intensity: maximum point of discomfort Moderate intensity: 120% point of discomfort Low intensity: point of discomfort x20s	High and moderate intensity SS increased ROM; high intensity resulted in largest change in ROM.
Freitas et a. (2015) ³⁰	Hamstrings	High intensity: 100% of maximum tolerable range for 5x90s Moderate intensity: 75% of maximum tolerable range for 5x135s Low intensity: 50% of maximum tolerable range for 5x180s	High intensity SS increased ROM and passive torque. Low and moderate intensity SS did not change ROM or passive torque.

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Freitas et al. (2015) ³¹	Hamstrings	High intensity: 100% maximum tolerable range for 90s repeated until couldn't increase anymore (mean = 243.5s) Low intensity: 50% of maximum tolerable range for 900s	High intensity SS increased ROM and passive torque. Low intensity SS did not alter ROM or torque.
Fukaya et al. (2020) ³²	Triceps surae	High intensity: 120% point of discomfort x 100s Low intensity: 50% point of discomfort x 240s	High and low intensity increased ROM increased passive torque, and decreased stiffness. High intensity increased ROM significantly more than low intensity.
Nakamura et al. (2021) ³³	Quadriceps	High intensity: 120% point of discomfort Moderate intensity: 100% of point of discomfort Low intensity: 80% of point of discomfort 3x60s	High and moderate intensity SS increased ROM. Moderate intensity decreased stiffness.
Oba et al. 2021 ³⁴	Triceps surae	High intensity: 100% maximum passive resisted torque Moderate intensity: 75% maximum passive resisted torque	High intensity increased ROM and decreased MTU stiffness. No differences between moderate and low intensity SS and control.

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		Low intensity: 50% maximum passive resisted torque Control: no stretching Constant torque, 5x60s	
Melo et al. (2021) ³⁵	Hamstrings	High intensity: Stretching at pain level. Moderate intensity: Stretching at a mild discomfort. Low intensity: Stretching at a comfortable level. Control: no stretching x30s	High-, moderate-, and low-intensity all increased ROM. Moderate intensity increased passive torque.
Takeuchi et al. (2021) ³⁶	Quadriceps	2 part study: Part 1: 3x20s or 3x60s at 120% of maximal ROM (high intensity) Part 2: 3x60s at 110% of maximal ROM (low intensity)	High intensity increased ROM after both durations. Low intensity increased ROM. There were no differences between either high intensity SS and the low intensity SS. Stiffness decreased only after the low intensity SS.
Munjai et al. (2017) ³⁷	Hamstrings	High intensity: point of pain Low intensity: point of discomfort 8x30s	High and low intensity SS increased ROM

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Marchetti et al. (2022) ³⁸	Hamstrings	High intensity: 85% of point of discomfort 3x40s Low intensity: 50% of point of discomfort 6x40s	Both intensities increased ROM. High intensity decreased peak isometric force.
Santos et al. (2020) ³⁹	Hamstrings	High intensity: 9-10 on numerical rating scale Low intensity: 1-2 on numerical rating scale 3x60s	High and low intensity SS increased ROM. No differences in stiffness or viscoelastic stress relaxation.
Takeuchi et al. (2021) ⁴⁰	Hamstrings	High intensity: 120% point of discomfort Low intensity: 100% point of discomfort 2x30s	High and low intensity increased ROM and passive torque. High intensity decreased stiffness.
Takeuchi et al. (2021) ⁴¹	Hamstrings	High intensity: 120% point of discomfort Low intensity: 100% point of discomfort 2x30s	High and low intensity increased ROM.
Fukaya et al. (2020) ³²	Hamstrings	Low intensity: 100% of maximum tolerable ROM without pain High intensity: 120% of maximum tolerable ROM without pain	High and low intensity SS acutely increased ROM, with greater relative changes after high intensity. Peak passive torque acutely increased, with greater increases after high intensity.

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		1x60s for 12 sessions (3 session/week over 4 weeks), with acute changes measured after each session	Passive stiffness acutely decreased following all high intensity sessions but decreased after only 50% of low intensity sessions. The decrease in passive stiffness was significantly greater in the high intensity.
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A high (120% MTS) and moderate (100% MTS) hamstring SS for 180s observed ROM and passive torque increases and decreases in stiffness and isometric muscle force compared to a low intensity SS (80% MTS x180s)¹². Muanjai et al., (2017) compared an 8x30s higher intensity (at POP) to a lower intensity (at POD) hamstring SS, finding ROM increased after both³⁷. In the calves, ROM increased and stiffness decreased following 5x60s held at constant torque (as opposed to a constant angle) of a high intensity (100% MTS) SS compared to 5x60s at submaximal intensities (50% and 75% MTS) and a control condition³⁴. Another study comparing a no stretch control to stretching at comfort level, mild discomfort, and pain level SS (using a scale called PERFLEX) for 30s in the hamstrings found all SS groups increased ROM compared to the control, and passive torque increased in the mild discomfort group only³⁵. Interestingly, quadricep SS for 3x60s at 80%, 100%, and 120% POD found increased ROM after 100% and 120%, but stiffness (SWE) decreased after 100% only³³. Three sets of 60s hamstring stretches at a low (1-2 NRS) and high (9-10 NRS) observed increased ROM after both, through greater relative changes in ROM occurred after the low intensity SS; neither intensity altered stiffness³⁹. Similarly, 60s of hamstring stretching at low (100% POD) and high (120% POD) intensities increased ROM and passive torque^{40,41}, but stiffness decreased after high intensity SS only⁴⁰. Takeuchi and Nakamura (2020) found that hamstring ROM increased after 20s of high intensity stretches (120% POD and maximum POD) compared to a lower intensity SS held at POD²⁵, though maximum POD resulted in the largest change in ROM. Finally, in a two part study comparing a high (120% POD for 3x20s and 3x60s) and low (110% POD for 3x60s) intensity quadricep SS, while ROM increased after all interventions, stiffness decreased only after the low intensity SS³⁶. Given the current results described, it remains unclear if greater intensity SS results in greater changes in ROM³ or stiffness, or if individual incidence of response is impacted

by SS intensity. Moreover, no research has explored the impact of differing SS intensities on upper limb musculature.

Incidence of Response

Although the literature consistently demonstrates that SS effectively increases ROM at the group level^{3,5}, traditional analyses of mean changes may obscure the considerable heterogeneity in individual responses to a standardized SS intervention¹¹⁸. For instance, one research group reported a 69% non-response rate in hamstring ROM following SS in individuals with chronic low back pain⁶⁴, despite observing statistically significant improvements in ROM at the group-level¹¹⁹. This significant incidence of non-response emphasizes that the benefits of SS are not uniformly experienced across all individuals, demonstrating that group averages can be misleading.

Given the considerable treatment response heterogeneity, or the clinically important individual differences in the magnitude of response to the same intervention¹²⁰, there is increasing recognition that such interventions should be individually tailored to optimize their effectiveness¹²¹. Accordingly, a growing body of research has begun classifying individuals as “responders” or “non-responders” to account for this interindividual variability and identify predictors of responsiveness. Factors such as baseline characteristics, sex, activity level, or other demographic or contextual factors (e.g., genetics) may influence an individual’s likelihood of responding to SS. Advancing toward personalized exercise prescriptions will require a deeper understanding of this interindividual variability and the determinants of responsiveness to SS¹²², thereby enabling more targeted and effective clinical interventions.

Responders are individuals who, at a given timepoint, exhibit positive changes in a variable of interest that exceed a predetermined threshold following an intervention¹²¹. These

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individuals, sometimes referred to as having “high sensitivity”^{121,123}, demonstrate a meaningful improvement in an outcome variable¹²⁴. For example, a responder to SS might display an increase in ROM beyond a defined threshold. Conversely, non-responders, or individuals with “low-sensitivity,” exhibit changes that fall below the distinct threshold^{121,123}, reflecting no meaningful improvement, no change at all, or even a negative response in the outcome variable^{124,125}. For example, a non-responder to SS may experience a minor improvement in ROM following the intervention below a threshold, no change in ROM, or a loss of ROM.

One commonly used threshold to determine response incidence is typical error (TE), which accounts for the role of measurement error. TE is advantageous over several other classification methods (**Table 2**) due to its consideration of both biological variation and technical error¹²⁶. Measurement error comprises two primary components: 1) validity error, or the difference between the observed and true values of a measurement (i.e., concurrent validity), and 2) reliability error, or the reproducibility of observed values across repeated measures¹²⁷. Biological variation (i.e., natural day-to-day fluctuations in true scores influenced by a variety of factors such as sleep, nutrition, motivation, physical activity etc¹²⁴; e.g., repeated ROM measurements being within a couple degrees) and random error (i.e., equipment noise) also contribute to overall measurement error¹²⁷.

Table 2. Summary of Methods Commonly Used to Classify Responders and Non-responders.

Name of Method for Classifying (Non-)Responders	Method Details
Typical error (TE) ¹²⁷	TE is the standard error of within-subject standard deviation from test-retest assessments of a measurement ¹²⁶ $TE = SD_{diff} / \sqrt{2}$ >±1 x TE considered a responder ^{126,128} >2 x TE considered a responder ^{129–133}

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Mean change ⁶⁴	Responder was considered a pre-post change equal to or greater than the mean change from the group-level analysis
Technical error of measurement (TEM) ^{134,135}	Calculated by taking the square root of the sum of squared differences of repeated measures divided by the total number of paired samples multiplied by 2: $TEM = \sqrt{((\sum(X_1 - X_2)^2)/2N)}$ >1 x TEM considered a responder¹³⁴ >2 x TEM in worsening direction considered adverse responder¹³⁵
Coefficient of variation (CV) ¹³⁶⁻¹³⁸	Expresses standard deviation (SD) of data as a percentage of its mean: $CV = 100 \times (\text{population mean} \times \text{within-subjects SD})^{137}$ >1 CV in positive direction= responder¹³⁸
Confidence intervals (CI) ^{127,139,140}	CIs of change in the control group were determined by Univariate General Linear Model. Upper 95% was used as lower limit for a significant individual training-induced change in training group. <upper limit of 95% CI=low responders^{127,139,140}
SD from the mean	Pre-post changes: < 1 SD = low responders¹⁴¹ >1 SD = high responders^{140,141}
Stratification of sample	Top and bottom 15-20% of sample = responders and non-responders, respectively¹⁴² Top and bottom quartiles = responders and non-responders, respectively¹⁴³
Minimal clinical important difference (MCID) or clinically important changes	Participants who experienced a change $\geq$ MCID ¹⁴⁴ or clinically important change ¹⁴⁵ were considered responders

In exercise science research, individual changes are often interpreted using the minimal clinically important difference (MCID), which reflects the minimum threshold for a clinically

meaningful improvement, such as decreased injury risk^{120,146}. This concept is particularly important when evaluating individual responses to SS interventions, as predetermined thresholds like TE may not align with the minimum change required to produce a meaningful clinical benefit. For example, if an individual's change surpasses the TE but does not exceed the MCID, it raises the question: is the observed change truly beneficial¹²⁰? The MCID is largely applied in patient-reported outcome measures¹⁴⁶; however, to the author's knowledge, MCID analyses do not appear in SS literature.

Various methods exist to determine the minimal important change (MIC) or MCID, including anchor-based, distribution-based, health economic, and standardized effect size approaches (**Table 3**). However, it has been argued that using group-derived MCID values to classify individual responders is problematic, as it can lead to overly optimistic estimates of treatment efficacy at the individual level¹⁴⁷. To address this, researchers recommend using alternatives such as the Reliable Change Index (RCI), which evaluates the statistical significance of individual-level changes in a manner analogous to t-tests for group-level comparisons¹⁴⁷. The RCI is calculated as:

$$RCI = (X_2 - X_1) / \sqrt{2 \text{ Standard error of measurement (SEM)}}$$

$$\text{Where SEM} = SD \sqrt{1 - \text{reliability}^2}$$

An RCI of 1.96 or greater indicates a responder¹⁴⁷.

Table 3. Summary of Methods Commonly Used to Determine the Minimal Clinically Important Difference.

Method	Description
Anchor	“Anchored” by using a patient’s or health professional’s judgement to define what an important difference is. For example, “a little better,” “a little worse,” “a lot better,” etc.
Distribution	Determine a value based upon distributional variation. A common approach is to use a value that is larger than the inherent imprecision in the measurement and therefore likely to represent a minimal level needed for noticeable difference (e.g., standard error of measurement or smallest detectable change).
Health Economic	Approaches that use principles in economic evaluation. These compare cost with health outcomes and define a threshold value for the cost of a unit of health effect that a decision-maker is willing to pay, to estimate overall incremental net benefit of one treatment versus the comparator.
Standardised Effect Size	The magnitude of the effect on a standardised scale defines the value of the difference (commonly expressed as Cohen’s <i>d</i>).

*Adapted from Cook et al. (2018)*¹⁴⁸

A central challenge in examining response variability is distinguishing true intervention effects from measurement error or natural biological fluctuations^{124,149}. To isolate true individual responses, it is recommended to compare the standard deviation of changes in the intervention group against that of a time-matched control group, which captures similar levels of measurement error and within-subject variability in the absence of the intervention^{124,150}.

Collectively, these approaches can help researchers and clinicians better understand the interindividual variability in response to SS interventions and support the development of more precise, individualized exercise prescriptions.

Pectoral Static Stretching

The pectoral muscle group, comprised of pectoralis major and minor, influence resting shoulder position and movement of the shoulder complex. A shortened pectoralis minor is associated with forward shoulder posture (FSP), which is characterized by an anterior deviation of the shoulder from the lateral plum line and abduction of the scapula^{54,55}. Collectively, this

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alters the movement or kinematics of the scapula⁵⁵ which may lead to the development of shoulder pathologies^{48–53}. Interestingly, the impact pectoralis major has on FSP and shoulder pathology is largely overlooked in research. Given its attachments from the clavicle/sternum and proximal humerus, a shortened pectoralis major may also contribute to FSP and altered scapular kinematics. As such, interventions aimed at lengthening the pectorals and decreasing FSP may be crucial for reducing the burden of shoulder injuries.

Pectoral SS is frequently prescribed by musculoskeletal therapists as a means of elongating the pectoral muscles, reducing FSP, and mitigating or rehabilitating shoulder pathologies. Specifically, passive horizontal abduction with the shoulder at 90° of abduction and external rotation is one of the most commonly prescribed pectoral SS¹⁵¹. However, little research evaluating the acute impact of this or similar pectoral stretches exists, as most evidence focuses on multi-week stretching protocols^{48,59–61} or a combination of stretching and exercise⁵⁷ or massage⁵⁸. For example, Lee et al., (2015) found that a 4x30s pectoral SS in addition to a scapular posterior tilting exercise acutely decreased FSP and increased pectoralis minor length compared to tilting exercise alone⁵⁷. Further, five sessions of pectoral SS improved scapular kinematics and FSP¹⁵², six sessions over three weeks reduced FSP⁶¹, and 5 sessions a week for 3 weeks increased pectoralis minor length and shoulder flexion ROM⁶⁰. In those with subacromial pain syndrome, pectoralis minor SS in addition to a standardized shoulder exercise program was compared to just the exercise program for 6 weeks, finding that, while the addition of the SS increased pectoralis minor length, there were no differences in pain reduction or functional improvement⁵⁹. Conversely, Rosa et al., (2017) examined a 6 week pectoral SS protocol in those with shoulder pain and those without, finding pectoralis minor length did not change in either group, but pain and function in the shoulder pain group improved⁴⁸. Collectively, this

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demonstrates that pectoral SS effectively increases pectoralis minor length^{57,59,60} and FSP^{57,61,152}. However, little research has assessed the acute impact of pectoral SS on muscle stiffness, FSP, pectoralis minor length, and ROM.

To the author's knowledge, only one research group has assessed the acute impact of pectoral SS on muscle stiffness to-date. Umehara et al., (2021)⁶³ conducted two studies that measured acute and prolonged effects of SS on pectoralis minor stiffness. Their first study measured stiffness before and after 10x30-second SS (total 5-minutes), as well as between each repetition. They found that stiffness decreased after the first repetition and continued to decrease gradually after each additional repetition. Completion of the third set (90s stretching) resulted in a more significant decrease in stiffness compared to the first repetition. In their follow-up study, pectoralis minor stiffness was measured before and up to 15 minutes after short (1x30s) or long (10x30s) duration pectoral SS. While they found that the long duration SS decreased stiffness immediately and up to 15 minutes after, the short duration's group immediate decrease in stiffness returned to baseline 5-minutes after the stretch. Furthermore, scapular motion was altered by increasing external rotation and posterior tilt during arm elevation⁶². While their pectoral SS executed at maximal intensity reduced stiffness of pectoralis minor, pectoralis major stiffness was not assessed. As such, it is unknown how different intensities of pectoral SS will affect pectoral stiffness, and the impact it may have on pectoralis minor length, FSP, and ROM. Moreover, the incidence of response to pectoral SS and the factors that contribute to response rate is unknown.

Purpose & Objectives

Literature consistently demonstrates that various SS protocols increase ROM^{3,5,83}, likely through improved stretch tolerance and reduced muscle stiffness^{6,86,114,153}. SS intensity, a

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modifiable dose parameter, is infrequently investigated due to its inherent subjectivity and challenges with standardization^{3,29}. While current evidence suggests that different SS intensities similarly improve ROM³, findings regarding their effects on stiffness and passive torque are inconsistent. For example, high intensity SS has been shown to both increase passive torque^{12,30,31} and decrease stiffness^{12,41}, while low³⁶ and moderate³³ intensities have also been associated with stiffness reductions. Moreover, both high and low intensities appear to decrease passive torque during stretching¹² and stiffness³², while similarly increasing passive torque^{32,41}. However, these findings are largely limited to the lower limb and often involve concurrent manipulation of other SS parameters (e.g., duration), making it difficult to isolate the effects of intensity^{30–32,38}. Consequently, significant gaps remain regarding the isolated influence of SS intensity on ROM and stiffness, particularly in the upper limb. Moreover, current evidence suggests that a substantial portion of individuals demonstrate non-response to SS despite mean group ROM improvements⁶⁴, though SS literature rarely reports incidence of response. To advance towards individualized exercise prescription, it is essential to understand if the impact on SS intensity on response rate, and the factors that may predict responsiveness.

SS is a foundational element of many preventative and rehabilitative protocols. Shortened pectoral muscles are associated with increased FSP^{54,55}, a factor implicated in the development of shoulder pathology^{48–53}. As such, SS is frequently prescribed to lengthen the pectoral muscles to prevent or rehabilitate shoulder pathology^{48,49,51–53}. Although acute SS performed to the point of discomfort has been shown to reduce pectoralis minor stiffness and alter scapular kinematics^{62,63}, limited research has examined how varying pectoral SS intensities affect ROM, pectoral stiffness, muscle length, or FSP. Given this context, the overarching goal of this dissertation was to examine group and individual responses to pectoral SS at various intensities.

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Study One. Given the current knowledge gaps regarding the impact of pectoral SS on ROM, pectoral stiffness, muscle length, and FSP, both generally and at different stretch intensities, the objective of study one (Chapter 3) was to:

1. Define the impact of an acute 3x30 second bout of passive pectoral SS at low and moderate intensities (110% and 120% of a baseline ROM) in asymptomatic shoulders on a) FSP; b) pectoralis minor length (PML); c) horizontal abduction range of motion (haROM); d) pectoralis major; and e) minor stiffness.

Given that SS at various intensities have previously been reported to reduce muscle stiffness^{12,32,33,36,41,86,114} and increase ROM³, it was hypothesized that both SS intensities would decrease pectoral stiffness, increase haROM, and thus increase PML and reduce FSP.

Study Two. Study one identified the necessity to implement a more ecologically valid, intense pectoral SS protocol that compared changes to a control condition. Given that SS is clinically prescribed based on subjective patient discomfort, it is imperative to understand how patient reported intensity influences outcomes such as ROM and stiffness, and how changes in in subjective discomfort relate to changes in these variables. As such, the primary objectives of study two (Chapter 4) were to determine:

1. The impact of an acute 3x30 second bout of a standing pectoral SS at moderate and maximal intensities using a standardized stretch intensity scale on: a) FSP; b) PML; c) horizontal abduction range of motion (haROM); d) pectoralis major; and e) minor stiffness, compared to a control.
2. The relationship between changes in participant reported stretch intensities throughout SS and pre-post changes in variables a-e.

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Previous work has demonstrated reductions in pectoralis minor stiffness following high intensity SS⁶³, and Study One demonstrated that low and moderate intensity pectoral SS decreased both pectoralis minor stiffness and FSP, with greater reductions in FSP following moderate intensity (Chapter 3). Thus, it was hypothesized that:

1. Both SS intensities will decrease pectoralis minor stiffness and FSP (with maximal intensity decreasing FSP more), increase ROM, given various SS intensities increases ROM³, and not alter PML and pectoralis major stiffness, compared to a control.
2. Changes in patient reported intensity will be greater following maximal stretching, and will be positively correlated with changes in FSP, but not with changes in muscle stiffness, PML or haROM.

While increases in shear wave velocity reflects the magnitude of tissue elongation during SS, it is unclear if elongation differs significant during different stretch intensities. Thus, a third objective was to determine:

3. If the magnitude of elongation (i.e., stiffness) of pectoralis major and minor differs during moderate and maximal SS intensities.

It was hypothesized that pectoralis major and minor stiffness will be higher during maximal SS intensity, indicating greater elongation.

Study Three. Based on the results of studies one and two, it was evident that interindividual variability in responses to SS existed for all variables. To better inform individual exercise prescription practice, further work examining factors that may predict responsiveness such as baseline characteristics is warranted. Thus, the objectives of Study Three (Chapter 5) were to:

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1. Determine the incidence of individual response or non-response of a) FSP; b) PML; c) horizontal abduction range of motion (haROM); d) pectoralis major; and e) minor stiffness to moderate and maximal pectoral SS intensities.
2. Determine the difference in baseline data of dependent variables between responders and non-responders to moderate and maximal intensities of pectoral SS.

Given the significant incidence of non-response reported previously with SS⁶⁴ and the data trends noted in study one and two (Chapter 3 and 4), it was hypothesized:

1. Individual response incidence for both SS intensities will be low for all variables, and that greater response rates will occur with higher SS intensities for FSP.
2. That baseline values will differ between responders and non-responders for a given variable.

Lastly, individual baseline values may determine the amount of change observed for a given variable. As such, a final objective was to:

3. Explore the relationship between participants' baseline values for each outcome variable and the amount of change they experienced following the SS intervention.

Given that the baseline value has previously been shown to affect the magnitude and direction of response or change in an outcome variable following an intervention^{154,155}, it was hypothesized that there will be a rate-dependent effect between participants' baseline values and change in all outcome variables.

Chapter 3: Comparison of two pectoral stretch intensities on shoulder posture, range of motion, muscle length, and stiffness: A randomized, single blinded crossover study.

Author Note & Contribution

Data and text from the following manuscript appears in:

Bohunicky, S., Franklin, R., Wang, S., Fulawka, K., Glazebrook, C. M., Ogborn, D. I., & Scribbans, T. D. (2025) Comparison of two pectoral stretch intensities on shoulder posture, range of motion, muscle length, and stiffness: A randomized, single blinded crossover study. *SportRxiv*. <https://doi.org/10.51224/SRXIV.653>

Sarah Bohunicky contributed to the study conceptualization, design, and piloting of methods, and led the data collection, data analysis, interpretation of results, and draft of manuscript.

Abstract

Various combinations of static stretching (SS) duration and repetitions increase range of motion (ROM); however, limited data exist on the effects of SS intensity, particularly in the upper extremity. Twenty-four participants ($n = 24$, 25 ± 5 years) with asymptomatic shoulders attended two sessions, receiving pectoral SS at 110% and 120% of a baseline ROM (bROM; angle of horizontal abduction a stretch sensation was first perceived). Forward shoulder posture (FSP), pectoralis minor length (PML), horizontal abduction ROM (haROM), and pectoralis major and minor stiffness (via shear wave elastography) were measured before (PRE) and after (POST) each intervention. SS interventions were three sets of 30 seconds of passive horizontal abduction with 30s of rest between. A two-way (2 intensity by 2 time) repeated measures ANOVA was conducted on all variables ($p < 0.05$). A significant two-way interaction was found for FSP, $F(1,23) = 6.21$, $p = 0.02$, $\eta_p^2 = 0.213$, with reductions following both 110% ($p = 0.025$) and 120% ($p < 0.001$). No significant interactions were found for PML, haROM, or pectoralis major and minor stiffness. Pectoralis minor stiffness decreased from PRE to POST ($p = 0.049$). A single SS bout reduced FSP and pectoralis minor stiffness. Higher intensity SS decreased FSP more than lower intensity SS, while pectoralis minor stiffness decreased regardless of intensity. These changes in FSP and pectoralis minor stiffness appear to be independent from changes in PML, haROM, and pectoralis major stiffness.

Introduction

Acutely, static stretching (SS) increases range of motion (ROM)^{3,5} due to increased stretch tolerance and reduced muscle stiffness^{6,153}. To increase ROM, clinicians instruct individuals to position themselves where a stretch sensation is felt or when a certain stretch intensity has been reached, often aiming for discomfort with no pain. While SS is a central component of most preventative and rehabilitative approaches, little work has defined the impact of SS intensity on the effects observed following SS^{3,5} (e.g., ROM, muscle stiffness, etc.). Thus, additional work is needed to define how SS intensity alters responses to SS to inform exercise prescription recommendations.

SS is a common approach used to lengthen the pectoral muscles to reduce forward shoulder posture (FSP) to prevent or rehabilitate shoulder pathology^{48,49,51,52}. Shortened pectorals are associated with increased FSP⁵⁴ which may increase the risk of shoulder pathology⁴⁸⁻⁵². Horizontal abduction with the shoulder and elbow flexed to 90° is a commonly prescribed stretch to lengthen the pectorals¹⁵¹, yet limited research has evaluated the impact of acute pectoral SS on outcomes such as pectoral stiffness, pectoral length, or FSP. Pectoralis minor stiffness is reduced immediately after one 30-second (30s) bout of pectoral SS (horizontal abduction with shoulder at 120° flexion) at the point of discomfort (POD) and significantly more after a third 30s bout (cumulative 90s)⁶³. Further, 10x30s of high-intensity pectoral SS acutely decreases pectoralis minor stiffness⁶². However, pectoralis major stiffness was not measured. Given that pectoralis major attaches from the sternum to the proximal humerus, increased stiffness may impact FSP, and it is currently unknown how pectoral SS affects the muscle's stiffness. Thus, while available literature supports that high intensity pectoral SS reduces pectoralis minor stiffness, it is not

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known how different SS intensities affect pectoralis major stiffness, FSP, ROM and muscle length.

As greater stretch intensities may cause pain and muscle soreness²⁹, lower intensity stretches may be preferable if they produce the same benefits as higher intensities. However, SS research often employs a high intensity SS (e.g., $\geq 100\%$ POD), and little acute SS research has examined responses to differing intensities as other dose factors such as sets, and duration are more easily manipulated. Moreover, stretch intensity is subjective, making it more difficult to standardize^{3,29}. Available literature suggests that high intensity SS increases ROM^{12,30,31,33} and passive torque^{12,30,31} and decreases stiffness^{12,41} and muscle force^{12,38} compared to low intensity. Further, low intensity SS decreases quadricep stiffness³⁶ compared to high intensity, while moderate intensity decreases quadricep stiffness compared to a low and high intensity³³. Additionally, high- and low-intensity SS decreases static passive torque during stretching¹² and stiffness³², and increases passive torque^{32,41} and ROM similarly^{32,36,38,41}. Importantly, many of these studies^{30-32,38} manipulated more than one variable (intensity and duration) making it challenging to isolate the effects of SS intensity. A recent review concluded that while SS increases ROM, there is insufficient evidence to conclude that different intensities lead to alternative changes³, which may be the result of inconsistencies with reporting or prescribing stretch intensity between studies²⁷. As such, considerable gaps in our knowledge persist regarding the impact of SS intensity on ROM and stiffness, especially in the upper limb.

Thus, the current study defined the impact of an acute bout of passive pectoral SS at two intensities (110% and 120% of baseline ROM) in asymptomatic shoulders on: 1) FSP; 2) pectoralis minor length (PML); 3) horizontal abduction range of motion (haROM); 4) pectoralis major; and 5) minor stiffness. Given that high- and low-intensity SS has been reported to reduce

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muscle stiffness^{12,32,33,36,41} and increase ROM³, it was hypothesized that both SS intensities would decrease pectoralis major and minor stiffness, increase haROM, and thus increase PML and reduce FSP.

Methods

Experimental overview

A repeated measures crossover design was conducted where participants attended two experimental sessions in a research laboratory at least 72 hours apart (**Figure 4**). Participants received one of two researcher-administered static pectoral stretches at a lower and higher intensity: 110% and 120% of a baseline ROM (bROM). These were delivered in a random order to a randomized arm at each session. FSP, PML, haROM, and pectoralis major and minor stiffness were measured before (PRE) and immediately after (POST) each intervention by a single, trained examiner (**Figure 5**). At the start and end of each stretch, participants reported their perceived stretch intensity using a visual analogue scale from 0 (no stretch) to 100 (extremely painful stretch).

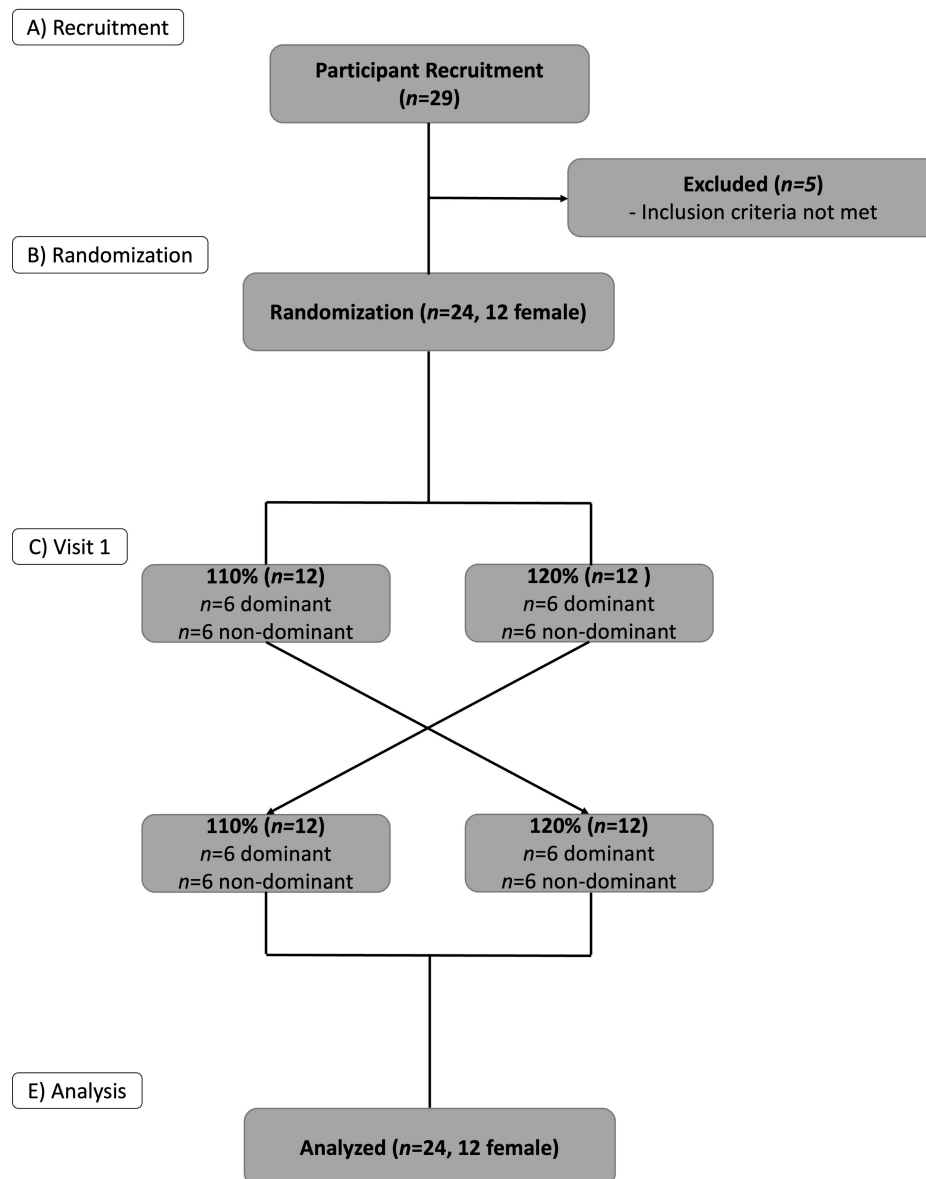


Figure 4: CONSORT Diagram

A) Twenty-nine individuals were recruited, five were excluded due to not meeting the inclusion criteria. B) Twenty-four participants were eligible and randomized to an intervention (110% and 120%) and arm (dominant or non-dominant). C) During their first visit, twelve participants received the 110% intervention (n=6 dominant arm, n=6 non-dominant arm), and twelve participants received the 120% intervention (n=6 dominant arm, n=6 non-dominant arm). D) Twenty-four participants attended a second session where they received the other intervention (110% or 120%) to the opposite arm (dominant or non-dominant) as Visit 1 (C). E) All data for every participant and outcome variable was analyzed.

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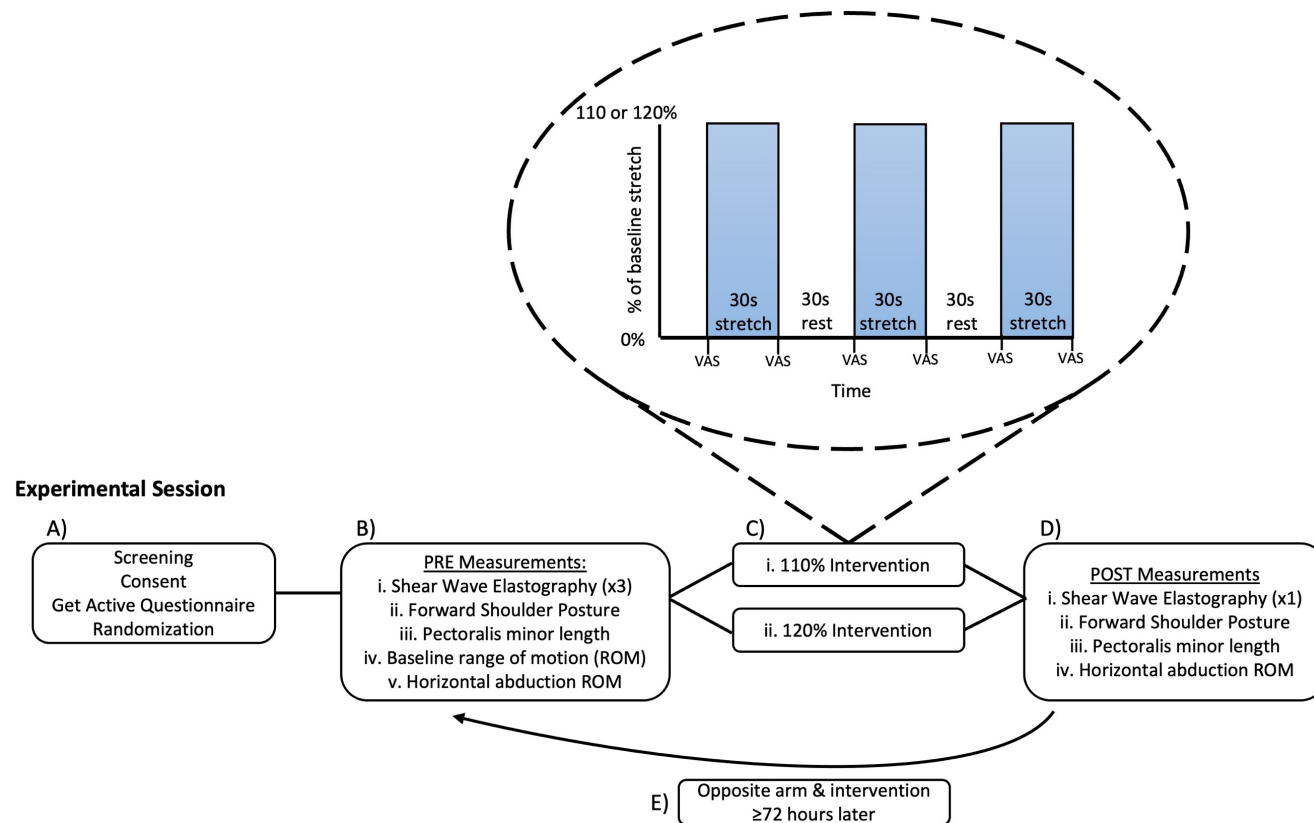


Figure 5: Timeline Schematic of Experimental Protocol

A) Participants were screened for eligibility, provided informed consent, filled out a Get Active Questionnaire, and underwent intervention arm and static stretch (SS) intensity randomization. B) PRE measurements: Shear wave elastography (x3 elastograms for reliability analysis), forward shoulder posture (FSP), pectoralis minor length (PML), baseline range of motion (bROM), and horizontal abduction range of motion (haROM) were measured. C) Participants received the 110% or 120% of bROM SS for 3x30s with 30s of rest between. Perceived SS intensity was collected at the start and end of each 30s SS using a 0-100 visual analogue scale (VAS). D) POST measurements were taken in the same manner as (B) without bROM, and only one elastogram was taken. E) Participants returned a minimum of 72 hours later and repeated B-D at the other SS intensity and other arm (C).

Participants

To detect an interaction between intervention (110% and 120%) and time (PRE and POST), an a priori analysis ($\alpha = 0.05$, $\beta = 0.8$) was performed using a moderate effect size of $\eta^2 = 0.06$ which yielded a sample size of 24. A convenience sample of 29 participants was recruited from the University of Manitoba and surrounding community between February and July 2023. Potential participants were assessed for eligibility through the following inclusion criteria prior to their first experimental session: 18-40 years old; no recent history (< 6 months) of pain, injury, or orthopedic disorders to either arm; no diagnosed neurological or musculoskeletal disorders; no contraindications to the use of ultrasound; no history (< 5 years) of participation in sport/activities requiring extreme upper body ranges of motion (e.g., gymnastics, yoga, baseball pitcher); not currently pregnant or breast feeding; and ≤ 5 Beighton's score for joint hypermobility (as assessed during the first session).

Twenty-four ($n = 24$, 12 females, age = 25 ± 5 years, height = 173 ± 10 centimeters, weight = 75 ± 20 kilograms) participants were eligible and participated. Handedness was determined using the Modified Edinburgh Handedness Index and participants completed the Canadian Society for Exercise Physiology's Get Active Questionnaire to determine activity level and identify any special considerations to receiving a stretch intervention (i.e., low blood pressure that might result in light-headedness when getting up). Participants were asked to refrain from upper body resistance training for 48 hours prior, cannabis and alcohol consumption for 24 hours prior, and caffeine consumption for six hours prior to each session. All experimental procedures were approved by the University of Manitoba Research Ethics Board and were conducted in accordance with the Declaration of Helsinki. Participants received all verbal and

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written details of the experimental procedures prior to providing informed consent, including the benefits and risks of the investigation.

Procedures

Once consented, participants' intervention (110% and 120%) and arm (dominant or non-dominant) order were block randomized and counterbalanced based on hand dominance. PRE measurements of pectoralis major and minor stiffness via shear wave velocity (SWV), FSP, PML, baseline ROM (bROM), and haROM were captured prior to each stretch intervention. Participants received the first stretch intervention on one arm, and the second stretch intervention at the second experimental session on the other arm. Participants were informed that they will be receiving two different intensities of SS, but were not told which intensity they would receive at either session. After the intervention, POST measurements were taken in the same order excluding bROM. Participants' height, weight, age, and sex assigned at birth were collected.

Shear Wave Elastography (SWE)

Pectoralis major and minor stiffness was measured using shear wave elastography (SWE; Logiq E10s, GE, Mississauga, Ontario, Canada), coupled with a transducer (2-10 MHz, XD clear linear probe, GE, Mississauga, Ontario, Canada), where the measured SWV represents stiffness. Participants sat on a plinth that was reclined to 30° with a bolster under their knees and arms rested by their sides (**Figure 6a**). Transmission gel coupled the probe approximately 1 inch below the clavicle and 1 inch lateral to the sternal edge (**Figure 6b**). The probe was aligned longitudinal to pectoralis major and minor fibers separately using B-mode ultrasound and recorded with permanent marker for consistency between measurements.

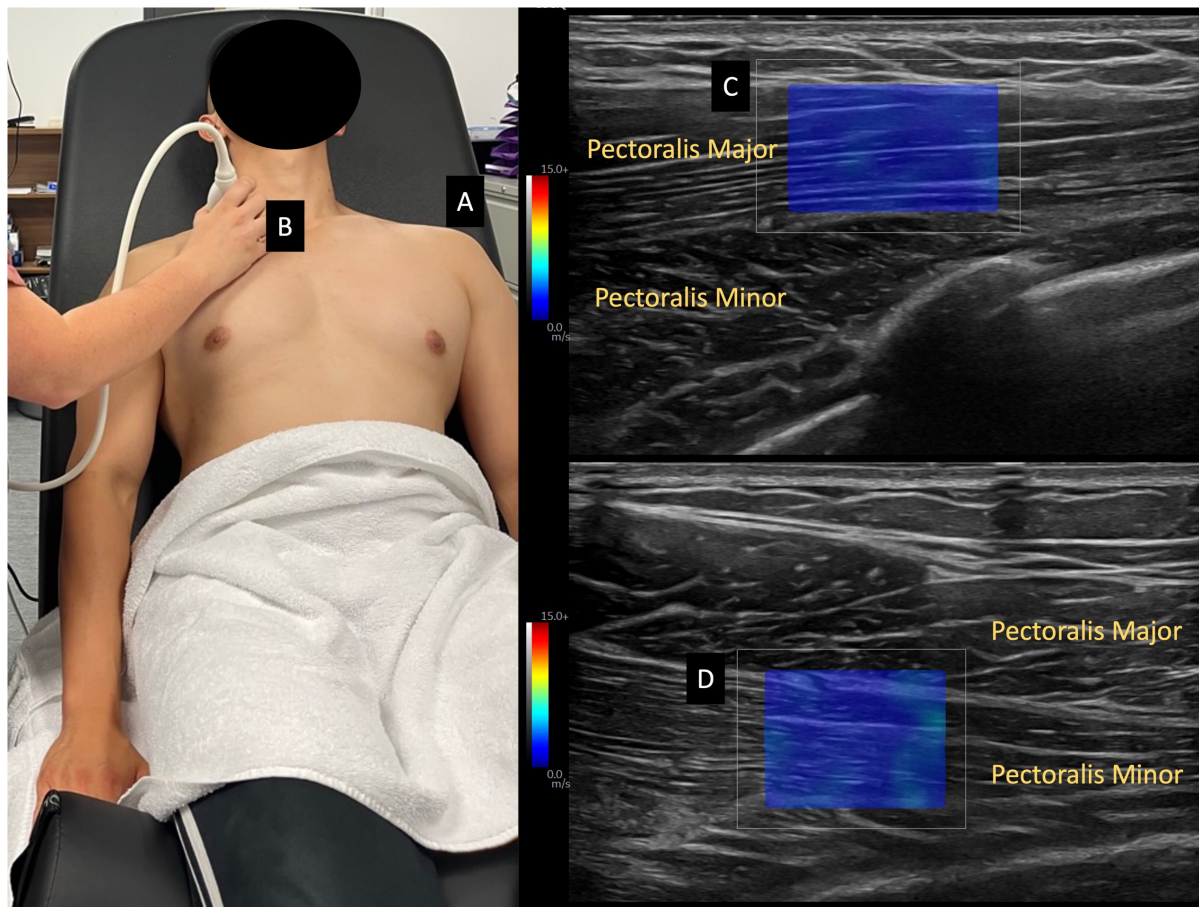


Figure 6: Shear Wave Elastography for Pectoralis Major and Minor Stiffness

Pectoralis major and minor muscle stiffness was measured using shear wave velocity (SWV). Participants sat on a 30° reclined plinth (A) with a bolster under their knees and arm rested by their sides. Using B-mode ultrasound, a 2-10 MHz transducer was aligned longitudinally with muscle fibres of the respective muscle being measured, approximately 1-inch below the clavicle and 1-inch lateral to the sternal edge (B). The appropriate probe location was marked and an elastogram was taken of pectoralis major while participants held their breath (C). Colour-coded elastograms of SWV within a region of interest (ROI) were produced, and three high-quality images (with little artifact) were selected and used for analysis. This was then repeated with pectoralis minor (D).

Given that muscle stiffness increases linearly with force production¹⁵⁶, it was necessary to ensure stiffness was measured in a rested state. Thus, muscle excitation of the pectoralis major was measured in 10 unique participants ($n = 10$, 5 female, 5 male; age: 27 ± 6 years) in the same position as SWV measurements. Bipolar surface electromyography (Quattrocento, OT Bioelettronica, Torino, Italy) was recorded for 10s, where the average root mean square (RMS) from seconds three to six was used for analysis. A maximal voluntary isometric contraction

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(MVIC) of the pectorals was used as a reference contraction to determine relative excitation at rest. Participants were seated at a chest press machine loaded with an immovable weight and were instructed to press as hard as possible for four repetitions of 5-second, all-out pushes. A 2-minute rest period was given between repetitions. The first repetition served as familiarization, and the middle three seconds of each of the last three repetitions were evaluated for the peak excitation. The average RMS of 0.5s around the highest peak was calculated and revealed that pectoralis major excitation was negligible in the rest position ($0.72 \pm 2.85\%$).

Shear wave mode was used to produce a colour-coded map (elastogram) of SWV within the largest region of interest (ROI) possible for each muscle (**Figure 6C and D**). To determine reliability of SWV measurements, three, ~10-second elastograms were captured for both muscles at PRE, while one ~10-second elastogram was captured at POST. Participants were instructed to take a deep breath in, exhale for a count of 3-seconds, and then hold for ~10-seconds as the elastogram was taken to limit movement during breathing⁶³. Three high-quality images with the least artifact (none or very little monochromatic artifact) were saved from each elastogram and used for analysis.

Forward shoulder posture (FSP)

FSP was measured using the double-square method. Participants stood with their back against a wall in a relaxed/neutral posture and the anterior acromion was marked. A modified combination square was used to measure the distance from the wall to their acromion three times, where the average was used for analysis. Between each measurement, participants were instructed to step forward and shake their arms to reset their natural posture.

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Pectoralis minor length (PML)

PML was measured in supine. Participants had their coracoid process and anterior-inferior edge of the 4th rib one finger-width lateral to the sternum marked with permanent marker. A flexible measuring tape was used to measure this distance three times, where the average of the three measurements was used for analysis.

Baseline range of motion (bROM)

To calculate the SS angle for each intervention, bROM was the haROM position where participants first felt a stretch in their pectoral region (**Figure 7**). Horizontal abduction, with the shoulder abducted to 90° or 150° and elbow flexed at 90°, has been shown to effectively stretch pectoralis minor⁵⁰. To target both pectoralis minor and the horizontal fibres of pectoralis major, the current study utilized a stretch position at 90° of shoulder abduction. While supine with their shoulder slightly over the edge of the plinth to allow for full horizontal abduction, each participant's relaxed arm was slowly passively horizontally abducted at 90° of shoulder and elbow flexion until the participant reported a stretch sensation in the pectoral region. This angle was measured using a goniometer and considered their bROM to calculate the 110% (x1.1) and 120% (x1.2) stretch intensities. Goniometric measurements were taken by one of two experienced researchers (> 3 years). Given that goniometry has high reliability when conducted by the same individual¹⁵⁷, all measurements for a given participant were conducted by the same researcher. Intra-rater reliability was calculated for both independent researchers, demonstrating excellent reliability (R1 Cronbach's $\alpha = 0.915$, R2 $\alpha = 0.982$).

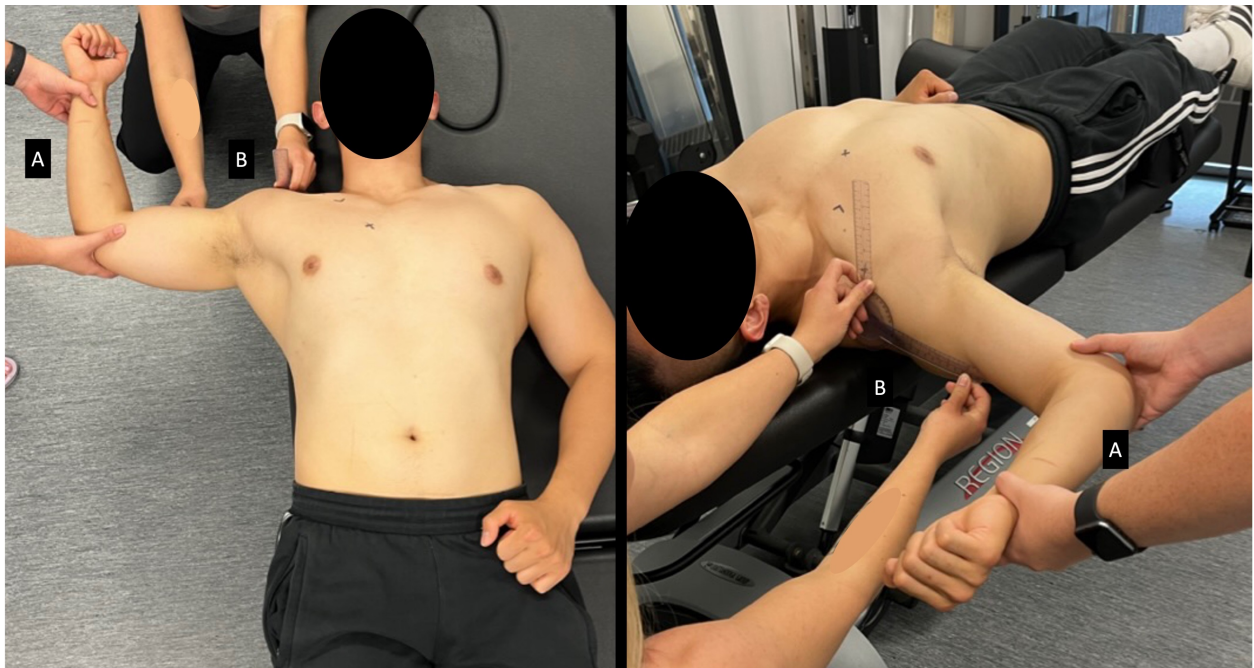


Figure 7: Position of baseline range of motion measurement (bROM), horizontal abduction range of motion measurement (haROM), and stretch intervention.

Participants' intervention shoulder and elbow were flexed to 90° (A). For the baseline range of motion (bROM) measurement, the researcher slowly horizontally abducted the participants' arm until the first sensation of a stretch in the pectoral region was felt. This angle was measured with a goniometer (B). Horizontal abduction range of motion (haROM) was measured in the same position, except the participant's relaxed into end range passive horizontal abduction, which was measured with a goniometer (B). The participants' arm was assisted back to 90° of shoulder and elbow flexion, where this was repeated twice more for a total of three times, where the average of 3 was used for analysis. The stretch intervention was also performed in this position. Participants' arms were horizontally abducted to either 110% (x1.1) or 120% (x1.2) of bROM (B). This position was held for 30 seconds before their arm was returned to rest by their side for 30 seconds. This was for a total of 3 sets of 30 seconds of stretching.

Horizontal abduction range of motion (haROM)

Passive horizontal abduction ROM (haROM) was measured in the same arm position as bROM (Figure 7). Each participant's arm was passively horizontally abducted with the shoulder and elbow at 90° of abduction and flexion, respectively. Participants were instructed to relax the arm towards the ground. The researcher maintained light touch with the participant's arm as they reached their full passive range of motion to maintain proper position (i.e., keeping their arm

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parallel to the ground to control shoulder rotation). The researcher did not apply any force to the participant's arm to increase range. Once the arm settled, haROM was measured with a goniometer. The participant's arm was then passively horizontally adducted to 90° of flexion before repeating this process twice for a total of three times with the average used for analysis.

Stretch intervention

Participants received three repetitions of a 30-second SS with 30-seconds of rest between each repetition at 110% or 120% of their bROM. SS doses of 3-4x30s are commonly recommended¹⁵⁸, and reduce pectoralis minor stiffness⁶³. The researcher responsible for conducting outcome variable measurements and data analysis exited the room during the stretch intervention to ensure blinding to intervention randomization. The SS was administered in the same way as the bROM (**Figure 7**), except that the shoulder was horizontally abducted to the prescribed range for each SS intensity. Participants were asked to relax and to inform researchers if pain exceeded a 7/10, in which case the stretch would be terminated ($n = 0$). After each repetition, the participant's arm was returned to their side to rest for 30-seconds.

Perceived stretch intensity

Participants were asked to rate their perceived stretch intensity at the start (~1-second) and end (~29-seconds) of each stretch repetition using a 100-point VAS where 0 represented no stretch and 100 represented an intolerable stretch (i.e., painful). These scores were averaged to yield one perceived intensity score for each intervention.

Second experimental session

Participants attended a second experimental session a minimum of 72 hours after the first which was identical to the first, except that the participants received the second SS intervention intensity to the other arm.

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Data processing

Shear wave velocity (m/s; i.e., muscle stiffness) was measured using the shear wave propagation speed computed by the system software by an investigator blinded to the intervention. Within the ROI for each set of measurement images (i.e., PRE or POST intervention for pectoralis major or minor), the largest possible area was measured that avoided artifact, and the diameter and measurement location was kept consistent between images for the same time point for a given participant. The average of the three measurements was used for analysis.

Statistical Analyses

Statistical analysis was performed using SPSS (v25, Chicago, IL, USA). Prior to statistical analysis, normality of the data was determined using Shapiro-Wilk test for normality ($p > 0.05$). Outliers were considered to be studentized residuals greater than ± 3 standard deviations. Data that was not normally distributed was transformed by log10 transformation.

A two-way (intensity*time) repeated measures ANOVA was conducted on PRE and POST measurements of all dependent variables (FSP, PML, haROM, pectoralis major and minor velocity). Data were considered statistically significant if $p < 0.05$. Post-hoc Bonferroni corrected t -tests were conducted on significant interactions to determine main effects. A paired samples t -test was conducted on the perceived stretch intensity between SS intensities. Small, medium, and large effect sizes were interpreted as: $\eta p^2 = \leq 0.01, \geq 0.06, \geq 0.14$, respectively; and $d = \leq 0.2, \geq 0.5, \geq 0.8$, respectively. Intrasection reliability of the dependent variables were determined by a two-way mixed model absolute agreement intraclass correlation using participants' repeated PRE-measurements from their first session. ICC values of $\leq 0.5, 0.5-0.75$,

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0.75-0.9, and ≥ 0.9 indicated poor, moderate, good, and excellent reliability, respectively. All data is presented as mean $\pm$ standard deviation unless otherwise stated.

Results

Raw data is available in the Open Science Framework repository (<https://osf.io/pbva8/>).

There were no outliers as assessed by studentized residuals ± 3 standard deviations. Data was not normally distributed for pectoralis major and minor SWV as assessed by Shapiro-wilk test for normality ($p < 0.05$) and thus a log10 transformation was applied, which corrected distribution ($p > 0.05$). Statistical analyses were conducted on transformed data where applicable. Data is presented in **Table 4** and **Figure 9** contains raw data.

There was a significant two-way intensity by time interaction for FSP, $F(1,23) = 6.21$, $p = 0.02$, $\eta_p^2 = 0.213$ (**Table 1; Figure 8**). Post hoc testing revealed FSP decreased following the 110% ($p = 0.025$, 95% CI 0.02 to 0.30, $d = 0.11$) and 120% ($p < 0.001$, 95% CI 0.21 to 0.52, $d = 0.16$) SS, though 120% reduced FSP greater than 110%. There was no difference in FSP at PRE ($p = 0.314$) or POST ($p = 0.825$) between experimental sessions.

There were no significant interactions between SS intensity and time for PML (**Figure 9a**), haROM (**Figure 9b**), pectoralis major (**Figure 9c**) or minor stiffness (**Figure 9d; Table 4**).

Analysis of main effects demonstrated pectoralis major stiffness approached significance over time ($p = 0.077$, $\beta = 0.425$) (**Figure 9c**). There was a significant main effect for pectoralis minor stiffness over time (mean difference = 0.214, SD = 0.095, $p = 0.049$, 95% CI: 0.17 to 0.410 m/s, $\eta_p^2 = 0.159$, $\beta = 0.514$) (**Figure 9d**).

Perceived stretch intensity was significantly greater for the 120% SS (31 ± 19) compared to the 110% SS (20 ± 17), $t(23) = 2.906$, $p = 0.008$, 95% CI 3 to 19, $d = 0.44$. ICC values for all

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outcome variables were good (pectoralis major SWV Cronbach's $\alpha = 0.885$) and excellent (pectoralis minor SWV $\alpha = 0.917$, FSP $\alpha = 0.994$, PML $\alpha = 0.999$, haROM $\alpha = 0.981$).

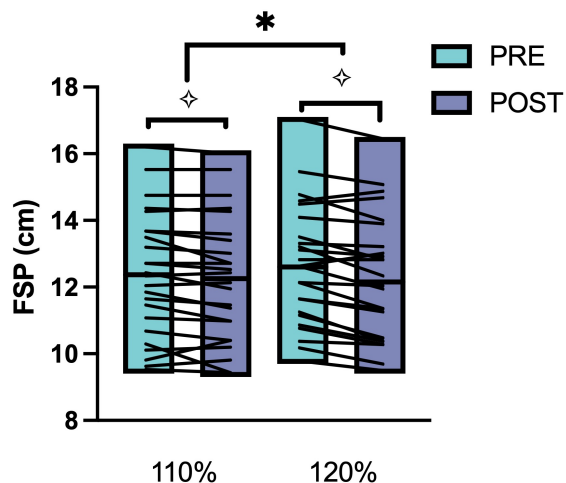


Figure 8: Forward Shoulder Posture (FSP) before (PRE) and after (POST) 3x30s of Pectoral Static Stretching at 110% and 120%.

$n=24$, cm =centimeters, * denotes significant 2-way interaction, ◊ denotes significant difference between PRE and POST.

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Table 4. Two-Way Intensity*Time Repeated Measures ANOVA: Forward Shoulder Posture, Horizontal Abduction Range of Motion, Pectoralis Minor Length, and Pectoralis Major and Minor Stiffness before (PRE) and after (POST) Pectoral Static Stretching at 110% and 120%.

	110%		120%		<i>f</i>	<i>p</i>	η_p^2	β
	PRE	POST	PRE	POST				
FSP (cm)	12.4 (1.9)	12.2 (1.9)	12.6 (1.9)	12.3 (1.9)	6.21	.02*	.213	.665
PML (cm)	14.3 (2.0)	14.3 (1.9)	14.3 (1.8)	14.3 (1.9)	.273	.607	.012	.079
haROM (degrees)	110 (10)	111 (10)	111 (11)	112 (12)	.408	.529	.017	.094
PMS (m/s)	3.12 (.91)	2.82 (.55)	3.00 (.85)	2.81 (.48)	.307	.585	.013	.083
PmS (m/s)	2.79 (.85)	2.51 (.58)	2.58 (.75)	2.44 (.62)	.462	.503	.02	.514

110%= 110% static stretch (SS); 120% = 120% SS; cm= centimeters; FSP= forward shoulder posture; haROM= horizontal abduction range of motion; m/s= velocity, meters per second; PMS= pectoralis major stiffness (shear wave velocity); PmS= pectoralis minor stiffness (shear wave velocity); PML= pectoralis major length; POST= Post-SS measurement; PRE = Pre-SS measurement. * $p < .05$.

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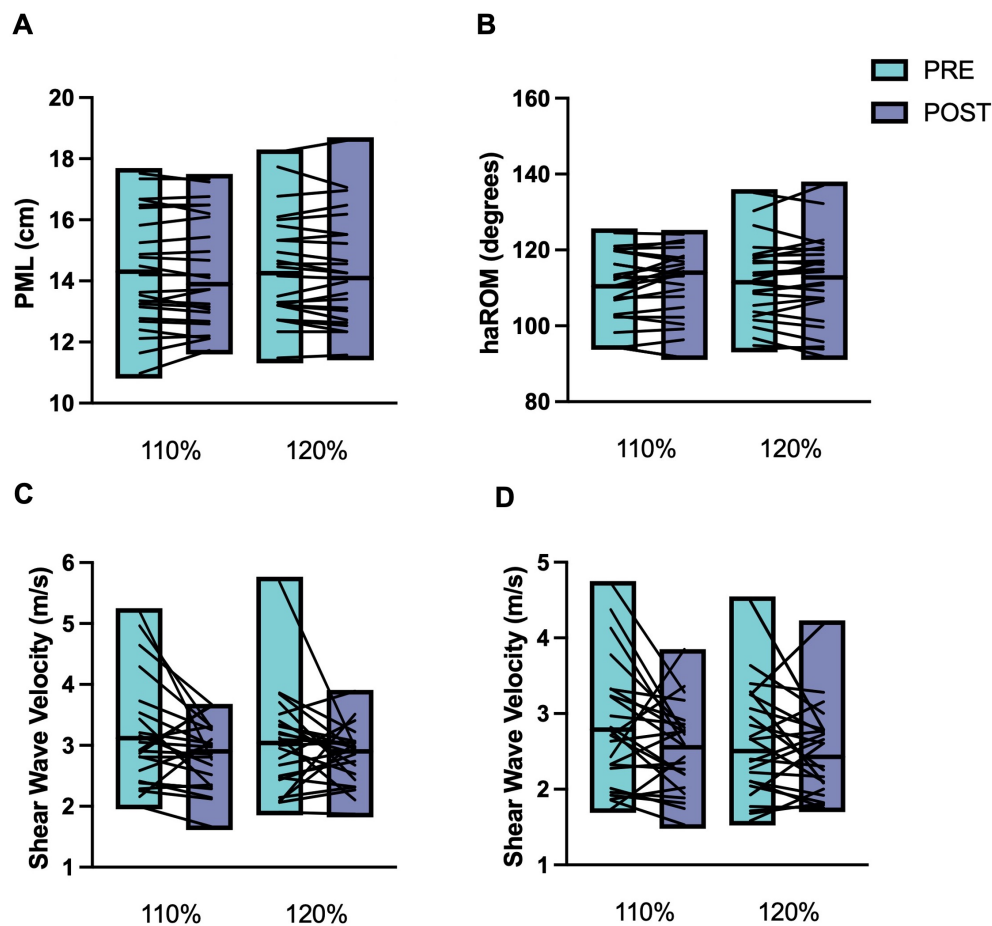


Figure 9. Pectoralis Minor Length (A), Horizontal Abduction Range of Motion (B), Pectoralis Major (C) and Pectoralis Minor Stiffness (D) before (PRE) and after (POST) 3x30s of Pectoral Static Stretching at 110% and 120%.

n=24, cm=centimeters; haROM = horizontal abduction range of motion; m/s = meters per second; PML = pectoralis minor length.

Discussion

The current study explored the impact of two different intensities of acute passive pectoral SS to asymptomatic shoulders on: FSP, PML, haROM, and pectoralis major and minor stiffness. In line with the hypothesis, the 120% SS reduced FSP to a greater extent than 110% SS. While pectoralis minor SWV decreased from PRE to POST as hypothesized, only the 110% SS intensity approached a significant reduction in stiffness. Further, neither intervention affected

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PML, haROM, or pectoralis major stiffness. To the authors' knowledge, this is the first study to examine the impact of different pectoral SS intensities on FSP, PML, and SWV of the pectoral muscles.

Our observation that both SS interventions significantly reduced FSP is consistent with previous reports demonstrating various massage therapy techniques^{159,160} applied to the pectoral region acutely decrease FSP. Interestingly, no published literature to the authors' knowledge examines the impact of a single pectoral stretch intervention on FSP. A single treatment of stretching and soft tissue mobilization of the pectoralis minor and SS immediately decreased FSP⁵⁸ but the relative contributions of each treatment to the observed reduction in FSP is unknown. Thus, our observed reduction in FSP after both SS interventions suggest SS may be capable of reducing FSP similarly to massage techniques^{58,159–161} alone and in conjunction with SS⁵⁸. Considering FSP is associated with shoulder injuries^{48–53}, simple and accessible rehabilitative and preventative strategies that can decrease FSP are essential to reducing the incidence and risk of shoulder pathology. Contrary to the hypothesis, more intense SS reduced FSP to a greater extent than the lower intensity SS (110%: -1.6%; 120%: -2.4%). While no previous studies report an intensity-dependent reduction in FSP following SS, higher intensity SS has increased ROM greater than a lower intensity SS^{12,30–33}.

Given that pectoralis major and minor attach to the proximal humerus and scapula from the sternum/ribs, respectively, we expect that the reduction in FSP following SS would be the result of increased length of the pectoral muscles. However, we observed no difference in PML or haROM, which suggests the reduction in FSP is the result of a different mechanism. For example, participants could have subconsciously corrected their FSP after the interventions. This is supported by previous work reporting FSP was immediately reduced in those in the control

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group¹⁶⁰. Further, we have previously observed that FSP decreased immediately after a pectoral myofascial release without a concurrent increase in ROM¹⁶⁰, which is consistent with the current study's findings. Moreover, while FSP was significantly lower after both SS, it is unknown if this change would meet the threshold for a minimal clinically important change that would reduce shoulder pathology risk, or how long FSP would remain reduced. Future studies should determine the mechanisms that control FSP (e.g., altered neuromuscular properties, reduced fascial stiffness, etc.), interventions capable of modifying them, and the minimum clinically important difference required in each to reduce shoulder pathology risk.

Given that a shortened pectoralis minor is associated with increased FSP⁴⁹, it is surprising that our observed decrease in FSP did not occur concomitantly with an increase in PML. These findings are opposed to those of previous research demonstrating pectoral stretching acutely increases PML¹⁶². Interestingly, pectoralis minor stiffness decreased from PRE to POST SS. Considering stiffness is an indicator of the viscoelastic properties of a muscle and a decrease in stiffness would indicate an increase in passive tension¹⁵, we would expect that a decrease in stiffness would occur parallel to an increase in pectoralis minor length. To evaluate the relationship between pectoralis minor length and stiffness, we performed an exploratory post-hoc Pearson Correlation between change in PML and change in pectoralis minor stiffness. There was no relationship between change in PML and change in pectoralis minor stiffness ($p = 0.866$, $r = -0.025$). As such, it seems pectoralis minor stiffness is unrelated to changes in its resting length.

We observed a main effect of time on pectoralis minor stiffness, which is consistent with previous literature demonstrating that pectoralis minor stiffness decreases after pectoral SS^{62,63}. For example, 30s of pectoral SS with the shoulder abducted at 135° and the arm stretched into horizontal abduction as far as tolerated without pain immediately decreased pectoralis minor

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stiffness, which was further decreased after three repetitions of 30s of SS⁶³. Additionally, pectoralis minor stiffness decreases after 10x30s of pectoral SS at 150° of abduction at the same intensity⁶². Interestingly, pectoralis minor stiffness was significantly different between PRE and POST (mean difference = 0.214, SD = 0.095, $p = 0.049$, 95% CI: 0.17 to 0.410 m/s, $\eta_p^2 = 0.159$, $\beta = 0.514$); however, only the 110% SS approached significance with a decrease of 0.283 m/s ($p = 0.078$; -10%) compared to 0.144 m/s ($p = 0.255$; -5%) following the 120% SS. This is a curious finding, given that previous work demonstrated a significant decrease in pectoralis minor stiffness at the greatest intensity tolerated by participants, even greater than our 120% intensity, after only 30s of SS⁶³. This might suggest that our SS intensities may be insufficient to elicit changes in stiffness. The shoulder abduction angle at which the pectoral stretch was implemented differed between the current study and previous reports (90° compared to 135° or 150°, respectively)^{62,63}, suggesting that pectoral SS at greater angles of shoulder abduction may reduce pectoralis minor stiffness to a greater extent than at 90°. However, the choice to employ a pectoral SS at 90° of abduction in the current study was two-fold: first, this position (and 150° of abduction) was the most effective at lengthening pectoralis minor in vivo^{49,50}; and second, this is a commonly prescribed pectoral SS position¹⁵¹. Moreover, while the ultrasound probe location was similar to that of the current study, Umehara et al. measured pectoralis minor stiffness in a more lengthened position with the participants' shoulder abducted and internally rotated 90°^{62,63}. Given that arm elevation elongates pectoralis minor¹⁶³, Umehara's measurement was taken in a lengthened position whereas the current study measured stiffness with the participant's arm at rest (i.e., with arms by their side). To the authors' knowledge, no studies to-date have examined pectoralis minor's slack angle or degree of lengthening through ranges of abduction; however, small increases (0.8-1.8%) in pectoralis minor length are observed up to 30° of flexion and

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scaption, and large increases (4.0-25.5%) are observed after that¹⁶³. As such, it would be reasonable to suspect that the slack angle exists somewhere between rest (the measurement position in the current study) and at 90° of abduction (the measurement position in Umehara's study). It is possible that changes in stiffness may only be captured past a muscle's slack angle, when stiffness would begin to increase. Moreover, stretching may have changed the muscle's slack angle, which may not necessarily result in changes in stiffness at rest as measured in the current study. Future work should examine pectoral slack angles and changes that occur in response to static stretching. Additionally, Umehara et al. used kilopascals (kPa) as their stiffness measurement, which is calculated from shear wave velocity and is dependent on a variety of assumptions that are frequently violated and thus, less consistent¹⁶⁴. Future work should standardize the stiffness measurement of pectoralis minor for optimal comparison between studies, including the position it is measured in and use shear wave velocity not kilopascals (m/s or kPa).

Given the pectoralis minor's influence on shoulder posture, we anticipated that changes in FSP would correspond to changes in pectoralis minor stiffness. Specifically, given that FSP decreased to a greater extent following the 120% SS, we expected pectoralis minor stiffness to be reduced to a greater extent following the 120% SS than the 110% SS. Contrarily, following the 110% SS, pectoralis minor stiffness approached significance ($p = 0.078$; -10%) compared to the 120% SS ($p = 0.255$; -5%). A post-hoc Pearson Correlation was conducted to explore the relationship between change in FSP and pectoralis minor stiffness. There was a very weak insignificant, negative relationship between change in FSP and change in pectoralis minor stiffness for 110% ($p = 0.368$, $r = -0.192$), and a weak insignificant negative relationship for 120% ($p = 0.144$, $r = -0.308$). This suggests that decreasing pectoralis minor stiffness/velocity

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does not have a major impact on FSP and that other mechanisms contribute to FSP. For example, pectoralis major excitation is increased in those with FSP compared to those without¹⁶⁵, but little research has been conducted on its role in FSP, or the muscle's response to SS in general¹⁶⁶. One study did report no change in pectoralis major stiffness before and after a three day/week, seven week pectoral SS protocol¹⁶⁶. While the current study defined the immediate change in pectoralis major stiffness following SS, these findings support those of the current study and suggest that neither acute nor chronic pectoral SS alters pectoralis major stiffness.

Lastly, contrary to the hypothesis, we did not observe a change in haROM following either SS. This contradicts the vast majority of the literature demonstrating that a single SS bout at high^{12,31,38,41} and low intensities^{38,41} acutely increases ROM. It is plausible that the intensity of stretching interventions used in the current work were not intense enough to elicit changes in ROM following stretching. Indeed, on average, participants reported their perceived stretch intensity as 20/100 and 31/100 for the 110% and 120% SS, respectively. These correspond to slight and slight to moderate on the stretch intensity scale used in the current study.

Recent evidence suggests ROM increases acutely with stretching regardless of stretch intensity^{3,5}, which may be attributed to neural adaptations (e.g., increased stretch tolerance) or decreased muscular or tendon stiffness⁶. While the current study observed a decrease in pectoralis minor stiffness pre-post, this did not coincide with increases in ROM. Few studies have examined the acute impact of pectoral stretching on ROM, and focus primarily on chronic SS (i.e., multiple sessions). For example, pectoral SS for three days/week for seven weeks increased shoulder extension ROM¹⁶⁶, and four days/week for eight weeks increased shoulder rotation ROM¹⁶⁷. These authors have cited mechanisms such as increased stretch tolerance⁶ and changes in muscle morphology¹⁶⁸ as contributors to increased ROM. For example, fascicle

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length has been found to increase after chronic SS, but only under high SS volumes and intensities¹⁶⁸. As such, the mechanisms leading to acute increases in ROM are largely unknown, but are speculated to be a result of changes in stiffness and stretch tolerance due to neuromuscular changes⁵. Future research should determine these mechanisms, and their response to various stretching dose parameters.

There are limitations to the current study. First, we included younger adult participants with asymptomatic shoulders; thus, these findings may not be generalized to other age groups or individuals with shoulder pathology. Secondly, most literature measures changes in ROM at the participant's POD, and the gravity used during haROM measurements in the current study did not apply a force adequate to take participants to their POD, which may be why we did not see changes in ROM. Third, we did not monitor, or control for, female menstrual cycles, which may have impacted their response to SS, given that hormonal fluctuation during the menstrual cycle alters ROM and stiffness¹⁶⁹. Lastly, stretch intensity is difficult to accurately manipulate given that it is subjective. Furthermore, no correlation was found between passive muscle tension and subjective pain¹⁷⁰, demonstrating that an individual's stretch perception may not indicate lengthening of the target muscle. As such, it is possible that our measured bROM, where participants reported their first sensation of a stretch, was interpreted differently by various participants and may not have adequately lengthened the pectorals. Thus, the 110% and 120% SS may not have been as intense as anticipated and were likely inconsistent across participants and interventions. For example, the mean perceived intensity score for the 110% and 120% intensities were 20 and 31 out of 100, respectively, demonstrating that our high-intensity SS averaged only a slight to moderate stretch intensity, according to our VAS. This may be why we did not observe changes in many of our outcome variables.

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Conclusion

In conclusion, three, 30s static stretches at 110% and 120% from baseline ROM reduces FSP and pectoralis minor stiffness, but does not affect PML, haROM, or pectoralis major stiffness. While stretching at 120% reduces FSP greater, 110% reduces pectoralis minor stiffness more than 120%, demonstrating that higher and lower SS intensities have differing benefits.

Chapter 4: Acute impact of moderate and maximal stretch intensities on pectoral muscle stiffness, length, forward shoulder posture, and range of motion, and the relationship between muscle stiffness and stretch intensity.

Author Note & Contribution

Data and text from the following manuscript appears in:

Bohunicky, S., Fulawka, K., Franklin, R., Peters, M., & Scribbans, T. D. (2025) Acute impact of moderate and maximal stretch intensities on pectoral muscle stiffness, length, forward shoulder posture, and range of motion, and the relationship between muscle stiffness and stretch intensity. *SportRxiv*. <https://doi.org/10.51224/SRXIV.653>

Sarah Bohunicky conceptualized, designed, piloted methods, collected data, analyzed data, interpreted data, and drafted the manuscript.

Abstract

Static stretching (SS) acutely increases range of motion (ROM) due to several purported mechanisms including an increase in mechanical muscle length due to reduced stiffness or increases in stretch tolerance. However, few studies have investigated acute changes following upper extremity SS, especially in response to different stretch intensities. In the shoulder, it is unknown if different pectoral SS intensities affect stiffness, ROM, pectoral muscle length (PML) and forward shoulder posture (FSP) following SS. Thirty participants ($n=30$, 15 female, 26 ± 4 years, 173 ± 10 cm, 73 ± 16 kg) completed two experimental sessions where three pectoral SS interventions were conducted: a control condition (CON), and moderate and maximal SS intensities. Pectoralis major and minor stiffness (via shear wave elastography), PML, FSP, and horizontal abduction ROM (haROM) were measured before (PRE) and after (POST) each intervention. The control condition was completed first where participants rested for 150s, followed by either moderate or maximal intensity SS. The remaining intervention was conducted in the second session. A standing doorway pectoral SS was completed, and moderate and maximal intensity were prescribed using a stretch intensity scale ranging from 0-150 where participants stretched at a 60 on the scale for the moderate intensity, and 100 (the greatest intensity achieved without pain) for the maximal. Each SS intervention was held for three sets of 30s with 30s of rest in between, and the same angle was used for all three repetitions. There was a significant 2-way interaction for haROM, $F(2,58)=58.195$, $p<.001$, $\eta_p^2=.667$. haROM decreased from PRE to POST CON (PRE= 105 ± 5 , POST= 103 ± 5 degrees; $p<.001$) but increased following moderate (PRE= 104 ± 5 , POST= 107 ± 6 degrees; $p<.001$) and maximal (PRE= 103 ± 5 , POST= 108 ± 5 degrees; $p<.001$). There were no significant interactions for FSP, PML, or pectoralis major or minor stiffness. The main effect of time was significant for PML and FSP; PML increased from PRE (14.3 ± 0.2 cm) to POST (14.4 ± 0.2 cm; $p=.020$) and FSP decreased from PRE (11 ± 0.3 cm) to POST (10.8 ± 0.3 cm); $p<.001$). A single bout of pectoral SS at moderate and maximal intensity increases haROM, with slightly greater gains with maximal intensity. These changes appear to be independent from changes in pectoral stiffness and PML, and do not result in changes in FSP, suggesting that mechanisms other than muscle stiffness may contribute to changes in ROM.

Introduction

Static stretching (SS) is well established to acutely increase range of motion (ROM)^{3,171}. However, the mechanisms underlying these alterations in ROM remain limited⁴. Increases in ROM are commonly attributed to two primary mechanisms: increased mechanical muscle length via reduced muscle stiffness^{110,112,153,172} and increased stretch tolerance^{6,84}. While reductions in muscle stiffness are thought to directly increase ROM, some studies report increased ROM without concomitant decreases in stiffness³⁹, suggesting stretch tolerance may play a more substantial role in increasing ROM.

Stretch tolerance refers to an individual's ability to endure muscle elongation, likely due to altered pain perception following SS^{7,8,173}. It is often assessed using subjective pain/intensity ratings³³ or objective measures such as the peak passive joint moment at maximal ROM^{4,153}. Though research has suggested that increases to stretch tolerance contribute to increases in ROM resulting from SS^{6,84}, how stretch tolerance responds to different SS intensities remains unclear.

SS intensity is a central exercise prescription variable based on subjective discomfort that may impact the resulting effects of SS. Traditionally, SS intensity is prescribed below a painful threshold yet still providing some level of discomfort¹⁷⁴. Current evidence on the effect of SS intensity on ROM is inconclusive, with some studies reporting greater gains with higher intensity SS^{12,25,30–34}, while others find similar improvements compared to lower intensities^{35–41}. Although high intensity SS may enhance both ROM and stretch tolerance^{30,31}, its effect on muscle stiffness remains uncertain. Given that SS is clinically prescribed based on subjective patient discomfort, it is imperative to understand how patient reported intensity influences outcomes such as ROM, stiffness, and stretch tolerance.

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Most existing research prescribes SS intensity using participants' point of discomfort (POD) or maximal tolerable stretch (MTS). High intensity SS (100-120% MTS or POD) decreases hamstrings^{12,25,33} and gastrocnemius³² stiffness relative to low intensity (at POD, or between 50-80% MTS or POD). However, other studies report that low (1-2 out of 10 on a numerical rating scale [NRS]) and high intensity (9-10 out of 10 on NRS) SS increase ROM without changes in stiffness, suggesting that stretch tolerance may account for increased ROM³⁹. In contrast, high intensity SS (at MTS) for 10, 15, or 20s increased ROM and reduced stiffness¹⁷⁵. These inconsistent findings, often confounded by simultaneous modification of two dose parameters (e.g., stretch intensity and duration)^{30-32,38}, make it challenging to isolate the effect of SS intensity.

Moreover, the impact of different SS intensities in the upper limb is unknown as current research has been conducted exclusively on lower-limb musculature. SS is commonly used to lengthen the pectoral muscles, particularly pectoralis minor, which is associated with increased forward shoulder posture (FSP)^{54,55}. FSP alters scapular kinematics⁵⁵ and increases the risk of developing shoulder pathology⁴⁸⁻⁵³. A single 30s pectoral SS at MTS can acutely reduce pectoralis minor stiffness⁶³; however, it is unclear whether pectoral SS impacts pectoralis major stiffness, pectoralis minor length, FSP, or ROM, and whether these responses differ by SS intensity. Thus, the primary objectives of the current study were to determine the impact of an acute bout (3x30s) of pectoral SS at moderate and maximal intensity on: (1) a) pectoralis major and minor stiffness, b) forward shoulder posture (FSP), c) pectoralis minor length (PML), and d) maximal horizontal abduction range of motion (haROM), compared to a control (CON); and (2) the relationship between the change in patient reported stretch intensities during SS and PRE-POST changes in variables a-d.

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Additionally, stiffness can reflect the magnitude of tissue elongation during SS, with increased stiffness resulting from greater muscle tension or stretch. While pectoralis minor is effectively elongated following pectoral SS at POD⁵⁰, it is unclear whether elongation (as inferred by increased stiffness) differs during different SS intensities. Given that intensity is primarily guided by patient sensation, it is critical to quantify the magnitude of elongation at different perceived intensities. Thus, objective (3) was to determine whether the magnitude of elongation (i.e., stiffness) of pectoralis major and minor differ during moderate and maximal SS intensities.

Previous work has shown reductions in pectoralis minor stiffness following SS at POD⁶³. Our group previously demonstrated that low and moderate intensity pectoral SS decreases both pectoralis minor stiffness and FSP, with greater reductions in FSP following moderate intensity (Chapter 3). However, neither intensity affected pectoralis major stiffness or PML. As such, it is hypothesized that (1) both SS intensities would decrease pectoralis minor stiffness and FSP compared to a control, with maximal intensity decreasing FSP significantly more. Moreover, it was hypothesized that PML and pectoralis major stiffness would remain unchanged across all interventions, and that haROM would increase after moderate and maximal SS intensities, as various intensities increase ROM³. It was further hypothesized that (2) changes in patient reported intensity would be greater following maximal stretching, and would be positively correlated with changes in FSP, but not with changes in muscle stiffness, PML, or haROM. Finally, it was hypothesized that (3) pectoralis major and minor stiffness would be higher during maximal SS intensity, indicating greater elongation.

Methods

Experimental Overview

A repeated measures crossover design was conducted where participants attended two experimental sessions at least 72 hours apart. During each session, participants were randomly allocated to receive one of two different intensities (moderate and maximal) of a standing self-administered pectoral SS to their dominant arm. A control condition was completed at the start of each participant's first session. Moderate and maximal SS intensity was determined using a stretch intensity scale (SIS)¹⁷⁶. Pectoralis major and minor stiffness, FSP, PML, and haROM were measured before (PRE) and immediately after (POST) each intervention. All experimental procedures were approved by the University of Manitoba Research Ethics Board 1 and were conducted in accordance with the Declaration of Helsinki¹⁷⁷.

Participants

A priori sample size calculation ($\alpha=.05$, $\beta=.8$) determined 27 participants would be required to detect an interaction between intervention (control, moderate and maximal) and time (PRE and POST) at a moderate to large effect ($\eta^2=.1$). A total of 37 individuals expressed interest and were assessed for the following inclusion criteria: between 18-40 years old, no recent history (< 6 months) of pain, injury, or orthopedic disorders to either arm; no diagnosed neurological or musculoskeletal disorders; no contraindications to the use of ultrasound (i.e., pacemaker, allergy to transmission gel); no history (< 5 years) of participation in sport/activities requiring extreme upper body ranges of motion (e.g., gymnastics, yoga, baseball pitcher); not currently pregnant or breast feeding; and ≤ 5 Beighton's score for joint hypermobility.

Thirty ($n=30$, 15 female, 26 ± 4 years, 173.0 ± 9.9 cm, 73.2 ± 15.6 kg) participants were eligible and completed both experimental sessions (**Figure 10**). Eligible participants handedness

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was determined using the Modified Edinburgh Handedness Index¹⁷⁸. Prior to each session, participants were asked to refrain from upper body resistance training for 48 hours and cannabis and alcohol consumption for 24 hours.

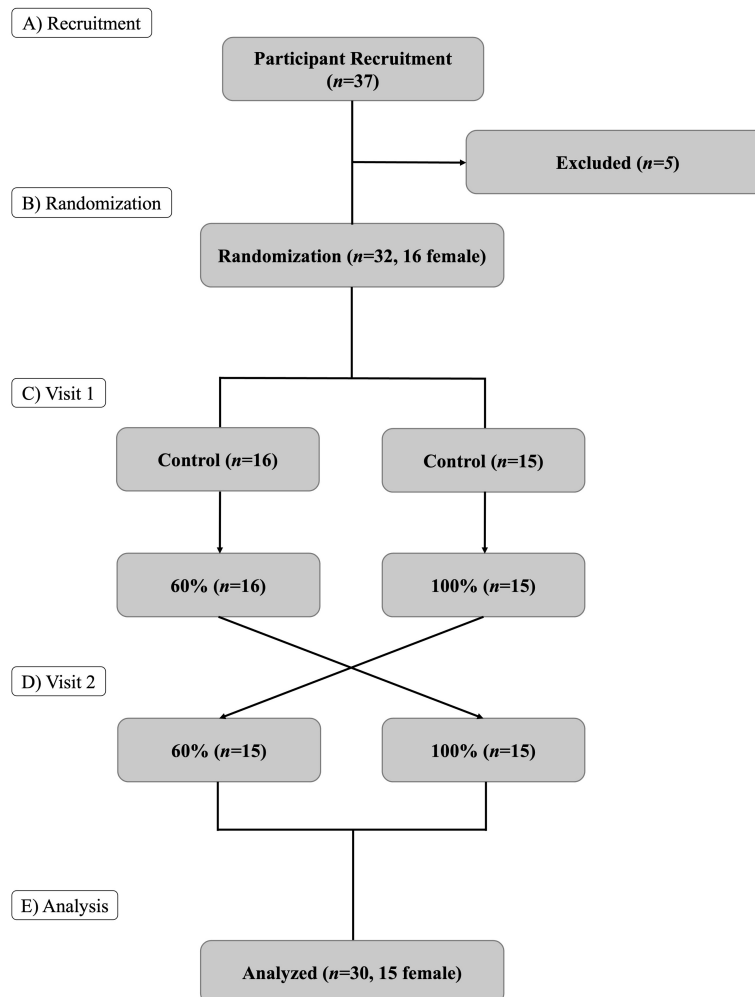


Figure 10. Consort Diagram

A) Thirty-seven participants were recruited and screened for eligibility; five were excluded as they did not meet eligibility criteria. B) Thirty-two participants were eligible and had their stretch intensity order randomized (60% or 100%). C) During the first visit, sixteen participants (n=16) stretched at a moderate (60%) intensity and fifteen (n=15) stretched at a maximum (100%) intensity. One participant was released and did not complete Visit 2 due to poor ultrasound image quality at the PRE-control measurement phase. D) Thirty participants returned for Visit 2 where they received the opposite stretch intensity as visit 1. One participant did not complete Visit 2 as they sustained an injury between visits. E) All data collected from 30 participants for every outcome variable was analyzed.

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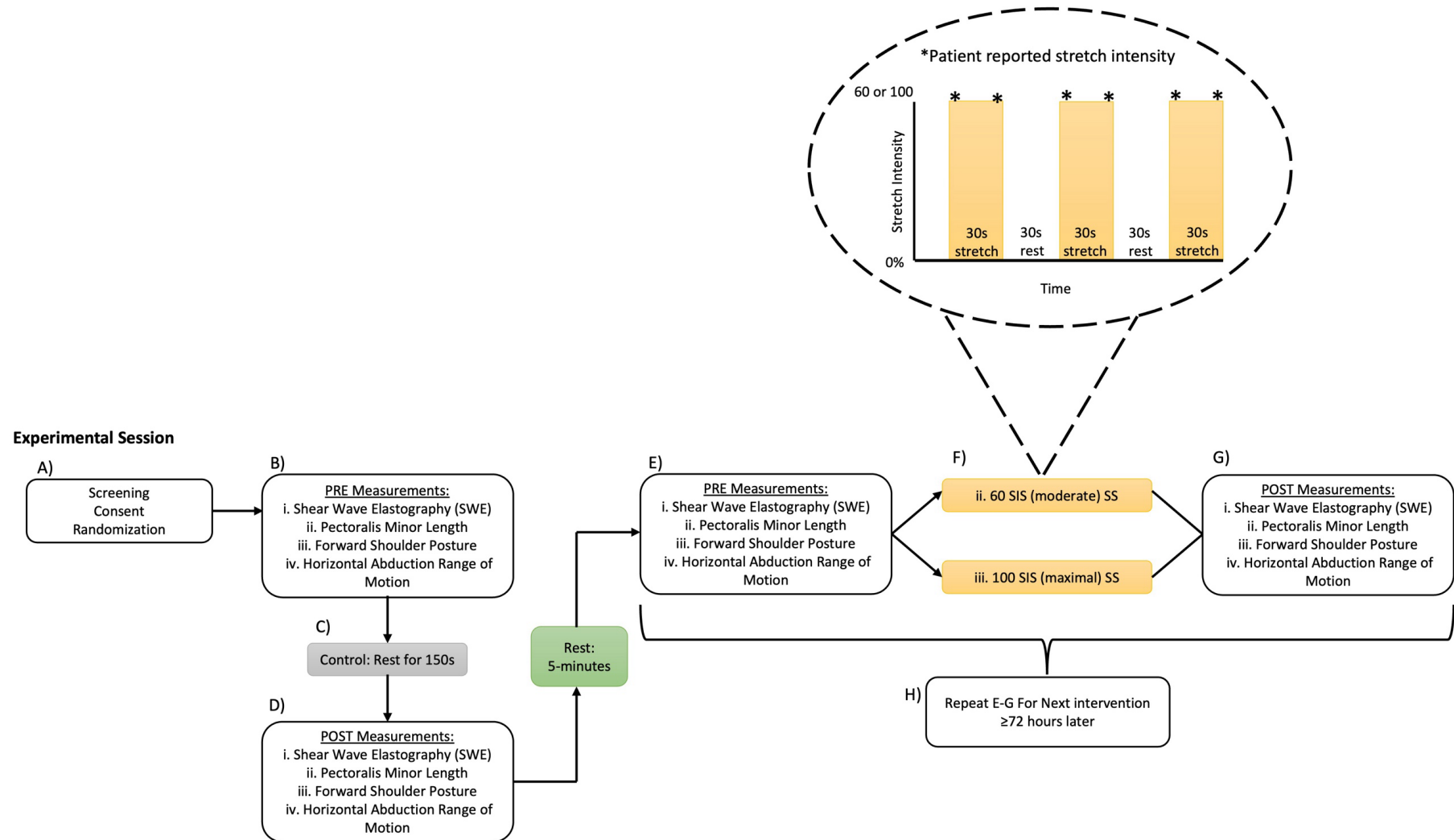


Figure 11. Timeline Schematic of Experimental Protocol

A) Participants were screened and if confirmed eligible, provided their informed consent. They were randomly assigned which intervention they received during each visit. B) Prior to completing the pre-control measurements, participants' age, sex assigned at birth, height, and weight were collected. Pre-control measurements (pectoralis major and minor stiffness via shear wave elastography

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*(SWE), pectoralis minor length, forward shoulder posture (FSP), and maximum shoulder horizontal abduction range of motion (haROM)) were taken. C) The control condition had participants rest for 150s after pre-control measurements. D) Post-control measurements followed the same protocol as B. E) After a 5-minute rest to reduce cross-over effects from the first set of measurements, participants then completed pre-intervention measurements in the same manner as B. F) Participants then completed their randomized stretch intensity intervention at either 60 (moderate) or 100 (maximal) on the stretch intensity scale for three sets of 30s with 30s intervals of rest between stretching bouts. At the beginning and end of each bout (denoted by the *), participants rated their perceived intensity of the stretch. G) Post-intervention measurements were conducted in the same manner as B and E. H) Participants returned for a second session a minimum of 72 hours after their first visit and completed phases E-G for the other stretch intensity on the same arm.*

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Procedures

During the first session, once informed consent was obtained, SS intensity (moderate and maximal) order was randomized according to a randomization schedule. Participants' reported their height, weight, age, and sex assigned at birth. Pectoralis major and minor stiffness (shear wave velocity, SWV), FSP, PML, and haROM was measured before (PRE) and immediately after (POST) each intervention on the dominant side, including a control condition completed at the beginning of the first experimental session (**Figure 11**).

Shear Wave Elastography

Pectoralis major and minor stiffness were captured using SWE (Logiq E10s, GE, Mississauga, Ontario, Canada; **Figure 12**) using a 2-10 MHz transducer (XDclear linear probe, GE, Mississauga, Ontario, Canada). A single, trained researcher applied transmission gel to couple the transducer with the skin approximately 1-inch below the clavicle and lateral to the sternal edge. Transducer location for each muscle was determined using B-mode ultrasound, where muscle fibres were aligned longitudinally so they appear parallel. These locations were marked with permanent marker to ensure consistency between measurements within a given session.

Shear wave mode produced a colour-coded map of shear wave velocity (SWV) within the region of interest (ROI). The ROI was maximized for each muscle within the myofascial borders that still produced a clear SWV map free from artifact (none or very little monochromatic artifact). With participants seated in a 30° reclined position, one, ~10s elastogram was acquired for each muscle. This position has been previously demonstrated to keep pectoral muscles relaxed and displace breast tissue, as both contraction and breast tissue may influence stiffness measurements (Chapter 3). Participants took a deep breath in, exhaled for a count of 3s, and then

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held their breath for ~10s as the elastogram was taken to limit movement⁶³. Three high-quality images with the least amount of artifact were saved from each elastogram and used for analysis (Figure 12C and D).

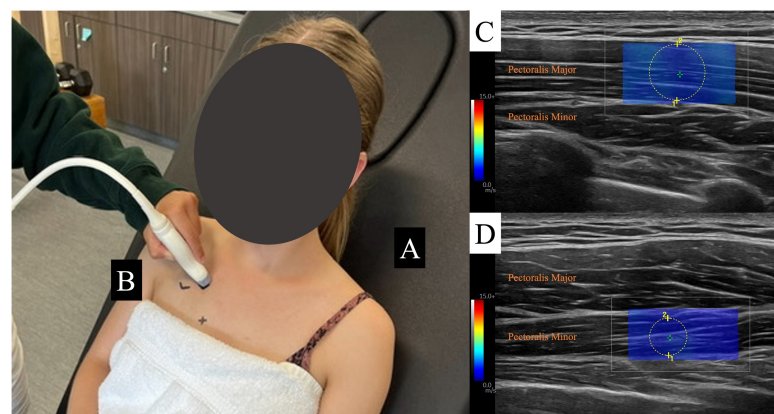


Figure 12. Shear Wave Elastography

A) Shear wave elastography (SWE) was used to measure the stiffness of pectoralis major and minor. Measurements were taken with participants sitting on a plinth reclined at 30 degrees. A bolster was placed under their knees, and they were instructed to relax their arms at their sides during measurements. B) The 2-10 MHz transducer was held parallel to the muscle fibres of the muscle being measured, approximately 1 inch lateral from the sternal edge and 1 inch inferior to the clavicle. These positions were determined with the ultrasound in B-mode and marked on the skin with a black marker. Images were taken in shear wave mode for pectoralis major (C) and minor (D), in which the region of interest (ROI) was superimposed over the B-mode image over the largest area possible within the muscle. SWE measurements produce a colour-coded map with colours ranging from dark blue to red. Images were taken while participants held their breath; they were instructed to inhale, exhale for 3 seconds, then hold at the bottom of their breath for approximately 10 seconds. Approximately 8-10 images were taken, and 3 high-quality images with little blank space (i.e., artifact) were saved for analysis.

Forward Shoulder Posture (FSP)

FSP was measured using the double-square method, which has moderate to good reliability compared to gold standard radiography¹⁷⁹. Standing with the back against a wall in a relaxed posture (i.e., not correcting their posture), a modified combination square measured the distance from the wall to the marked acromion process. This measurement was conducted three

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times where the average was used for analysis. Between each measurement, participants were instructed to step forward and shake their arms to reset their posture.

Pectoralis Minor Length

PML was measured while the participants laid supine. Participants had their coracoid process and anterior-inferior edge of the 4th rib 1 finger-width lateral to the sternum landmarked and marked with permanent marker. A flexible measuring tape was used to measure this distance 3 times, where the average of the 3 measurements was used for analysis.

Horizontal Abduction Range of Motion

Horizontal abduction range of motion (haROM) was measured in a standing position where participants horizontally abducted their shoulder against an upright post that served as a stretching apparatus (**Figure 13**). Standing next to the apparatus, participants flexed and abducted their elbow and shoulder to 90°, respectively, placing their forearm against the apparatus. They were directed to take steps forward to horizontally abduct their shoulder until they reached a 100 on a stretch intensity scale (SIS). The SIS is a validated tool for prescribing stretch intensity¹⁷⁶ (**Figure 14**) and ranges from 0 (no stretch) to 150 (maximal achievable range of motion with pain) and uses 100 as the maximal range of motion achievable before pain onset¹⁷⁶. Once the participant reported a 100, a researcher stationed behind them used a goniometer to measure the angle of horizontal abduction. The same researcher conducted all goniometric measurements, as goniometry has high intra-rater reliability¹⁵⁷. After the haROM angle was measured, the participant returned to the starting position. This was repeated for a total of three measurements where the average was used for analysis.

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Figure 13. haROM and stretching position

A) Participants stood next to the stretching apparatus with their shoulder abducted and elbow flexed to 90°, respectively, resting their forearm against the vertical post. B) A researcher was positioned behind and above the participant holding a goniometer. The researcher marked the participant's lateral arm (as indicated by the arrow) for a consistent measurement location for the goniometer's arm.

The participant was instructed to take steps forward until they felt a stretch of 100 on the stretch intensity scale (SIS). The goniometer's arm was moved to align with the mark on the participant's arm and the haROM angle was recorded. This was repeated three times, and the average was used for analysis.

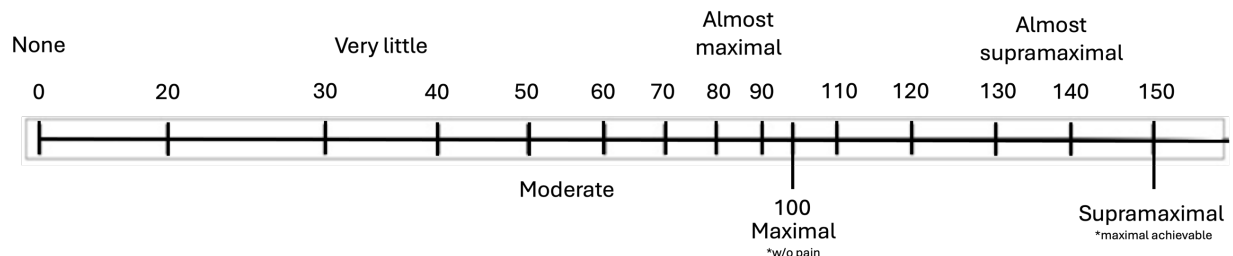


Figure 14. Stretch Intensity Scale

Adapted from Freitas et al. (2015)¹⁷⁶

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Control Condition

At the first session after PRE measurements, participants then rested for 150s in a seated position on a plinth for a control condition. POST measurements were taken after the control condition. Following a 5-minute rest period, PRE measurements were taken again prior to completing the first SS intervention.

Stretch Intervention

After 5-minutes of rest and a second set of PRE-measurements, participants completed three repetitions of a moderate or maximal intensity 30s SS at 60 or 100 on the SIS, respectively, in the same position as haROM (**Figure 13**). For the moderate intensity, the participants horizontally abducted their arm until they reported a 60 on the SIS. This angle was measured with the goniometer and recorded. For the maximal intensity, the average haROM angle from the PRE measurements was used ($n=6$ participants reported their perceived intensity was less than 100 at the start of their first stretching bout; range: 70-98). Once the prescribed intensity was reached, that position was held for the first 30s. After 30s, the participant released the stretch and stood with their arms by their side for 30s. This was repeated for a total of 3 SS bouts of 30s, and the angle for each bout of stretching for a given intervention was held constant.

At the start (~1s) and end (~29s) of each stretching bout, participants reported their perceived stretch intensity using the SIS to capture changes in perceived stretch intensity.

Experimental Session Two

Participants attended a second experimental session where the same procedures were followed except participants received the second SS intensity and no control condition was collected. Experimental sessions were a minimum of 72-hours apart (**Figure 11E-H**).

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Magnitude of Elongation During Stretching

To determine the magnitude of elongation of the pectorals during the two SS intensities, a subset of participants ($n=10$, 5 female; 26 ± 4 years) attended an additional session where pectoralis major and minor stiffness were measured during both SS intensities in random order. Following the same methods of the two experimental sessions, participants first sat reclined on a plinth while a single, trained examiner located and marked the probe locations of the pectoralis major and minor of the dominant limb. Participants then assumed the starting position of the SS intervention against the stretching apparatus (90° of elbow flexion and shoulder abduction, forearm against the post). The probe was placed over the marked location for pectoralis major, and participants then stretched to either a 60 or 100 on the SIS, based on the randomization order. This position was held until three high-quality images were taken. A goniometer was used to measure the degree of haROM as described previously. Before measuring pectoralis minor stiffness at the same angle, participants were asked to rest for two-minutes with their arms by their side. After pectoralis minor stiffness elastograms were taken, a five-minute rest was provided, and the same procedure was followed for the second stretch intensity.

Data Processing

Velocity (m/s) of shear wave propagation generated by the system software was used to quantify stiffness. An investigator blinded to SS intensity closely examined all three images extracted from each elastogram for artifact and measured the largest possible area within the ROI. The diameter and location of the measurement within the ROI was kept consistent between images for the same timepoint for a given participant. The average of the three SWV measurements from each elastogram were used for analysis.

Statistical Analysis

Statistical analyses were performed using SPSS (v25, Chicago, IL, USA) by a blinded investigator. The Shapiro-Wilk test was used to determine normality where data were normally distributed if $p > .05$. Outliers were studentized residuals greater than ± 3 standard deviations.

To achieve objective 1, a 3x2 (intensity*time) repeated measures ANOVA was conducted on all dependent variables. Data that were not normally distributed underwent transformation. ANOVA post-hoc pairwise comparisons were conducted on significant interactions using a Bonferroni correction. Small, medium, and large effect sizes were interpreted as: $\eta p^2 = 0.01$, 0.06, and 0.14, respectively; $d = 0.2$, 0.5, 0.8, respectively¹⁸⁰. To achieve objective 2, Pearson Correlations were conducted on changes in perceived intensity (start of first stretch bout to end of third stretch bout) and PRE to POST changes of all dependent variables. Non-parametric Spearman rank-order alternative tests were conducted if data were not normally distributed for the correlation analyses. The change in patient reported stretch intensity was considered the difference from the beginning of the first stretching bout to the end of the last stretching bout. This was correlated to the PRE-POST difference for each dependent variable, calculated by subtracting PRE measures from POST measures. A negative change represents a decrease in a dependent variable. Pearson Correlation coefficients (r) were interpreted as: weak < 0.3 , moderate $= 0.3-0.5$, and strong > 0.5 . To achieve objective 3, paired samples t -tests were conducted on stiffness measurements during moderate and maximal SS intensities for both pectoralis major and minor. Data were considered statistically significant if $p < .05$. All data is presented as mean $\pm$ standard deviation unless otherwise stated.

Intrasession reliability of the dependent variables were determined by a two-way mixed model absolute agreement intraclass correlation using both PRE-measurements from the first

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experimental session. Intraclass correlations of ≤ 0.5 , $0.5-0.75$, $0.75-0.9$, and ≥ 0.9 indicated poor, moderate, good, and excellent reliability, respectively¹⁸¹.

Results

Intraclass correlations for haROM were good (Cronbach's $\alpha=.849$), and excellent for FSP ($\alpha=.930$), PML ($\alpha=.994$), pectoralis major ($\alpha=.978$) and minor stiffness ($\alpha=.934$).

For objective 1, outliers were found in haROM, pectoralis major SWV, and pectoralis minor SWV, and not normally distributed ($p<.05$). A log10, square root, and inverse transformation was applied to pectoralis major and minor SWV which did not correct distribution ($p<.05$). Thus, all analyses were conducted on non-transformed data. There was a significant 2-way intervention by time interaction for haROM, $F(2,58)=58.195$, $p<.001$, $\eta_p^2=.667$ (**Figure 15; Table 5**). Main effects of both treatment ($p=.029$) and time ($p<.001$) were significant. Pairwise comparisons revealed haROM significantly decreased following the CON intervention ($p<.001$, mean difference -2.6° , 95% CI -1.6° to -3.6°), while haROM significantly increased following moderate SS ($p<.001$, mean difference 2.7° , 95% CI 1.4° to 4.0°) and maximal SS ($p<.001$, mean difference 4.8° , 95% CI 3.7° to 5.9°).

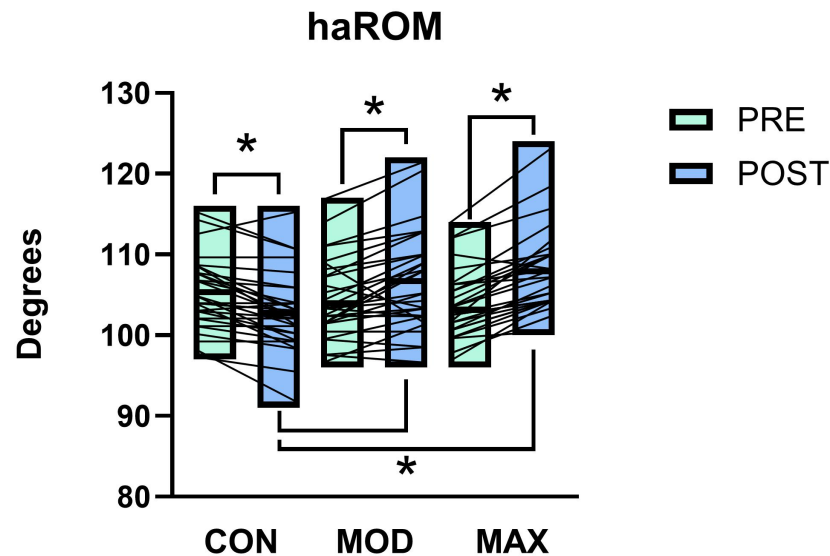


Figure 15. Horizontal Abduction (haROM) before (PRE) and after (POST) 3x30s of Pectoral Static Stretching at Moderate (60/100) and Maximal (100/100) Stretch Intensities.

n = 30, haROM = horizontal abduction range of motion

*denotes significant pairwise comparison

There was no significant interaction for pectoralis major stiffness, pectoralis minor stiffness, FSP, or PML (**Figure 16 & 17**). There was no significant intervention or time main effects for pectoralis major or pectoralis minor stiffness. There was a significant main effect of time on PML ($p=.020$, mean difference .07cm, 95% CI .01 to .12) and FSP ($p<.001$, mean difference -.17, 95% CI -.25 to -.09; **Figure 18**).

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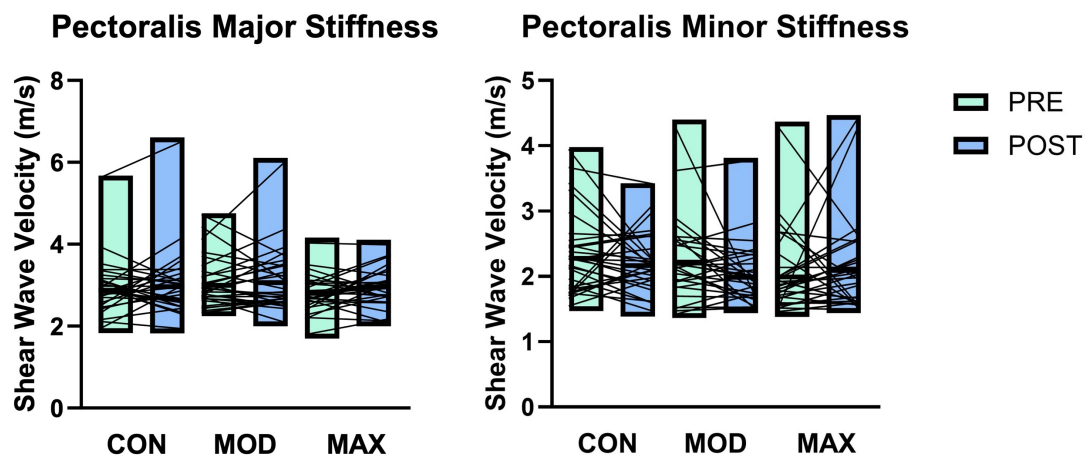


Figure 16. Pectoralis Major and Minor Stiffness before (PRE) and after (POST) 3x30s of Pectoral Static Stretching at Moderate (60/100) and Maximal (100/100) Stretch Intensities.

$n = 30$

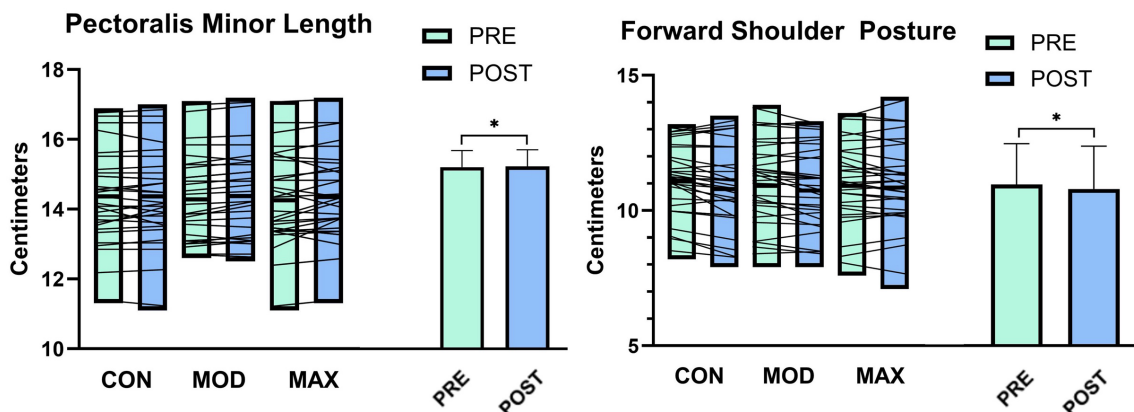


Figure 8. Pectoralis Minor Length (PML) and Forward Shoulder Posture (FSP) before (PRE) and after (POST) 3x30s of Pectoral Static Stretching at Moderate (60/100) and Maximal (100/100) Stretch Intensities.

$n = 30$; *denotes a main effect of time

For objective 2, data were not normally distributed for changes in pectoralis minor stiffness at moderate ($p < .001$) and maximal ($p = .005$) intensities. As such, a Spearman Rank correlation was conducted on the relationship between changes in perceived intensity and pectoralis minor stiffness. There was a significant correlation between changes in perceived

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intensity and haROM at maximal stretch intensities ($r=-.461, p=.010$), indicating that a decrease in perceived intensity from first to last stretch was moderately correlated with an increase in haROM (**Table 6**). Changes in perceived intensity were not significantly correlated with changes in any other variables at any stretch intensity.

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Table 5. Descriptive Statistics and Two-way (Intervention*Time) ANOVA Results

Variable	Control		Moderate		Maximal		<i>f</i>	<i>p</i>	η_p^2	β
	PRE	POST	PRE	POST	PRE	POST				
Pec Maj SWE (v/s)	2.87(.69)	2.96(.85)	2.99(.62)	3.06(.80)	2.80(.48)	2.96(.51)	.197	.821	.007	.079
Pec Min SWE (v/s)	2.28(.65)	2.17(.47)	2.21(.65)	1.99(.49)	1.99(.63)	2.09(.74)	1.3	.280	.043	.271
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
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
haROM (degrees)	105(5)	103(5)	104(5)	107(6)	103(5)	108(5)	58.195	<.001*	.667	1

60= moderate intensity SS; 100= maximal intensity SS; cm= centimeters; FSP= forward shoulder posture; haROM= horizontal abduction range of motion; m/s= velocity, meters per second; Pec= pectoralis; PML= pectoralis major length; POST= Post-SS measurement; PRE = Pre-SS measurement; SWE=shear wave elastography. * $p < .001$

Table 6. Correlations between Change in Perceived Intensity from First to Last Stretch and PRE-POST Changes in Stiffness, Pectoralis Minor Length, Forward Shoulder Posture, and Horizontal Abduction Range of Motion for Moderate and Maximal Stretch Intensities

Variable	Δ POST-PRE moderate	Correlation with Δ perceived intensity at moderate		Δ POST-PRE maximal	Correlation with Δ perceived intensity at maximal	
		<i>p</i>	<i>r</i>		<i>p</i>	<i>r</i>
Pec Maj SWE (m/s)	.07 (.66)	.779	-.053	.16 (.53)	.274	-.206
Pec Min SWE (m/s)[⊥]	-.23 (.64)	.540	.116	.1 (.92)	.594	-.101
PML (cm)	.09 (.16)	.156	.265	.1 (.34)	.810	.046
FSP (cm)	-.2 (.4)	.119	.291	-.1 (.4)	.08	.325
haROM (degrees)	3 (4)	.884	-.028	5 (0)	.010*	-.461
Perceived Intensity	9 (15)	-	-	17 (17)	-	-

60= moderate intensity SS; 100= maximal intensity SS; cm= centimeters; FSP= forward shoulder posture; haROM= horizontal abduction range of motion; m/s= velocity, meters per second; Pec= pectoralis; PML= pectoralis major length; POST= Post-SS measurement; PRE = Pre-SS measurement; *r*= Pearson *r* or Spearman rho; SWE=shear wave elastography; Δ = change score;

[⊥]= spearman rank correlation; * $p < .05$ (Negative values represent a decrease from PRE to POST)

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A paired samples t -tests exploring the change of perceived intensity demonstrated a significant difference between moderate (decrease of 9 ± 15) and maximal (decrease of 17 ± 17) stretch intensities, $t(1,29) = -3.52$, $p = .001$, $d = -.643$.

For objective 3, there were no outliers and data was normally distributed. Stiffness did not differ between stretching at moderate or maximal intensities on the SIS for pectoralis major, $t(1,9) = .0694$, $p = .946$, $d = .0219$, or pectoralis minor, $t(1,9) = -.619$, $p = .551$, $d = -.196$ (**Table 7**).

Table 7: Pectoralis Major and Minor Stiffness During Moderate and Maximal Stretch Intensity

Variable	Mean (Standard Deviation)		t	p	d
	MOD	MAX			
Pec Maj SWE (m/s)	7.46 (1.55)	7.42 (1.55)	0.0694	0.946	0.0219
Pec Min SWE (m/s)	3.8 (1.0)	4.0 (0.68)	-0.619	0.551	-0.196

m/s= velocity, meters per second; Maj= major; Min= minor; MOD= moderate intensity SS; MAX= maximal intensity SS; Pec= pectoralis; SWE=shear wave elastography

Discussion

This study had three objectives: 1) determine the impact of two intensities of acute pectoral SS to asymptomatic shoulders on pectoral stiffness, FSP, PML, and haROM, compared to a control condition; 2) examine the relationship between changes in patient reported stretch intensity during stretching and changes in these same variables from PRE to POST; and 3) assess the difference in stiffness, and thus elongation, of pectoralis major and minor during MOD and MAX SS intensities. The major findings of the current work are that: 1) both SS intensities increased haROM compared to the control; however, no significant changes were found in pectoral stiffness, FSP, or PML compared to a control; 2) There was a significant moderate negative correlation between changes in perceived intensity and changes in haROM at maximal

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SS intensity. No significant correlations were found for haROM at moderate SS, or between changes in perceived intensity and changes in pectoral stiffness, FSP, or PML; and 3) Opposing our hypothesis, there were no significant differences between the shear wave velocity at moderate or maximal for either muscle.

Horizontal abduction ROM increased similarly following moderate and maximal pectoral SS, which is consistent with prior work showing that both high and low SS intensities increase ROM similarly^{3,171}. To the authors' knowledge, this is one of the first studies to investigate SS intensity in the upper body, likely due to challenges using standardized equipment such as isokinetic dynamometers with the upper limb. Our group previously examined a 3x30s therapist-administered pectoral SS at low and moderate intensity (110% and 120% of a baseline ROM) but found no changes in haROM (Chapter 3). Methodological limitations, including passive haROM measurement without external overpressure, use of a non-validated stretch intensity scale, and insufficient stretch intensity, may explain the absence of effect. In contrast, the present study employed more rigorous methods, including a maximal pain-free ROM measurement and more intense SS protocols prescribed by a validated intensity scale¹⁷⁶, resulting in significant increases in haROM. These findings align with the broader literature demonstrating that different SS intensities elicit comparable improvements in ROM^{3,171}.

While our findings support prior evidence that ROM can increase following SS regardless of intensity, they also suggest a potential dose-response effect between stretch intensity and ROM, with average haROM increasing $\sim 2^\circ$ more following maximal. Prior work has shown that the magnitude of ROM gains are larger with higher SS intensities^{12,25,30-33}. A potential mechanism is enhanced stretch tolerance, reflected by a larger reduction in perceived intensity after maximal SS (-17/100) compared to moderate SS (-9/100). Although the

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mechanism underlying increased tolerance remains unclear¹⁸², proposed contributors include altered pain perception, neural adaptations, greater willingness to tolerate discomfort, and expectancy effects^{183,184}. Our results align with a recent systematic review stating that evidence is inconclusive³; however, methodology heterogeneity, such as varied intensity prescriptions (e.g., point of discomfort, point of pain, point before discomfort, etc) and manipulation of both stretch intensity and duration, complicates comparisons of SS intensity across studies. Additionally, prior research has largely involved male participants (~82%), limiting generalizability relative to our balanced sample (50% female). Evidence regarding sex-based differences in ROM adaptations to acute SS is mixed, with some studies reporting differences¹⁸⁵ and others finding none^{186,187}. While female flexibility is influenced by hormonal fluctuations across the menstrual cycle¹⁶⁹, muscle stiffness, tolerance to muscle stretch, and muscle slack length is not influenced by phases of the menstrual cycle¹⁸⁸. As such, it is unknown if female responsiveness to SS varies across the menstrual cycle. Because we did not control for cycle phase, such fluctuations could have contributed to variability in our results and may partly explain the null findings reported in previous studies. A clearer intensity-response relationship may emerge if standardized methods of prescribing SS intensity are applied, intensity is isolated from other dose variables (e.g., duration), and sex-specific factors such as menstrual cycle timing are controlled. Future research should aim to disentangle these factors and clarify the mechanisms underlying stretch tolerance and ROM adaptation to better inform evidence-based stretching prescriptions.

Although both intensities produced meaningful and similar improvements in haROM, our findings may suggest a potential dose-response relationship, with maximal intensity eliciting slightly greater gains. This pattern may indicate that mechanistic variables such as stretch tolerance and stiffness may respond differently across intensities, contributing to greater ROM

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improvements at higher intensity. However, examination of individual changes scores (**Figure 15**) revealed considerable variability in responses, with some participants showing increases in ROM while others exhibited little change or even decreases. As such, it is evident that these ROM adaptations are not uniform and vary considerably between individuals, emphasizing the importance of accounting for inter-individual variability and baseline characteristics when prescribing interventions. Clinically, both moderate and maximal intensities can be effective for improving ROM, but maximal stretching may yield greater benefits. Therapists should therefore monitor individual responses and adjust prescriptions accordingly to optimize outcomes.

Muscle stiffness contributes to extensibility and, in turn, joint ROM. Reductions in stiffness are a purported mechanism underlying ROM gains, and have been reported following SS^{76,110,112,189,190}. In the present study, however, ROM increased without concomitant reductions in pectoral stiffness at either intensity. This contrasts previous work that reported decreased pectoralis minor stiffness after 30- and 90-seconds of horizontal abduction SS at 135° of humeral elevation with external rotation to maximal, pain-free ROM⁶³. Methodological differences may explain this discrepancy. Specifically, Umehara's stretch position (135° of humeral elevation with external rotation) may have imposed a more effective stretch on the pectoralis minor, compared to our stretch position of 90° of humeral elevation without added external rotation, leading to stiffness reductions. Secondly, Umehara assessed stiffness at 90° of arm elevation while the current work measured stiffness at rest. Assessing stiffness in a tensioned state (i.e., beyond slack length) may increase sensitivity to detect changes. Prior studies have shown stiffness reductions at various muscle lengths during SWE, including at rest¹⁹¹, and in both lengthened^{32,33,112} and shortened¹⁹² positions. While stiffness has been reported to not vary below slack length following SS¹⁹⁰, changes in stiffness measured at various lengths appear to be

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muscle dependent¹⁹³. It is therefore possible that changes would have been observed had we measured stiffness in a more tensioned position, as found by Umehara et al. Notably, our intervention may have increased slack length, or the joint angle in which passive tension begins to develop^{194,195}, contributing to the observed increases in ROM despite unchanged stiffness at rest.

Interestingly, we previously reported reductions in pectoralis minor stiffness following low- and moderate-intensity SS, despite using the same measurement position as in the current study. This inconsistency highlights methodological challenges, particularly the lack of standardization in SWE. Unlike electromyography, where values are often normalized to a reference (e.g., maximal voluntary contraction), SWE is traditionally reported in absolute terms, historically limited by saturation thresholds (i.e., upper measurement limits). With recent advances allowing higher saturation ceilings, relative stiffness normalization is now more feasible¹⁹⁶. Future studies should pursue standardized approaches to SWE quantification to improve comparability across investigations.

We hypothesized that pectoral SS would elongate pectoralis minor and thereby increase ROM. The observed time main effect on PML suggests that SS, performed at either moderate or maximal intensity, did not alter PML relative to the control condition. Although PML did not change at rest, it is possible that the extensibility increased, contributing to the observed improvements in haROM. Measuring PML at maximal haROM, rather than at rest, may have revealed significant SS-induced lengthening.

Shortened PML has been shown to alter scapular kinematics⁵⁶ and is commonly observed in individuals with shoulder pathology¹⁹⁷. Furthermore, reduced PML is associated with increased FSP⁵⁴, which in turn is thought to contribute to abnormal scapulohumeral rhythm,

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subacromial impingement, and glenohumeral instability, thereby elevating shoulder injury risk¹⁹⁸. Consequently, lengthening pectoralis minor remains a key objective in many shoulder injury preventive and rehabilitation programs.

Although the SS position used in the present study effectively lengthens pectoralis minor during stretching⁴⁹, the implemented dose may not have been sufficient to elicit changes at rest following stretching. Previous literature has reported increases in PML following 90° of horizontal abduction SS for 2x30s at end range in asymptomatic swimmers¹⁶², 5x30s in basketball players¹⁹⁹, 4x30s combined with a scapular posterior tilting exercise in those with short pectoralis minors⁵³, and twice-daily SS for 14 days in those with moderate forward head and rounded shoulders²⁰⁰. These findings suggest that our protocol (3x30s at 90° of horizontal abduction) was likely sufficient to lengthen pectoralis minor, particularly at maximal intensity, but the effect may be population specific. Because our participants were not recruited or stratified based on sport, posture, or baseline muscle length, sample heterogeneity may have obscured potential effects. Further investigation is warranted to examine whether stretch dose or participant characteristics (e.g., posture, sport, baseline muscle length) are primary factors influencing responsiveness of pectoralis minor to SS.

Posture is adaptive, developing through habitual or repetitive positions or activities²⁰¹, and FSP is similarly complex and multifactorial. Pectoralis minor length is only one of several contributing factors. For example, muscle imbalances or weakness in the periscapular and cervical muscles⁵⁴, repetitive overhead movements²⁰², prolonged and repetitive positions (e.g., office workers, texting), previous injuries, other postures such as thoracic kyphosis or forward head posture⁵⁴, psychological factors such as depression²⁰³ or self esteem²⁰⁴, and demographic or lifestyle factors such as age, gender, physical activity and BMI^{205,206} may all contribute to the

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development of FSP. While FSP has been associated with decreased subacromial space and altered scapular kinematics²⁰⁷, its direct role in shoulder pathology remains unclear. Indeed, several studies report no relationship between posture and shoulder injury^{52,208–210} and some argue that “ideal posture” is uncommon and unrealistic^{211–213}.

In the present study, pectoral SS did not reduce FSP more than a control condition, highlighting its variability and dynamic nature. A more comprehensive rehabilitative approach, encompassing stretching, strengthening, and other corrective exercises, may be necessary to meaningfully improve FSP²¹⁴. Future research should investigate the impact of FSP on the development of shoulder pathology, whether FSP severity influences shoulder pathology risk, and whether reducing FSP translates to meaningful reductions in shoulder injury risk.

Limitations

These results should be interpreted considering further limitations than those previously discussed. First, generalizability is limited, as our sample consisted of younger adults with asymptomatic shoulders and such results may not extend to older or symptomatic populations. Second, the maximal intensity SS intervention was standardized to each participant’s average pre-measurement haROM angle, which may not have fully reflected their transient maximal intensity at the time of stretching. In contrast, the moderate intensity condition allowed participants to determine intensity in real time. Finally, stretch intensity is inherently subjective and difficult to control, as individual perceptions of intensity can vary substantially, potentially leading to inconsistencies in the implementation of SS protocols.

Conclusion

This study demonstrated that both moderate and maximal intensity SS of the pectoral muscles acutely increased haROM in asymptomatic shoulders, with maximal stretching eliciting

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slightly greater gains. These improvements occurred without concomitant reductions in pectoralis major or minor stiffness, pectoralis minor length, or forward shoulder posture, suggesting that mechanisms other than muscle stiffness, such as enhanced stretch tolerance or altered slack length, may underlie ROM changes. Clinically, both moderate and maximal intensities appear effective, though maximal stretching may result in greater ROM gains in some individuals. Future research should examine inter-individual variability in response to SS, particularly across different intensities, to better inform evidence-based stretching prescriptions.

Chapter 5: Beyond Group Means: Individual Responsiveness to Moderate and Maximal Pectoral Static Stretch Intensities

Author Note & Contribution

Data and text from the following manuscript appears in:

Bohunicky, S., Scribbans, T.D. (2025) Individual responses to pectoral static stretch intensities. *SportRxiv*. <https://doi.org/10.51224/SRXIV.681>

Sarah Bohunicky conceptualized, designed, piloted methods, collected data, analyzed data, interpreted data, and drafted the manuscript.

Abstract

Static stretching (SS) is well-established to increase range of motion (ROM) at the group level; however, considerable inter-individual variability exists in physiological responses to exercise interventions. To optimize individual exercise prescriptions, it is important to understand variability in responsiveness to SS and identify factors associated with meaningful changes. This study examined the incidence of individual responses to different intensities of pectoral SS on forward shoulder posture (FSP), pectoralis minor length (PML), horizontal abduction ROM (haROM), and pectoral stiffness. It also explored whether baseline values differed between responders and non-responders, and how these influenced the magnitude of change. Thirty participants ($n=30$) completed two experimental sessions consisting of a control condition (CON) and two pectoral SS intensities: moderate and maximal. Pectoralis major and minor stiffness, PML, FSP, and haROM were measured before and after each intervention. The CON involved 150s of rest, while the SS interventions consisted of three 30s standing doorway stretches. Responders were defined as individuals demonstrating changes at least two times the typical error in the desired direction calculated from CON. Following moderate intensity, 43% of participants experienced a meaningful increase in haROM, compared to 70% following maximal intensity. Similarly, 37% and 40% of participants demonstrated meaningful increases in PML after moderate and maximal SS, respectively. Response rates for pectoralis major and minor stiffness and FSP were negligible but exhibited substantial inter-individual variability. No differences were found in baseline values between responders and non-responders; however, a moderately positive relationship was observed between baseline haROM and change in haROM. Higher stretch intensity increased the likelihood of meaningful ROM improvements, yet substantial inter-individual variability was evident across outcomes. These findings underscore the importance of individualized tailoring of SS interventions to optimize clinical outcomes.

Introduction

Static stretching (SS) is a widely prescribed intervention to increase range of motion (ROM), with a large body of evidence demonstrating group-level improvements in ROM through mechanisms such as decreased stiffness and/or increasing stretch tolerance^{4,6,153,172,215–217}. Although group-level analyses demonstrate improvements in ROM following SS, significant variation exists in individual responses, which are often concealed by overall group effects¹¹⁸. Individual response incidence is rarely reported, with only one study to date examining individual responsiveness to an acute bout of SS. Dobija et al., (2024) found that, despite significant group-level improvements in hamstring ROM following SS in individuals with chronic low back pain¹¹⁹, only 31% of participants were classified as responders⁶⁴. Similarly, in our own work, we observed significant average increases in ROM following both moderate and maximal intensity pectoral SS, yet several participants experienced no change or a decrease in ROM (Chapter 4, **Figure 15**). Likewise, although pectoralis minor stiffness decreased at the group level, only a small percentage of individuals (10% for low intensity, 30% for moderate intensity) were considered responders²¹⁸.

Clinically, identifying individual response patterns and non-response is essential for advancing personalized exercise prescription and rehabilitation strategies, leading to increased interest in classifying individuals as “responders” or “non-responders.” A non-responder is defined as someone whose change in a given outcome does not exceed the expected measurement error or predefined threshold derived from a control condition^{138,219}. One common method of classifying response uses the typical error (TE) to determine whether a change exceeds measurement error^{127,129,132}. Calculating TE in response to a control condition enables researchers to differentiate genuine changes in a given variable in response to an intervention as

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opposed to changes that occur randomly, such as biological variability, measurement error, or placebo effects²²⁰. For example, a non-responder to a SS intervention is an individual whose change in ROM does not exceed the measurement error, or falls below a defined threshold of meaningful change, including no change, a small change below the threshold, or a decrease in ROM.

To date, it remains unclear whether responsiveness to SS occurs uniformly across multiple outcome variables, whether response rates differ by SS intensity, and whether baseline characteristics distinguish responders from non-responders. Moreover, no studies to our knowledge have explored individual responses to upper-body SS. Thus, the primary objective of the current study was to: 1) determine the incidence of individual response or non-response of horizontal abduction range of motion (haROM), pectoralis minor length (PML), pectoralis major and minor stiffness, and forward shoulder posture (FSP) to moderate and maximal pectoral SS intensities. Secondly, identifying baseline characteristics that differentiate responders from non-responders may help to explain the variability in SS outcomes and inform more individualized stretching prescriptions. Therefore, a secondary objective was to: 2) determine the difference in baseline data of dependent variables with a meaningful number of responders compared to non-responders to moderate and maximal intensities of pectoral SS. We hypothesized that: 1) given the low response rates reported previously^{64,218} and the results of my previous work (Chapter 3 and 4), individual response incidence for both SS intensities will be low for all variables and greater response rates will occur with higher SS intensities for FSP, while response rates will be similar for PML and haROM; 2) baseline values will differ between responders and non-responders for a given variable. Lastly, it is plausible that individual baseline values could determine the amount of change observed for a given variable. Therefore, a final objective was to

3) explore the relationship between participants' baseline values for each outcome variable and the amount of change they experienced following the SS intervention (i.e., rate dependence).

Given that baseline value affects the magnitude and direction of response or change in an outcome variable following an intervention^{154,155}, we hypothesized that: 3) there will be a rate-dependent effect between participants' baseline values and change in all outcome variables.

Methods

Experimental Overview

A repeated measures crossover design was conducted to determine individual responses in ROM, pectoralis major and minor stiffness, and forward shoulder posture (FSP) following two pectoral SS at two intensities (moderate and maximal). Participants attended two experimental sessions separated by at least 72 hours, where one SS intensity was delivered to the dominant arm (identified using the Modified Edinburgh Handedness Index¹⁷⁸) at each session in a randomized order (**Figure 17**). A control intervention where the participants rested for 150 seconds was completed at the start of their first session. Pectoral stiffness (major and minor shear wave velocity, SWV), FSP, PML, and horizontal abduction ROM (haROM) were measured before (PRE) and after (POST) each SS intervention. All experimental procedures were approved by the University of Manitoba Research Ethics Board and were conducted in accordance with the Declaration of Helsinki¹⁷⁷. All SS and outcome measurements were delivered to the participants' dominant arm.

Participants

This was a retrospective analysis of a larger study (Chapter 4), where 24 participants were required for recruitment. Thirty-seven individuals from the University of Manitoba and surrounding community expressed interest in participation. To participate, participants had to be

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18-40 years old with no recent history (< 6 months) of pain, injury, or orthopedic disorders to either arm; have no diagnosed neurological or musculoskeletal disorders; no contraindications to the use of ultrasound (i.e., pacemaker, allergy to transmission gel); no history (< 5 years) of participation in sport/activities requiring extreme upper body ranges of motion (e.g., gymnastics, yoga, baseball pitcher); not currently pregnant or breast feeding; and not considered hypermobile (≤ 5 Beighton's score). To account for attrition and data errors, 30 participants ($n=30$, 15 female, 26 ± 4 years, 173.0 ± 9.9 cm, 73.2 ± 15.6 kg) were eligible and completed both experimental sessions.

Session One Overview

Prior to each session, participants were asked to refrain from upper body resistance training for 48 hours, and cannabis and alcohol consumption for 24 hours. Once informed consent was obtained, each participants self-reported their height, weight, age, and sex assigned at birth. Pectoralis major and minor stiffness, PML, FSP, and haROM were taken PRE and POST the SS and control (CON) interventions. Further details of methods can be found in Chapter 4. Participants completed CON at the beginning of the first visit, where PRE measurements were taken, followed by 150s of rest (**Figure 17**) and POST measurements. A five-minute rest was provided as a washout period, followed by PRE measurements, the first randomized SS intensity (moderate or maximal), and POST measurements.

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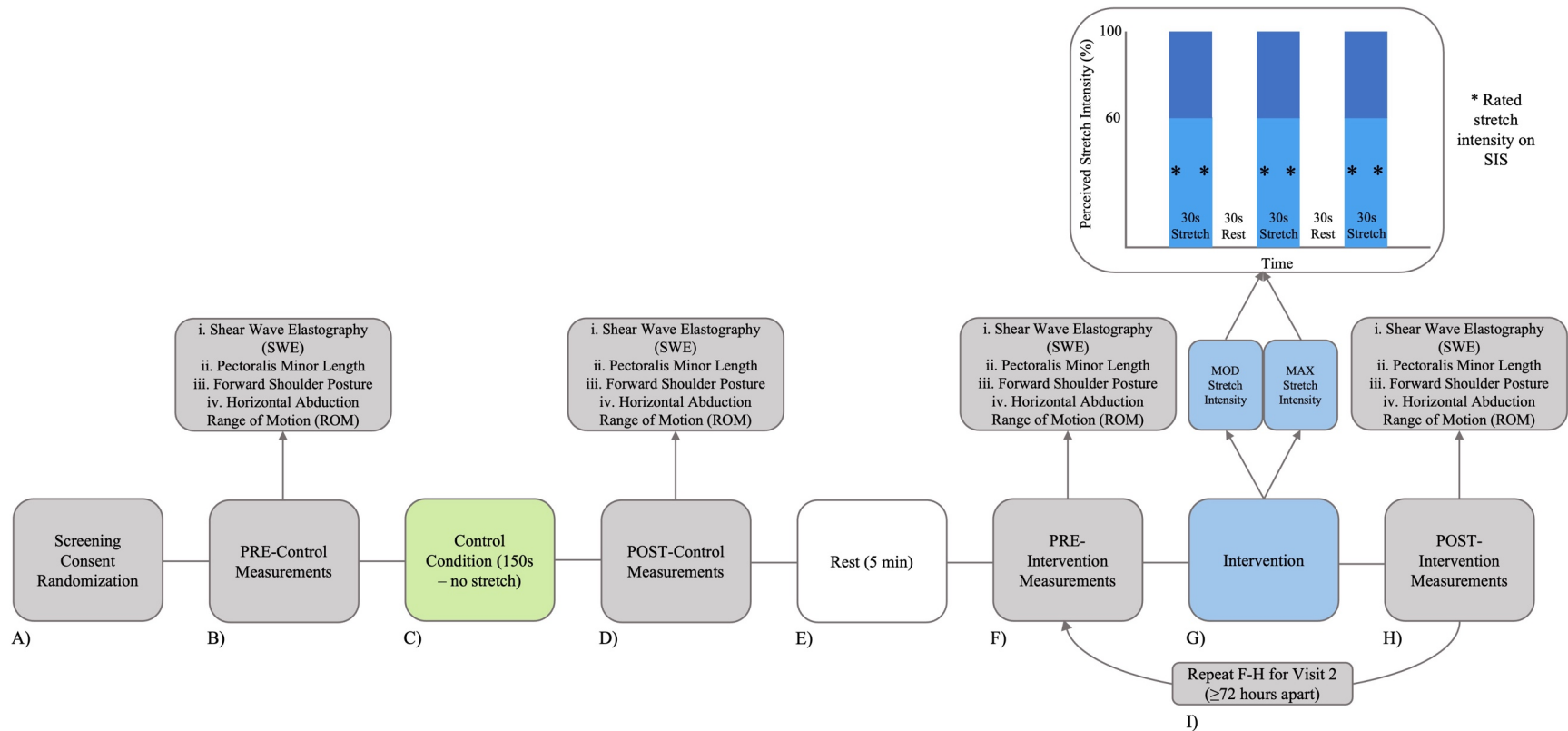


Figure 17. Schematic of Experimental Protocol

A) Once screened, eligible participants provided informed consent and received stretch intensity order randomization. B) PRE-intervention measurements: shear wave elastography (SWE) of pectoralis major and minor, pectoralis minor length, forward shoulder posture, and horizontal abduction range of motion were taken. C) Participants completed a control intervention where they rested for 150s. D) POST-intervention measurements were taken following the same procedures of PRE measurements (B). E) Participants rested for 5 minutes before F) completing PRE-intervention measurements again. G) A moderate (60) or maximal (100) on the stretch intensity scale standing doorway pectoral static stretch was completed for 3 sets of 30 seconds. Participants rated their perceived stretch intensity at the start and end of each stretching repetition. H) POST-intervention measurements were taken following the same procedures. I) Participants attended a second session a minimum of 72 hours later where the same procedures were followed (F-H) except they received the other stretch intensity.

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Shear Wave Elastography

Shear wave velocity (SWV) was used to assess pectoralis major and minor muscle stiffness via ultrasound (Logiq E10s, GE, Mississauga, Ontario, Canada). Transmission gel was applied to the XDclear linear probe (2-10 MHz, GE, Mississauga, Ontario, Canada), positioned approximately 1-inch below the dominant clavicle and lateral to the sternal edge. B-mode imaging identified the best location for pectoralis major and minor separately, where muscles fibres aligned longitudinally with the probe. This location was marked to ensure consistent probe location between measurements for a given session.

Shear wave mode generated a colour-coded SWV map within a region of interest (ROI). The ROI was set for the largest area possible within the fascial borders of each muscle that yielded a high-quality image with little or no artifact (none or very little monochromatic artifact). One, ~10s elastogram was taken for each muscle (pectoralis major, then minor), while the participant held their breath after a 3s exhale⁶³. Three high-quality images with minimal artifact were saved from each elastogram.

Pectoralis Minor Length

Pectoralis minor length (PML) was measured while the participants laid supine. Participants' coracoid process and anterior-inferior edge of the 4th rib, 1 finger-width lateral to the sternum were both landmarked and marked with permanent marker. The distance between these sites were measured 3 times using a soft measuring tap, with the average used for analysis of responder incidence.

Forward Shoulder Posture

Participants stood in a relaxed posture (i.e., not correcting their posture) with their back against a wall and had their anterior acromion process marked. A researcher then took a modified

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combination square and measured the distance from the wall to their marked acromion process (double-square method)¹⁷⁹. A total of three measurements were taken and between each, participants took a step forward and shook their arms to reset their natural posture. The average of the three measurements was used for analysis.

Horizontal Abduction Range of Motion (haROM) and Patient Reported Stretch Intensity

A validated stretch intensity scale (SIS)¹⁷⁶ ranging from 0 (no stretch) to 150 (maximal achievable ROM with pain) was adapted (**Figure 18**) and used for both the haROM and SS intensity measurements. HaROM was measured in the same position as the stretch intervention was conducted. Participants stood next to an upright stretching apparatus. They placed their forearm against the stretching apparatus with their shoulder abducted and elbow flexed to 90°. For haROM, they horizontally abducted their arm using the apparatus by taking steps forward to until they reached a 100 on the SIS, or the maximal achievable range of motion without provoking pain. A goniometer was used to measure the angle of horizontal abduction. All goniometric measurements were conducted by the same Athletic Therapist with over 9 years of experience. Once this angle was measured, the participant returned to the starting position and repeated the measurement two more times, where the average was used for analysis.

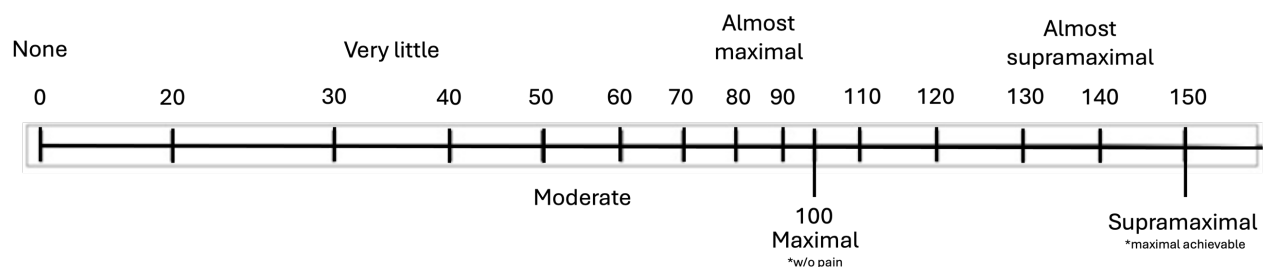


Figure 18. Stretch Intensity Scale

Adapted from Freitas et al. (2015)¹⁷⁶

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Control Intervention

Participants rested for 150s in a seated position on a plinth. After 150s, SWV, PML, FSP, and haROM POST-measurements were re-measured. The participant then rested for five minutes before completing PRE-measurements, followed by either the moderate or maximal SS intervention.

Static Stretch Intervention

Participants completed three sets of 30s of standing pectoral SS at either 60 or 100 on the SIS, respectively. If the participant was randomized to complete the maximal SS, they stretched at the average angle measured during PRE haROM measurements, as measured by the goniometer. If they were completing the moderate stretch, they horizontally abducted their arm until they reached a perceived 60 on the SIS, and a goniometer was used to measure this angle. Once the respective stretch intensity was reached, the position was held for 30s. The same angle was used for all three repetitions for each SS intensity. After 30s, the participant released the stretch and stood with their arm by their side for 30s before repeating two more sets of 30 seconds at the same angle as the first repetition. POST-measurements were conducted after each SS intervention.

Experimental Session Two

The second experimental sessions occurred a minimum of 72-hours after the first session. Following the same procedures as session 1 without CON (**Figure 17F-H**), participants completed the remaining SS intensity (moderate or maximal).

Data Processing

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Velocity (m/s) of shear wave propagation generated by the on-board system software (Logiq E10s, GE, Mississauga, Ontario, Canada) was extracted by a blinded investigator who closely examined all three images extracted from each elastogram. Avoiding artifact, they measured the largest possible area within the ROI. The same diameter and measurement location within the ROI was kept as consistent as possible between images for the same measurement for a given participant. The average of the three measurements was used for analysis.

To account for individual differences in height and limb length, pectoralis minor index (PMI)⁵⁶ was calculated by dividing each participant's PML by their height and used to compare baseline values between responders and non-responders.

Statistical Analysis

Intrasession reliability of the dependent variables were determined by a two-way mixed model absolute agreement intraclass correlation (ICC) using PRE-CON measurements. ICC values were interpreted as: poor (≤ 0.5), moderate (0.5-0.75), good (0.75-0.9), and excellent (≥ 0.9).

To achieve objective 1, typical error (TE) of all dependent variables (pectoralis major and minor stiffness, FSP, PML, and haROM) was calculated from the CON using the standard deviation (SD) of the pre-post difference scores using the following equation^{129,132}:

$$TE = SD_{diff}/\sqrt{2}$$

Participants were considered a responder if their pre-post change was greater than two times the calculated TE (i.e., $TE \times 2$) in the desirable direction for that variable. Consequently, if their change score was less than this number they were classified as a non-responder.

Inferential statistics were conducted in Jamovi (v. 2.5.4, The jamovi project). Normality of the data was determined using Shapiro-Wilk test for normality, where $p < .05$ would indicate a

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violation of normality. Outliers were considered studentized residuals greater than ± 3 standard deviations¹⁸¹. Data were considered statistically significant if $p < .05$.

To achieve objective 2, independent samples t -tests were conducted on dependent variable baseline values of responders and non-responders for each SS intensity with at least 10 responders (one third of all participants). Small, medium, and large effect sizes (Cohen's d) were interpreted as: 0.2, 0.5, and 0.8, respectively¹⁸⁰. Mann-Whitney U tests were conducted on non-parametric data. Effect sizes for the Mann-Whitney U tests were calculated using rank-biserial correlation (r_{rb}) and were interpreted as: negligible (< 0.1), small (0.1–0.3), intermediate (0.3–0.5), and large (> 0.5)¹⁸⁰.

To achieve objective 3, the Oldham method was used²²¹, whereby the relationship between pre-post changes (Δ) and the sum of PRE and POST values divided by 2 ($[(PRE+POST)/2; \bar{x}]$) was explored for each dependent variable. This method provides a more accurate way of measuring baseline dependent changes that reduces bias from mathematic coupling (i.e., using the same value in both variables being correlated) or regression to the mean (i.e., the natural tendency to move closer to the average over time)²²¹. Pearson correlations were conducted on normally distributed data and Spearman rank correlations were conducted on data that was not normally distributed. Correlation coefficients (r or rs) were interpreted as low < 0.3 , moderate 0.3–0.49, and high > 0.5 . Data are presented as mean and standard deviation for all parametric analyses and median and interquartile range for non-parametric analyses.

Results

Intraclass correlations for haROM were good (Cronbach's $\alpha = .849$), and excellent for FSP ($\alpha = .930$), PML ($\alpha = .994$), pectoralis major ($\alpha = .978$) and minor stiffness ($\alpha = .934$).

Objective 1: Responders and Non-responders

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Table 8 presents a summary of individual responses to both intensities of stretching for all variables.

For haROM, 2xTE was 4° (TE = 2°), resulting in 43% (13/30) of participants as responders to moderate, 70% (21/30) as responders to maximal, 37% (11/30) as responders to both and 23% (7/30) did not respond to either SS intervention (**Table 8, Figure 19**).

For pectoralis major stiffness, 2xTE was .92 m/s (TE = .46 m/s), resulting in 7% (2/30) of participants as responders to moderate, while 0% of participants responded to maximal or both, leaving 93% (28/30) as non-responders to both SS interventions.

For pectoralis minor stiffness, 2xTE was .94 m/s (TE = .47 m/s), resulting in 13% (4/30) of participants as responders to moderate, 10% (3/30) as responders to maximal, and 3% (1/30) responded to both SS interventions.

The 2xTE for PML was .33 cm (TE = .16 cm), resulting in 37% (11/30) of participants responding to moderate, 40% (12/30) responding to maximal, and 13% (4/30) responding to both, while 37% (11/30) responded to neither SS intervention (**Table 8**).

For FSP, the 2xTE was .63 cm (TE = .31 cm), resulting in 17% (5/30) of participants responding to moderate, 7% (2/30) responding to maximal, while 0% responded to both SS interventions.

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Table 1. Individual Responses of Horizontal Abduction Range of Motion, Pectoralis Minor Length, Pectoralis Major and Minor Stiffness, and Forward Shoulder Posture to Moderate and Maximal Static Stretching

Variable	Participant																														Total Responders /30		
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	MOD	MAX	Both
haROM	MOD			MOD	MOD		MOD	MOD					MOD	MOD	MOD	MOD	MOD	Both	Both	Both	MOD	Both	Both	MOD	Both	Both	Both	Both	Both	Both	Both	Both	Both
Pectoralis Minor Length	MOD	MOD	MOD	Both	MOD	MOD			MOD		Both	MOD	MOD			MOD	MOD		Both	Both	Both	MOD	MOD	MOD			MOD			Both			
Pectoralis Major Stiffness				MOD						MOD																							
Pectoralis Minor Stiffness	MOD					MOD					Both		MOD														MOD						
Forward Shoulder Posture				MOD					MOD	MOD				MOD	MOD								MOD							MOD			

MOD = MOD responder

MOD = MOD responder

Both = both responder

Non-responder = non-responder

haROM= horizontal abduction range of motion; MAX= maximal static stretch intervention; MOD= moderate static stretch intervention

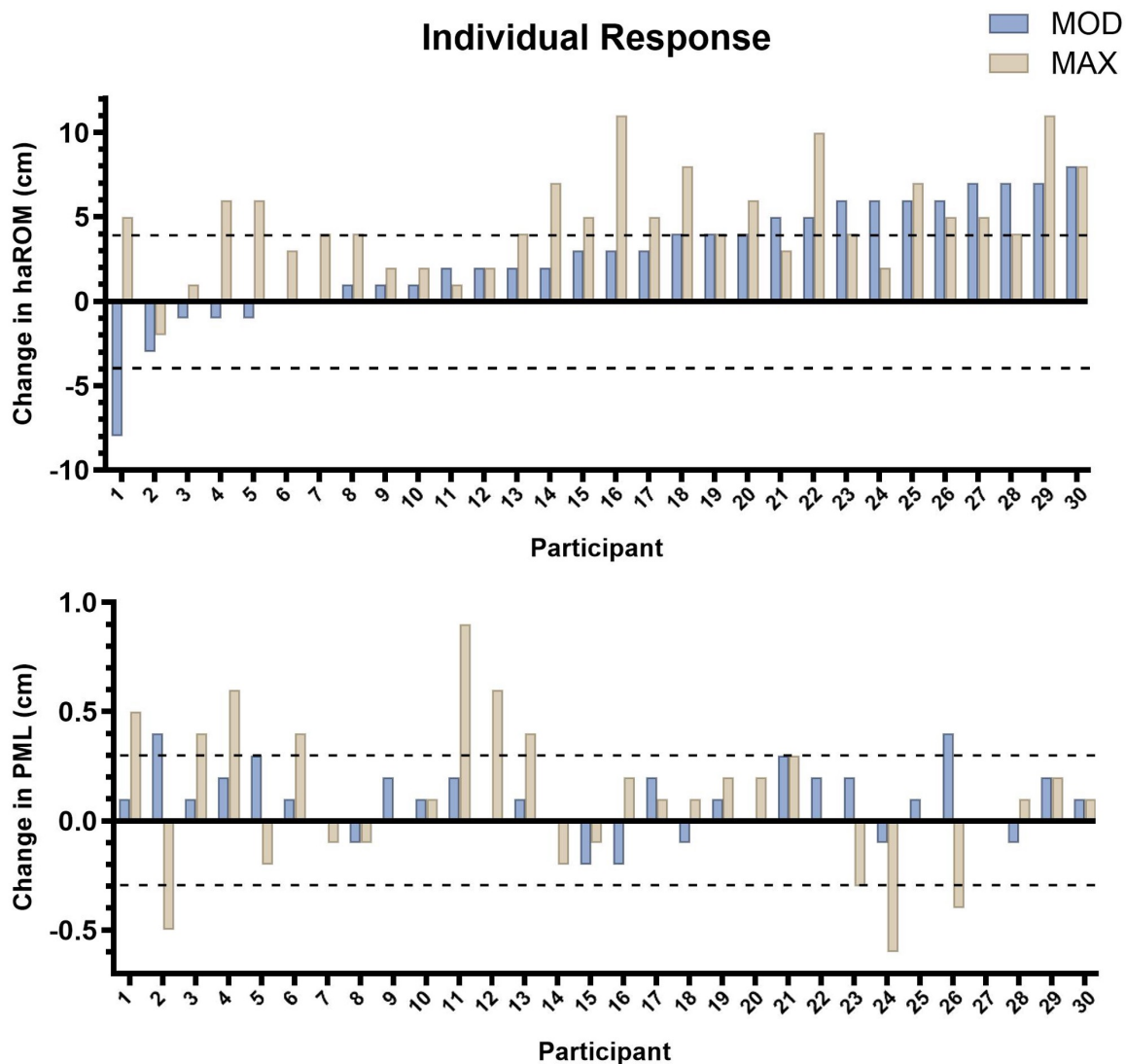


Figure 19. Individual Change Responses of Horizontal Abduction Range of Motion (haROM; top) and Pectoralis Minor Length (bottom) to Moderate (MOD) and Maximal (MAX) Static Stretch Intensities in Ascending Order of haROM response to MOD.

Objective 2: Baseline Differences Between Responders and Non-Responders

Baseline haROM for responders in the maximal intensity condition were not normally distributed ($p=.038$), thus a Mann-Whitney U test was used for that comparison. For the moderate intensity, there were no differences in baseline haROM between responders ($105\pm6^\circ$;

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$n=13$) and non-responders ($103\pm4^\circ$; $n=17$), $t(1,28)=-1.13$, $p=.269$, $d=-.415$, nor in the baseline PMI between responders ($8.21 \pm .55$; $n=11$) and non-responders ($8.31\pm.69$; $n=19$), $t(1,28)=.425$, $p=.674$, $d=-.161$. For the maximal intensity, there were no differences in baseline haROM between responders ($101\pm5^\circ$; $n=21$) and non-responders ($102\pm4^\circ$; $n=9$), $z=71.5$, $p=.307$, nor in baseline PMI between responders ($8.37\pm.53$; $n=12$) and non-responders ($8.09 \pm .83$; $n=18$), $t(1,28)=-1.15$, $p=.262$, $d=-.427$.

Objective 3: Rate Dependence

Data were not normally distributed ($p<.05$) for $\bar{x}$ (i.e., sum of PRE and POST values divided by 2) maximal haROM, $\bar{x}$ moderate pectoralis major SWV, and $\bar{x}$ and Δ moderate and maximal pectoralis minor SWV; thus, Spearman rank correlations were conducted on these variables. The correlation between $\bar{x}$ and Δ for haROM at moderate intensity was significant ($p=.036$, $r=.384$). The correlations were insignificant between $\bar{x}$ and Δ for all other variables and intensities (**Table 9**).

Table 8. Rate Dependence Results of the Average PRE and POST Scores ($\bar{x}$) and Change Scores (Δ) of Horizontal Abduction Range of Motion, Pectoralis Minor Length, Pectoralis Major Stiffness, Pectoralis Minor Stiffness, and Forward Shoulder Posture for Moderate and Maximal Stretching (n=30)

Variable	Moderate				Maximal			
	$\bar{x}$	Δ	p	r/rs	$\bar{x}$	Δ	p	r/rs
haROM (degrees)	105±5	3±4	.036*	.384	105±5	5±3	.301	.195 [⊥]
PML (cm)	14.3±1.3	0.09±0.16	.293	.199	14.3±1.3	0.1±0.3	.690	-.076
P.Maj SWV (m/s)	2.78±0.81	.08±0.71	.412	.155 [⊥]	2.88±0.42	0.16±0.53	.735	.064
P.Min SWV(m/s)	2.07±0.5	-.11±0.42	.360	-.173 [⊥]	1.92±0.55	0.1±0.54	.392	.162 [⊥]
FSP (cm)	10.8 ±1.6	-0.18±0.4	.664	.083	10.9±1.6	-0.1±0.4	.988	.003

$\bar{x}$ = (PRE+POST)/2; Δ = PRE-POST; cm = centimeters; FSP = forward shoulder posture; haROM = horizontal abduction range of motion; m/s = meters per second (velocity); P.Maj SWV = pectoralis major shear wave velocity (stiffness); P.Min SWV = pectoralis minor shear wave velocity (stiffness); PML = pectoralis minor length; r = Pearson's r ; rs = Spearman's rho; [⊥] = Spearman's Rank Correlation (median & interquartile range reported for $\bar{x}$ and Δ); * p < .05

Discussion

This study evaluated individual response and non-response in haROM, PML, pectoralis major and minor stiffness, and FSP following two pectoral SS of differing intensities. The key findings were: (i), a substantial portion of individuals were non-responders to one or both SS interventions, despite group-level haROM improvements for both; (ii) PML showed moderate non-response and FSP considerable non-response, despite group-level improvements over time (increased PML and decreased FSP); (iii) individual response patterns were inconsistent across outcome variables, although every participant responded to at least one variable following one intensity; (iv) baseline characteristics did not significantly differ between responders and non-responders for haROM or PML; and (v) a moderate rate-dependent effect was observed for

haROM at moderate intensity, suggesting that higher baseline haROM values result in greater changes in haROM.

Incidence of Responders

The primary finding of this study is the substantial non-response rate for haROM, with 57% and 30% of participants failing to show meaningful improvement in haROM following moderate and maximal, respectively. This occurred despite significant group level improvements in haROM at the group level for both intensities (Chapter 4), which aligns with the majority of literature demonstrating the efficacy of SS for improving ROM^{3–5,83}. These findings challenge a common clinical assumption that SS yields improvements in ROM for all individuals, revealing a more complex reality of inter-individual physiological response variability.

This variability is consistent with recent work by Dobija et al. (2024), who reported a 69% non-response rate in ROM following 1-minute of hamstring SS in individuals with chronic low back pain⁶⁴, but reported significant increases in the group analysis¹¹⁹. Their higher non-response rate (69% compared to 30-57% in the current study) may reflect methodological differences in defining responders. Specifically, Dobija et al. classified individuals as responders if their ROM met or exceeded the group mean change (≥ 7)⁶⁴, whereas our study used the typical error (TE) as the threshold. The use of the group mean does not distinguish true intervention effects from biological variability and measurement error, risking misclassification of individuals²²². In contrast, TE provides a more conservative but robust criterion for detecting true change due to its consideration of biological variability and measurement error^{132,135}. Thus, while Dobija et al. observed greater apparent variability across individuals, our approach suggests that a larger proportion of participants demonstrated true, meaningful improvements. Future research should use consistent and standardized responder criteria, specifically the 2xTE threshold which

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accounts for biological variability and measurement error, to improve comparability across studies.

Our results demonstrate that more individuals haROM responded to the maximal intensity SS (70%) compared to the moderate SS (43%). Although both groups improved similarly in haROM (3° vs. 5°), the $\sim 2^\circ$ greater change with maximal intensity was not significant and fell below the minimal detectable change (MDC_{95})²²³ of 2.9° , indicating that it was within measurement error. Thus, while group-level effects appear comparable, the markedly higher response rate to maximal intensity suggests that moderate intensity is not equally as effective as maximal. Notably, 37% responded to both intensities, whereas only 7% (2/30) responded exclusively to the moderate intensity, and 33% (10/30) exclusively to the maximal intensity, suggesting that the most effective SS intensity may vary across individuals. From a clinical standpoint, if a patient fails to demonstrate beneficial changes from one intensity, it may indicate that the intervention is ineffective for that individual and modifying the dose and/or intervention type may yield more optimal outcomes. For example, if an individual does not respond with increased ROM to moderate intensity SS, shifting to maximal intensity may result in improvements, as it did for 10 of our participants. As such, clinicians should adopt a responsive, data-informed approach by tracking individual responses, thereby enabling more effective and personalized prescriptions that ultimately improve patient outcomes. Taken together, these findings indicate that higher intensity stretching increases the likelihood of eliciting meaningful ROM improvements across individuals compared to lower intensity, and that clinicians should tailor stretching prescriptions based on individual responsiveness to a given stretch intensity.

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As increases in muscle length and reductions in stiffness are proposed mechanisms underlying SS-induced ROM gains^{110,112,153,172}, we anticipated that participants who gained ROM would also show corresponding changes in muscle length and/or stiffness. However, considerable variability was also evident across other outcome measures, with inconsistent response patterns emerging between ROM, muscle length, and stiffness. For example, participants who responded in haROM did not necessarily respond in PML or muscle stiffness to the same SS intervention. Changes in mechanical muscle length, thought to result from alterations in muscle morphology such as stiffness, are a purported mechanism for increasing dorsiflexion^{112,153,172} and knee extension¹¹⁰ ROM. As such, we anticipated that individuals showing meaningful haROM gains would also demonstrate increases in PML and reduction in pectoral stiffness. However, of the 34 haROM responders across both intensities, only 13 (38%) also responded in PML, while 0 and 2 (6%) responded in pectoralis major and minor stiffness, respectively. This suggests that ROM gains may arise from diverse, individual-specific mechanisms, rather than consistent changes in muscle stiffness or length. Notably, PML decreased in 11 participants without a concomitant increase in haROM, further indicating that muscle lengthening does not necessarily translate to improvements in ROM. Collectively, these findings indicate a complex inter-individual variability of stretching responses to several outcomes variables and reinforces the notion that effective SS prescriptions require individualization rather than reliance on group-level trends. Notably, this is only the second study to examine response rates to SS, and the first to do so in a pain-free population, further underscoring the novelty of these findings.

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Baseline Characteristics Between Responders and Non-Responders

Baseline characteristics may help to explain the variability in SS outcomes; however, contrary to the hypothesis, there were no significant differences in baseline haROM or PMI between responders and non-responders at either SS intensity. These results suggest that baseline flexibility and muscle length alone do not predict their responsiveness to SS. These results indicate that the benefits of SS appear to occur independently of initial ROM or muscle length, and that individuals with more restricted ROM do not necessarily experience greater responsiveness.

To our knowledge, no previous studies have directly compared baseline ROM or muscle length values between SS responders and non-responders. One investigation examined clinical characteristics (e.g., age, sex, body mass index [BMI], type of work, living environment, smoking status, and various other indexes) between responders and non-responders to hamstring stretching, reporting only higher BMI in non-responders, with no other significant differences⁶⁴. Given the high variability in SS responses, the dearth of research defining factors influencing responsiveness is surprising and highlights the need to identify potential predictors. Such work could explore whether individuals with shorter or longer baseline muscle lengths, or differing levels of baseline flexibility, demonstrate distinct response patterns. Moreover, future work should investigate other characteristics that may predict stretch response, such as prior flexibility training, which has been shown to influence responsiveness²²⁴. From a clinical perspective, these findings suggest that individuals can experience meaningful changes in ROM and muscle length from SS, independent of baseline values. As such, clinicians should monitor individual changes in ROM over time and adapt SS prescriptions accordingly.

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Rate Dependence

We observed a moderate positive correlation between baseline haROM and change in haROM at moderate intensity, indicating that individuals with higher initial haROM tended to show greater improvements in ROM. No such relationship was found at maximal intensity, suggesting that higher intensity may elicit improvements more universally, independent of starting ROM. Contrary to the hypothesis, no other rate-dependent effects were found for PML, pectoralis major or minor stiffness, or FSP, demonstrating that the direction or rate of change in these variables were not impacted by their baseline values.

To our knowledge, this is the first investigation to directly examine rate dependence in response to SS, which may partially explain the inter-individual variability in responsiveness discussed above. Previous studies have explored the influence of baseline flexibility on stretching outcomes with mixed findings. Donti et al., (2014) reported that baseline flexibility did not affect acute hip ROM gains in elite gymnasts, though they did not systematically evaluate baseline effects²²⁵. In contrast, Fraulin et al., (2021) found greater chronic ROM improvements in office workers with lower baseline flexibility following 3 months of stretching²²⁶. Taken together, these results^{225,226} and the results from the current work suggest that baseline flexibility may interact with both the type of stretching (acute vs. chronic) and the intensity of stretch to influence changes in ROM.

While our group-level analysis (Chapter 4) revealed improvements in haROM, PML and FSP following both intensities, the magnitude of change did not differ if an individual had greater or lesser ROM, muscle length, or FSP, except for the haROM-moderate intensity relationship. Clinically, this suggests that individuals with relatively high baseline ROM may benefit from more moderate intensity SS, while maximal intensity may be a more reliable

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strategy across diverse populations. Further, while no group effect was observed for pectoralis major or minor stiffness, treatment response was also not impacted by baseline levels of these variables. These findings reinforce the need for individualized monitoring and prescription, as stretching responses are not uniform and may depend on both baseline status and applied intensity.

Limitations

The findings from the current study should be interpreted in light of several limitations. First, results are specific to the dominant side of a young, asymptomatic population, which may limit generalizability to clinical or older populations. Secondly, we did not examine sex-specific responses, nor did we monitor menstrual cycle phase in female participants. Given that hormonal fluctuation across the menstrual cycle can influence ROM and muscle stiffness¹⁶⁹, this may have contributed to variability in responses to SS. Third, pectoral stiffness was measured with the arms at rest. Although prior research has demonstrated stiffness changes in resting positions following SS¹⁹¹, it is possible that alterations in pectoral stiffness are more apparent when measured in a lengthened position (beyond slack length). Fourth, the manipulation of stretch intensity is inherently subjective. Despite the use of a validated scale, individuals likely perceived and applied moderate and maximal stretch intensities differently, introducing variability in the intervention dose. Finally, although the 2xTE threshold is a conservative and robust criterion for defining responders, alternative approaches such as MCD or the smallest worthwhile change may yield different responder classifications.

Conclusion

This study demonstrated considerable inter-individual variability in acute responses to static stretching, with a substantial proportion of participants classified as non-responders despite

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group-level improvements in haROM, PML, and FSP. Higher stretch intensity increased the likelihood of eliciting meaningful ROM gains, although some individuals responded only to moderate intensity. Notably, changes in muscle length or stiffness were not consistently associated with haROM responsiveness, despite being two purported mechanisms for ROM improvement. Baseline haROM and PML did not distinguish responders from non-responders, underscoring the importance of individualized monitoring rather than reliance on initial flexibility or muscle length measures. Collectively, these findings emphasize that static stretching prescriptions should be tailored to individual responses to optimize clinical and performance outcomes.

Chapter 6: General Discussion

The central aim of this thesis was to investigate both group and individual responses to varying intensities of pectoral SS in asymptomatic adults. Specifically, this dissertation evaluated the group effects of low and moderate intensity therapist-administered pectoral SS, as well as group and individual effects of moderate and maximal intensity self-administered SS on changes in ROM, pectoral stiffness, muscle length and FSP. Although pectoral SS is commonly prescribed in shoulder injury prevention and rehabilitation programs, its acute effects have received little empirical attention. This work contributes to the literature in three novel ways: first, it is the first study to examine the acute impact of pectoral SS on FSP, pectoralis major stiffness, and haROM; second, it is the first to compare different SS intensities in the upper body; and third, it is the first to explore the incidence of individual responsiveness to SS in asymptomatic individuals, and the second to explore responsiveness to SS generally. The following discussion is organized into two sections: group-level effects and individual-level responses.

Impact of Pectoral Static Stretching: Group-Level Effects

Horizontal Abduction Range of Motion

The present work produced mixed findings regarding the effects of different pectoral SS intensities on haROM. Moderate and maximal intensity self-administered SS resulted in significant improvements in haROM, whereas low and moderate intensity therapist-administered did not elicit any changes. These findings are partially consistent with the broader literature demonstrating that a single bout of SS acutely increases ROM, irrespective of intensity^{3,171}.

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However, it is worth noting that all previous investigations on SS intensity have focused exclusively on the lower limb, highlighting the novelty of the current upper-limb findings.

Chapter 3 demonstrated that low and moderate therapist-administered pectoral SS did not alter haROM, a finding that contrasts previous literature reporting ROM gains across a range of intensities of SS^{3,171}, including low intensity protocols^{38,41}. Several methodological factors may account for these null findings. First, haROM was assessed passively without external overpressure, which limited the evaluation of pectoral muscle extensibility and prevented participants from reaching a true end range of motion. Second, stretch intensities were defined as 110% and 120% of baseline ROM, where baseline was the first sensation of a stretch reported by the participant as the therapist slowly moved the arm through horizontal abduction. Because the perception stretch sensation is highly subjective, variability in participants' perceptions of initial stretch likely influenced the classification of baseline ROM and, consequently, the relative intensity of the 110% and 120% conditions. Although these intensities were selected based on pilot data indicating that they represented moderate and high stretch intensities, participants' perceived intensity ratings averaged only 20/100 ("slight") and 31/100 ("slight to moderate"), respectively. Given this discrepancy, it is plausible that the intensities tested were not sufficient to elicit meaningful adaptations, and abundant variation in intensity prescription methods in the broader literature make it difficult to compare across studies. However, with previous work demonstrating ROM improvements following low intensity SS^{3,38,41,171}, the lack of effect observed in the present study is surprising.

The results of Study One (Chapter 3) highlighted the need to implement a more rigorous pectoral SS protocol capable of assessing true muscle extensibility, while also including a control condition for comparison. Furthermore, it underscored the importance of examining differences

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between types of SS, particularly given that self-administered protocols are anecdotally more frequently performed and may better reflect ecologically valid practices, where stretching is typically prescribed based off perceived intensity. With these modifications, the findings from Study Two (Chapter 4) demonstrated that both moderate and maximal intensity SS produced significant increases in haROM, supporting previous research indicating that SS across a range of intensities can enhance ROM^{3,171}, even in the upper limb. Notably, maximal intensity increased haROM by an average of $\sim 2^\circ$ more than moderate, providing preliminary evidence for a potential dose-response relationship between stretch intensity and ROM.

Enhanced stretch tolerance may represent one mechanism underlying this effect, as evidenced by a greater reduction in perceived stretch intensity from the start to the end of the maximal condition (-17/100) compared to the moderate condition (-9/100). Several prior studies have reported larger ROM gains with higher SS intensities compared to lower intensities^{12,25,30–33}. However, comparison across studies is complicated by methodological heterogeneity, including approaches used to prescribe intensity and the manipulation of multiple dose variables (e.g., intensity and duration)²⁷. As such, the present results align with a recent systematic review suggesting that the effect of stretch intensity on ROM remain inconclusive³. It is suggested that a more objective approach to prescribing and assessing SS intensity may be warranted, such as the degree of muscle stiffness²⁷. Alternatively, a more ecological approach to prescribing intensity may involve using the percentage of maximal ROM²⁷.

Given the methodological differences in haROM assessment between Studies One and Two, it cannot be conclusively determined that lower intensity, therapist-administered SS does not affect haROM compared to higher intensity, self-administered SS. Despite these varied results, several important gaps remain. A primary gap concerns the mechanisms underlying ROM

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improvements following stretching. Although reductions in muscle stiffness and increases in stretch tolerance, potentially mediated by neural adaptations altered pain perception, and expectancy effects, have been proposed, these mechanisms are not yet fully understood^{6,84,110,112,153,172,183,184}.

Another gap concerns potential sex differences in the response to SS. Evidence suggests that males and females may differ in ROM adaptations to stretching¹⁸⁵, and hormonal fluctuations across the menstrual cycle further influence flexibility outcomes¹⁶⁹. Previous research has predominantly involved male participants (~82%), whereas the present study included a sex-balanced sample (50% female) but did not control for the menstrual cycle phase. Consequently, the female response to SS across the different phases of the menstrual cycle remains unclear. Collectively, these factors complicate comparisons both within the current participant cohort and across studies. Future research should therefore examine sex differences in SS adaptations more systematically, including the influence of the menstrual cycle phase on ROM outcomes.

Pectoral Stiffness

A primary mechanism posited for improved ROM following SS is a decrease in muscle-tendon unit stiffness^{110,112,153,172}. Given the acute increases in ROM commonly observed after SS^{3,171}, the current work hypothesized that SS would decrease pectoralis major and minor stiffness, thereby contributing to enhanced ROM. However, the findings from the current investigations revealed that low, moderate, and maximal intensity SS did not significantly influence pectoralis major stiffness. Pectoralis minor stiffness demonstrated a temporal decrease in Study One, examining low and moderate intensity therapist-administered pectoral SS, yet it remained unaffected by moderate and maximal intensity self-administered pectoral SS in Study

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Two. These results largely diverge from existing literature that consistently reports decreased stiffness following various SS protocols.

Stiffness measurement methodologies have evolved significantly. Conventional techniques, such as dynamometry, assess passive torque-ROM relationships to derive a global muscle-tendon unit stiffness. This approach inherently encompasses all soft tissue crossing a joint, including muscle, tendon, and fascia. In contrast, novel methods like shear wave elastography (SWE), offer greater specificity, enabling direct, localized measurement of elastic properties within specific soft tissues, such as distinct regions within a muscle. SWE also extends measurement capabilities to areas not accessible via dynamometry, such as the pectoral muscles. While previous research, employing both passive torque¹⁰² and SWE¹⁹³, has demonstrated acute decreases in stiffness after a single bout of SS, the effects of SS on shear modulus appear to be muscle-specific and exhibit considerable variability¹⁹³. For instance, stiffness in the semimembranosus and semitendinosus (medial hamstrings) decreased in 80% of studies following SS, whereas the biceps femoris (lateral hamstring) showed decreased stiffness in only 60% of studies, and only when SS durations exceeded 120 seconds¹⁹³.

The current work contributes to a limited body of research examining shear elastic modulus (hereon stiffness) changes in the upper limb and represents only the second research group to specifically investigate pectoral muscle stiffness. Umehara and colleagues reported a decrease in pectoralis minor stiffness after as little as 30 seconds of pectoral SS at 135° of humeral elevation with external rotation to maximal, pain-free ROM⁶³. Critically, Umehara measured pectoralis minor stiffness with the arm at 90° of elevation compared to measuring at rest as done in the current work. This difference in measurement position supports previous assertions that stiffness significantly decreases in a stretched versus a relaxed or slack position,

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underscoring the substantial role of measurement posture in observing changes in stiffness^{193,227,228}. Consequently, it is plausible that the interventions employed in the current work would have yielded more pronounced effects on pectoralis major and minor stiffness had measurements been conducted with the muscles in a more lengthened position, which is a limitation of the current work.

Despite the advancements offered by SWE, these findings, in conjunction with prior SWE literature, expose existing methodological challenges and knowledge gaps. Traditionally, SWE measurements are presented as an absolute expression of stiffness, limited by older versions' saturation limits, which hindered accurate measurement of very stiff tissue, such as those during high-intensity contractions¹⁹⁶. In contrast, electromyographic methods are routinely expressed relative to a reference contraction, such as a maximal voluntary isometric contraction (MVIC). Recent technical advancements in SWE have expanded the measurable shear modulus range, facilitating accurate measures of stiffer tissues, including during MVIC¹⁹⁶. Nevertheless, research continues to report stiffness as absolute measures, while relative expressions may offer improved comparability between muscles and across studies.

Furthermore, resting muscle stiffness does not reliably predict muscle stiffness during contraction, and the rate of stiffness increase during contraction is dependent on each individual's strength²²⁹. Therefore, while the SS interventions in these studies could have altered pectoralis major and minor stiffness during contraction, the measurement at rest and the lack of normalization relative to a maximal contraction may explain the absence of observed stiffness changes. Thus, a significant limitation of the current work is that the stiffness measurements conducted provide limited insight into the acute stiffness adaptations of the pectoralis muscles to the employed SS interventions. Future research should prioritize relative stiffness measures and

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assess stiffness in both resting, lengthened, and/or contracted states to provide a more comprehensive understanding of SS-induced stiffness adaptations.

Pectoralis Minor Length

A purported mechanism for increased ROM following SS is an increase in mechanical muscle length. As such, it was hypothesized that PML would increase after SS, particularly in instances where ROM improved. PML is a validated measure for assessing interventions designed to lengthen muscle^{230,231}. Interestingly, while the current findings revealed a temporal increase in PML in Study Two, no changes in PML were found in Study One. Moreover, in Study Two, both moderate and maximal intensity SS elicited similar increases in PML to the control condition, suggesting that the employed SS intensities did not affect resting PML.

Despite pectoral stretching being a cornerstone of many shoulder injury prehabilitation and rehabilitation protocols^{51,52,232}, literature evaluating the acute impact of pectoral SS on PML is notably sparse. In contrast to the current work, existing studies suggest that a single bout of pectoral SS acutely increases PML^{53,162,199}. Specifically, a 5x30 second doorway stretch¹⁹⁹ (similar to that of Study Two) and a 2x30 second supine horizontal abduction stretch¹⁶² (similar to that of Study One), and 4x30 second supine horizontal abduction pectoral stretch combined with a posterior tilting exercise⁵³ have all been shown to significantly increase PML. Crucially, these acute studies measured PML in a standing position, whereas the current work utilized a supine position. This postural difference may influence changes in PML and could be more sensitive to length alterations. Therefore, it is plausible that measuring PML in a standing posture in the current work might have revealed changes in PML.

Although resting PML did not change, it is conceivable that pectoralis minor extensibility increased, contributing to the improvements observed in haROM in Study Two. Consequently,

measuring PML in the maximal haROM position might have yielded significant changes. Given these considerations, the current work cannot definitively confirm that PML increased following pectoral SS, nor can it ascertain if such an increase would contribute to the observed ROM improvements in Study Two, or in Study One had end ROM been assessed. Future research should further investigate the contribution of muscle length to change in ROM following SS.

Forward Shoulder Posture

FSP is a common postural deviation associated with a shortened pectoralis minor, reduced subacromial space, and altered scapular kinematics consistent with shoulder pathology²⁰⁷. Accordingly, increasing PML and reducing FSP are central goals in many shoulder injury prevention and rehabilitation programs. It was hypothesized that pectoral SS would decrease pectoralis minor stiffness, thereby increasing PML and reducing FSP. Study One supported this hypothesis, demonstrating significant decreases in FSP following both mild and moderate pectoral SS concomitant with transient decreases in pectoralis minor stiffness. However, Study Two found that reductions in FSP did not differ significantly from a control condition, suggesting that these changes were not attributable to the stretching intervention.

Limited research has examined the acute effects of a single pectoral stretch on FSP. Previous studies have reported that various pectoral massage therapy techniques, both alone^{58,159–161} and in combination with SS⁵⁸, can acutely decrease FSP. However, the clinical relevance of these changes remains unclear¹⁵⁹, particularly given that reduction in FSP have also been observed following control or placebo conditions^{159,160}, consistent with the present findings. The decreases in FSP observed across both experimental and control conditions underscore the high variability of postural measures, which appear independent of changes in haROM, PML, or pectoral stiffness.

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This variability raises important questions: does the FSP measurement reliably capture clinically meaningful postural changes, and are observed decreases in FSP genuine adaptations or transient adjustments influenced by conscious or subconscious awareness? Posture is inherently multifactorial, shaped by habitual movement patterns, muscular imbalances, prior injuries, psychological factors (e.g., depression, self-esteem), and demographic or lifestyle variables such as age, sex, physical activity, and body mass index^{54,201,204–206}. Given this complexity, a single bout of pectoral SS may not produce lasting or clinically significant reductions in FSP sufficient to lower shoulder injury risk.

The present results highlight FSP's dynamic nature, characterized by transient fluctuations following both stretching and control conditions. This suggests that a single FSP measurement may not accurately represent a persistent postural deviation relevant to shoulder pathology. Future research should determine the threshold for clinically significant change in FSP, identify the factors contributing to such changes, and evaluate whether specific therapeutic interventions like SS can meaningfully and sustainably modify FSP.

Impact of Pectoral Static Stretching: Individual Effects

Group-level analyses can obscure considerable inter-individual variability in response to SS. Understanding the individual response incidence is essential for clinicians seeking to move beyond a one-size-fits-all approach to prehabilitation and rehabilitation and toward personalized exercise prescription. Although group-level results from Study Two resulted in a significant increase in haROM following both moderate and maximal intensity pectoral SS compared to CON, individual analyses revealed substantial non-response rates: 57% of participants were non-responders in the moderate intensity, and 30% of individuals were non-responders for the maximal intensity. The greater responsiveness to higher-intensity SS aligns with the group-level

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dose-response trend, indicating that higher intensities may produce larger haROM gains more consistently across individuals.

To date, only one known study has examined individual SS response rates. Dobija et al reported that 69% of participants with low back pain failed to achieve meaningful ROM improvements following 1 minute of hamstring SS⁶⁴, despite significant group-level increases¹¹⁹. Differences in responder classification likely contributed to the discrepancy in response rates between their study (69%) and the current work (30-57%). Specifically, Dobija et al. defined responders as those who met or exceeded the group mean change⁶⁴, whereas the current study used a threshold of two times the TE derived from the control condition. The TE approach, though conservative, is considered robust for identifying true change as it accounts for biological variability and measurement error^{132,135,222}. Nevertheless, both studies underscore considerable inter-individual variability in SS-induced ROM changes, highlighting the limitations of relying solely on group-level data.

Baseline characteristics may partially explain response variability. Previous work suggests that individuals with limited baseline ROM exhibit greater improvements following SS²³³. Accordingly, the present study examined differences in baseline haROM between responders and non-responders and explored rate-dependent effects. No significant differences in baseline haROM were found between responders and non-responders, suggesting that initial flexibility does not influence responsiveness to either moderate or maximal intensity SS. Interestingly, a moderate positive rate-dependent relationship emerged for moderate intensity SS, indicating that participants with higher baseline haROM experienced greater improvements. This pattern was absent in the maximal intensity condition, suggesting that higher intensity SS may elicit benefits more universally, independent of initial ROM. These findings contribute to the

limited and mixed literature, where some evidence supports greater ROM gains among less flexible individuals²³³, while others report no baseline effect²²⁵. Collectively, the present results imply that moderate intensity SS may be more impactful for individuals with higher baseline ROM, whereas maximal intensity SS may be effective across a broader range of flexibility levels.

Several considerations should inform the interpretation of these findings. The results are limited to the dominant, healthy shoulders of young adults free from pathology, with relatively homogenous baseline ROM values (mean= 105°, standard deviation= 5°). This limited variability may have reduced the ability to detect baseline-dependent effects. Other unmeasured factors, such as prior flexibility training, may also influence responsiveness²²⁴. Although participants were screened to exclude those who engaged in sports or activities that require extreme ranges of motion (e.g., gymnastics, baseball), no formal assessment of flexibility training history was conducted. Consequently, determinants of SS responsiveness remain largely unknown, particularly in those with shoulder pathology or across the lifespan. Future research should aim to identify predictors of SS response to better inform clinicians' ability to tailor SS prescriptions based on individual characteristics.

Clinical Relevance

The findings of this thesis have several important implications for clinical practice, particularly for musculoskeletal clinicians who prescribe SS of the pectoral musculature to improve ROM, posture, or to prevent shoulder injury. While SS is a simple and accessible therapeutic intervention that remains a foundational component to many sporting, physical fitness and clinical settings, empirical evidence supporting its acute impact has been limited. For example, previous literature has found that SS at various doses leads to increased ROM^{3,233}.

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However, despite these results, stretch intensity is often poorly reported²³³, and literature examining different intensities has been exclusively conducted on lower limb musculature. The findings from the current work addresses these gaps and demonstrates that moderate and maximal intensity self-administered pectoral SS can acutely enhance haROM, whereas lower intensity, therapist-administered protocols did not produce similar changes in ROM under the conditions tested. However, the current work cannot conclusively determine that lower intensities are ineffective, as methodological factors (e.g., absence of end ROM testing) may have influenced these results. Therefore, it remains possible that lower intensity SS may also increase ROM.

Importantly, the absence of significant changes in pectoralis major and minor stiffness and PML indicates that acute gains in haROM likely reflect increased stretch tolerance rather than structural or physiological alterations to muscle tissue. Clinicians should therefore recognize that short-term flexibility improvements may be transient and primarily neurological.

Most importantly, the high degree of inter-individual variability observed in SS responsiveness underscore the importance of personalized SS prescription. Despite the standardized conditions, up to 57% of participants did not achieve meaningful haROM improvements following moderate intensity SS, highlighting the limitations of a universal SS prescription approach. However, given the lower non-response rate for maximal intensity (30%), and the potential dose-response relationship between higher intensities and greater increases in ROM, clinicians may consider prescribing higher initial pectoral SS intensities. This may be especially relevant for individuals with lower initial ranges of motion, given the rate-dependent findings in the current work. Regardless, clinicians should monitor individual response to SS

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interventions and adjust intensity, duration, and frequency based on observed outcomes rather than relying solely on standardized protocols.

Although pectoral SS is often implemented to correct FSP, the present findings suggest that a single SS intervention does not reliably alter FSP relative to control conditions. Given the multifactorial nature of posture, interventions aimed at reducing FSP should integrate stretching with other components of rehabilitation (e.g., strengthening and/or ergonomic education) rather than relying on singular SS interventions alone.

Collectively, these results suggest that pectoral SS can be a valuable adjunct to shoulder mobility programs when performed at sufficient intensity and tailored to the individual. However, clinicians should temper expectations regarding its immediate effects on FSP, pectoral stiffness, and PML. Future work is necessary to determine if individualized pectoral SS prescriptions produce improved acute and chronic adaptations in ROM, stiffness, muscle length and posture in both healthy and symptomatic populations.

Chapter 7: Conclusion

This thesis was the first work to investigate both group and individual responses to varying intensities of pectoral SS in asymptomatic adults, addressing key gaps in the literature related to the impact of SS on ROM, muscle stiffness, muscle length, and posture. Across three studies, this work demonstrated that moderate and maximal intensity self-administered SS acutely increased haROM, while low and moderate intensity therapist-administered SS did not elicit significant changes. Although these findings suggest a potential dose-response with higher intensities increasing ROM to a greater extent, the current work cannot conclusively determine that lower intensities are ineffective, as methodological factors may have limited the capacity to produce measurable adaptations.

Contrary to expectations, pectoralis major stiffness and PML remained unchanged following all stretching conditions, and pectoralis minor stiffness decreased following only low and moderate intensity pectoral SS, which did not result in increases in haROM. Taken together, this suggests that acute improvements in haROM are likely mediated by neural adaptations such as increased stretch tolerance rather than structural alterations to muscle tissue. Similarly, changes in FSP were inconsistent and not unique to SS conditions, suggesting that shoulder posture is influenced by factors beyond pectoralis minor length and flexibility alone.

At the individual level, substantial variability in response to SS was observed, with 57% of participants classified as non-responders following moderate intensity pectoral SS and 30% following maximal intensity SS. These findings emphasize the importance of individualized prescription and monitoring when using SS clinically, as uniform protocols may fail to produce consistent outcomes across individuals.

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Collectively, the results of the current work highlight that while pectoral SS can acutely enhance shoulder mobility when performed at sufficient intensity, its effects on stiffness, muscle length, and posture are limited and highly variable. Future research should aim to identify the mechanisms and predictors of responsiveness to SS, investigate the effects of repeated or long-term stretching interventions, and explore these relationships in a variety of populations such as in different age groups and in clinical populations.

References

1. Chaabene H, Behm DG, Negra Y, Granacher U. Acute effects of static stretching on muscle strength and power: An attempt to clarify previous caveats. *Front Physiol.* 2019;10(November). doi:10.3389/fphys.2019.01468
2. Behm DG, Kay AD, Trajano GS, Alizadeh S, Blazevich AJ. Effects of stretching on injury risk reduction and balance. *J Clin Exerc Physiol.* 2021;10(3):106-116. doi:10.31189/2165-6193-10.3.106
3. Bryant J, Cooper DJ, Peters DM, Cook MD. The effects of static stretching intensity on range of motion and strength: A systematic review. *J Funct Morphol Kinesiol.* 2023;8(2):37. doi:10.3390/jfmk8020037
4. Behm DG, Blazevich AJ, Kay AD, McHugh M. Acute effects of muscle stretching on physical performance, range of motion, and injury incidence in healthy active individuals: a systematic review. *Appl Physiol Nutr Metab.* 2016;41(1):1-11. doi:10.1139/apnm-2015-0235
5. Behm DG, Alizadeh S, Daneshjoo A, et al. Acute effects of various stretching techniques on range of motion: A systematic review with meta-analysis. *Sport Med - Open.* 2023;9(1):107. doi:10.1186/s40798-023-00652-x
6. Weppeler CH, Magnusson SP. Increasing muscle extensibility: A matter of increasing length or modifying sensation? *Phys Ther.* 2010;90(3):438-449. doi:10.2522/ptj.20090012
7. Hatano G, Suzuki S, Matsuo S, et al. Hamstring stiffness returns more rapidly after static stretching than range of motion, stretch tolerance, and isometric peak torque. *J Sport Rehabil.* 2019;28(4):325-331. doi:10.1123/jsr.2017-0203
8. Magnusson SP, Simonsen EB, Aagaard P, Sørensen H, Kjaer M. A mechanism for altered

- flexibility in human skeletal muscle. *J Physiol.* 1996;497(1):291-298.
doi:10.1113/jphysiol.1996.sp021768
9. Brusco CM, Blazeovich AJ, Pinto RS. The effects of 6 weeks of constant-angle muscle stretching training on flexibility and muscle function in men with limited hamstrings' flexibility. *Eur J Appl Physiol.* 2019;119(8):1691-1700. doi:10.1007/s00421-019-04159-w
10. Medeiros DM, Martini TF. Chronic effect of different types of stretching on ankle dorsiflexion range of motion: Systematic review and meta-analysis. *Foot.* 2018;34(September 2017):28-35. doi:10.1016/j.foot.2017.09.006
11. Chagas MH, Magalhães FA, Peixoto GHC, Pereira BM, Andrade AGP, Menzel H-JK. Exploratory factor analysis for differentiating sensory and mechanical variables related to muscle-tendon unit elongation. *Brazilian J Phys Ther.* 2016;20(3):240-247.
doi:10.1590/bjpt-rbf.2014.0152
12. Kataura S, Suzuki S, Matsuo S, et al. Acute effects of the different intensity of static stretching on flexibility and isometric muscle force. *J Strength Cond Res.* 2017;31(12):3403-3410. doi:10.1519/JSC.0000000000001752
13. Bouillard, K, Nordez A, Hug F. Estimation of individual muscle force using elastography. *PLoS One.* 2011;6(12):e29261. doi: 10.1371/journal.pone.0029261
14. Creze M, Nordez A, Soubeyrand M, Rocher L, Maître X, Bellin M-F. Shear wave sonoelastography of skeletal muscle: Basic principles, biomechanical concepts, clinical applications, and future perspectives. *Skeletal Radiol.* 2018;47(4):457-471.
doi:10.1007/s00256-017-2843-y
15. Bilston LE, Tan K. Measurement of passive skeletal muscle mechanical properties in vivo: recent progress, clinical applications, and remaining challenges. *Ann Biomed Eng.*

- 2015;43(2):261-273. doi:10.1007/s10439-014-1186-2
16. Mizuno T, Matsumoto M, Umemura Y. Viscoelasticity of the muscle–tendon unit is returned more rapidly than range of motion after stretching. *Scand J Med Sci Sports*. 2013;23(1):23-30. doi:10.1111/j.1600-0838.2011.01329.x
 17. Matsuo S, Suzuki S, Iwata M, et al. Acute effects of different stretching durations on passive torque, mobility, and isometric muscle force. *J Strength Cond Res*. 2013;27(12):3367-3376. doi:10.1519/JSC.0b013e318290c26f
 18. Magnusson SP, Simonsen EB, Aagaard P, Dyhre-Poulsen P, McHugh MP, Kjaer M. Mechanical and physiological responses to stretching with and without preisometric contraction in human skeletal muscle. *Arch Phys Med Rehabil*. 1996;77(4):373-378. doi:10.1016/S0003-9993(96)90087-8
 19. Magnusson SP, Simonsen EB, Aagaard P, Sørensen H, Kjaer M. A mechanism for altered flexibility in human skeletal muscle. *J Physiol*. 1996;497(1):291-298. doi:10.1113/jphysiol.1996.sp021768
 20. Halbertsma JPK, van Bolhuis AI, Göeken LNH. Sport stretching: Effect on passive muscle stiffness of short hamstrings. *Arch Phys Med Rehabil*. 1996;77(7):688-692. doi:10.1016/S0003-9993(96)90009-X
 21. Mchugh MP, Nesse M. Effect of stretching on strength loss and pain after eccentric exercise. *Med Sci Sport Exerc*. 2008;40(3):566-573. doi:10.1249/MSS.0b013e31815d2f8c
 22. Herda TJ, Cramer JT, Ryan ED, McHugh MP, Stout JR. Acute effects of static versus dynamic stretching on isometric peak torque, electromyography, and mechanomyography of the biceps femoris muscle. *J Strength Cond Res*. 2008;22(3):809-817. doi:10.1519/JSC.0b013e31816a82ec

23. Ruan M, Li L, Chen C, Wu X. Stretch could reduce hamstring injury risk during sprinting by right shifting the length-torque curve. *J Strength Cond Res.* 2018;32(8):2190-2198. doi:10.1519/JSC.0000000000002645
24. Balle SS, Magnusson SP, McHugh MP. Effects of contract-relax vs static stretching on stretch-induced strength loss and length–tension relationship. *Scand J Med Sci Sports.* 2015;25(6):764-769. doi:10.1111/sms.12399
25. Takeuchi K, Nakamura M. Influence of high intensity 20-second static stretching on the flexibility and strength of hamstrings. *J Sports Sci Med.* 2020;19(2):429-435.
26. Weir DE, Tingley J, Elder GCB. Acute passive stretching alters the mechanical properties of human plantar flexors and the optimal angle for maximal voluntary contraction. *Eur J Appl Physiol.* 2005;93(5-6):614-623. doi:10.1007/s00421-004-1265-4
27. Warneke K, Blazevich AJ, Jochum D, et al. Perception - based methods and beyond : a current opinion on how to assess static stretching intensity. *Sport Med.* 2025;(0123456789). doi:10.1007/s40279-025-02307-1
28. Garber CE, Blissmer B, Deschenes MR, et al. Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults. *Med Sci Sport Exerc.* 2011;43(7):1334-1359. doi:10.1249/MSS.0b013e318213fefb
29. Apostolopoulos N, Metsios GS, Flouris AD, Koutedakis Y, Wyon MA. The relevance of stretch intensity and position—a systematic review. *Front Psychol.* 2015;6(August):239-244. doi:10.3389/fpsyg.2015.01128
30. Freitas SR, Vilarinho D, Vaz JR, et al. Responses to static stretching are dependent on stretch intensity and duration. *Clin Physiol Funct Imaging.* 2015;35(6):478-484.

doi:10.1111/cpf.12186

31. Freitas S, Vaz J, Bruno P, Andrade R, Mil-Homens P. Stretching effects: High-intensity & moderate-duration vs. low-intensity & long-duration. *Int J Sports Med.* 2015;37(03):239-244. doi:10.1055/s-0035-1548946
32. Fukaya T, Kiyono R, Sato S, et al. Effects of static stretching with high-intensity and short-duration or low-intensity and long-duration on range of motion and muscle stiffness. *Front Physiol.* 2020;11(November). doi:10.3389/fphys.2020.601912
33. Nakamura M, Sato S, Murakami Y, et al. The comparison of different stretching intensities on the range of motion and muscle stiffness of the quadriceps muscles. *Front Physiol.* 2021;11(January):1-7. doi:10.3389/fphys.2020.628870
34. Oba K, Samukawa M, Nakamura K, et al. Influence of constant torque stretching at different stretching intensities on flexibility and mechanical properties of plantar flexors. *J Strength Cond Res.* 2021;35(3):709-714. doi:10.1519/JSC.0000000000002767
35. Melo RRV, Cerqueira MS, Barbosa GM, et al. Static stretching at pain-tolerated intensity is not necessary to increase knee range of motion in amateur soccer players: a randomized trial. *Muscle Ligaments Tendons J.* 2021;11(03):536. doi:10.32098/mltj.03.2021.19
36. Takeuchi K, Sato S, Kiyono R, et al. High-intensity static stretching in quadriceps is affected more by its intensity than its duration. *Front Physiol.* 2021;12(July):1-7. doi:10.3389/fphys.2021.709655
37. Muanjai P, Jones DA, Mickevicius M, et al. The acute benefits and risks of passive stretching to the point of pain. *Eur J Appl Physiol.* 2017;117(6):1217-1226. doi:10.1007/s00421-017-3608-y
38. Marchetti PH, Miyatake MMS, Magalhaes RA, et al. Different volumes and intensities of

- static stretching affect the range of motion and muscle force output in well-trained subjects. *Sport Biomech.* 2022;21(2):155-164. doi:10.1080/14763141.2019.1648540
39. Santos CX, Beltrão NB, Pirauá ALT, Durigan JLQ, Behm D, de Araújo RC. Static stretching intensity does not influence acute range of motion, passive torque, and muscle architecture. *J Sport Rehabil.* 2020;29(1):1-6. doi:10.1123/jsr.2018-0178
 40. Takeuchi K, Akizuki K, Nakamura M. Association between static stretching load and changes in the flexibility of the hamstrings. *Sci Rep.* 2021;11(1):1-7. doi:10.1038/s41598-021-01274-7
 41. Takeuchi K, Akizuki K, Nakamura M. Time course of changes in the range of motion and muscle-tendon unit stiffness of the hamstrings after two different intensities of static stretching. Cè E, ed. *PLoS One.* 2021;16(9):e0257367. doi:10.1371/journal.pone.0257367
 42. Behm DG, Kay AD, Trajano GS, Blazevich AJ. Mechanisms underlying performance impairments following prolonged static stretching without a comprehensive warm-up. *Eur J Appl Physiol.* 2021;121(1):67-94. doi:10.1007/s00421-020-04538-8
 43. Apostolopoulos NC, Metsios GS, Taunton J, Koutedakis Y, Wyon M. Acute inflammation response to stretching: a randomised trial. *Ital J Sport Rehabil Posturology.* 2015;2(4):368-381. doi:10.17385/ItaJSRP.015.3008
 44. Apostolopoulos N, Corresponding B, Nevill A. Stretch intensity vs. inflammation: a dose-dependent association? *Int J Kinesiol Sport Sci.* 2015;3(1). doi:10.7575/aiac.ijkss.v.3n.1p.27
 45. Gray H, Standring S, Ellis, Harold, Berkovitz BKB. *Gray's Anatomy: The Anatomical Basis of Clinical Practice.* 39th ed. Edinburgh: New York: Elsevier Churchill Livingstone.; 2005.

46. Veeger HEJ, van der Helm FCT. Shoulder function: The perfect compromise between mobility and stability. *J Biomech.* 2007;40(10):2119-2129.
doi:10.1016/j.jbiomech.2006.10.016
47. Mehta S, Gimbel JA, Soslowsky LJ. Etiologic and pathogenetic factors for rotator cuff tendinopathy. *Clin Sports Med.* 2003;22(4):791-812. doi:10.1016/S0278-5919(03)00012-7
48. Rosa DP, Borstad JD, Pogetti LS, Camargo PR. Effects of a stretching protocol for the pectoralis minor on muscle length, function, and scapular kinematics in individuals with and without shoulder pain. *J Hand Ther.* 2017;30(1):20-29. doi:10.1016/j.jht.2016.06.006
49. Borstad JD, Ludewig PM. Comparison of three stretches for the pectoralis minor muscle. *J Shoulder Elb Surg.* 2006;15(3):324-330. doi:10.1016/j.jse.2005.08.011
50. Umehara J, Nakamura M, Fujita K, et al. Shoulder horizontal abduction stretching effectively increases shear elastic modulus of pectoralis minor muscle. *J Shoulder Elb Surg.* 2017;26(7):1159-1165. doi:10.1016/j.jse.2016.12.074
51. Ludewig PM, Borstad JD. Effects of a home exercise programme on shoulder pain and functional status in construction workers. *Occup Environ Med.* 2003;60(11):841-849.
doi:10.1136/oem.60.11.841
52. McClure PW, Michener L a, Karduna AR. Shoulder function and 3-dimensional scapular kinematics in people with and without shoulder impingement syndrome. *Phys Ther.* 2006;86(8):1075-1090. doi:10.1093/ptj/86.8.1075
53. Lee J-H, Cynn H-S, Yoon T-L, et al. Comparison of scapular posterior tilting exercise alone and scapular posterior tilting exercise after pectoralis minor stretching on scapular alignment and scapular upward rotators activity in subjects with short pectoralis minor. *Phys Ther Sport.* 2015;16(3):255-261. doi:10.1016/j.ptsp.2015.01.002

54. Lee J-H, Cynn H, Yi C-H, Kwon O, Yoon T-L. Predictor variables for forward scapular posture including posterior shoulder tightness. *J Bodyw Mov Ther.* 2015;19(2):253-260. doi:10.1016/j.jbmt.2014.04.010
55. Borstad JD. Resting position variables at the shoulder: Evidence to support a posture-impairment association. *Phys Ther.* 2006;86(4):549-557. doi:10.1093/ptj/86.4.549
56. Borstad JD, Ludewig PM. The effect of long versus short pectoralis minor resting length on scapular kinematics in healthy individuals. *J Orthop Sport Phys Ther.* 2005;35(4):227-238. doi:10.2519/jospt.2005.35.4.227
57. Lee J, Cynn H, Yoon T, et al. The effect of scapular posterior tilt exercise, pectoralis minor stretching, and shoulder brace on scapular alignment and muscles activity in subjects with round-shoulder posture. *J Electromyogr Kinesiol.* 2015;25(1):107-114. doi:10.1016/j.jelekin.2014.10.010
58. Wong CK, Coleman D, DiPersia V, Song J, Wright D. The effects of manual treatment on rounded-shoulder posture, and associated muscle strength. *J Bodyw Mov Ther.* 2010;14(4):326-333. doi:10.1016/j.jbmt.2009.05.001
59. Gutiérrez-Espinoza H, Araya-Quintanilla F, Gutiérrez-Monclus R, et al. Does pectoralis minor stretching provide additional benefit over an exercise program in participants with subacromial pain syndrome? A randomized controlled trial. *Musculoskelet Sci Pract.* 2019;44(August):102052. doi:10.1016/j.msksp.2019.102052
60. Hasan S, Iqbal A, Alghadir AH, Alonazi A, Alyahya D. The combined effect of the trapezius muscle strengthening and pectoralis minor muscle stretching on correcting the rounded shoulder posture and shoulder flexion range of motion among young saudi females: A randomized comparative study. *Healthcare.* 2023;11(4):500.

doi:10.3390/healthcare11040500

61. Majeed A, Kayani S, Riaz S, Yasin MM, Naeem A, Mansoor SR. Comparative effectiveness of pectoralis minor stretching and rhomboids strengthening on resting position of scapula. *Rawal Med J.* 2021;46(3):576-579.
62. Umehara J, Nakamura M, Nishishita S, Tanaka H, Kusano K, Ichihashi N. Scapular kinematic alterations during arm elevation with decrease in pectoralis minor stiffness after stretching in healthy individuals. *J Shoulder Elb Surg.* 2018;27(7):1214-1220.
doi:10.1016/j.jse.2018.02.037
63. Umehara J, Nakamura M, Saeki J, et al. Acute and prolonged effects of stretching on shear modulus of the pectoralis minor muscle. *J Sport Sci Med.* 2021;20(1):17-25.
64. Dobija L, Pereira B, Dupeyron A, Coudeyre E. Comparison of clinical characteristics between responders and non-responders to hamstring stretching in individuals with chronic low back pain. *Diagnostics.* 2024;14(19):2117. doi:10.3390/diagnostics14192117
65. Masi AT, Hannon JC. Human resting muscle tone (HRMT): Narrative introduction and modern concepts. *J Bodyw Mov Ther.* 2008;12(4):320-332.
doi:10.1016/j.jbmt.2008.05.007
66. Simons GD, Mense S. Understanding and measurement of muscle tone as related to clinical muscle pain. *Pain.* 1998;75(1):1-17. doi:10.1016/S0304-3959(97)00102-4
67. Ganguly J, Kulshreshtha D, Almotiri M, Jog M. Muscle tone physiology and abnormalities. *Toxins (Basel).* 2021;13(4):282. doi:10.3390/toxins13040282
68. Usgu S, Ramazanoğlu E, Yakut Y. The relation of body mass index to muscular viscoelastic properties in normal and overweight individuals. *Medicina (B Aires).* 2021;57(10):1022. doi:10.3390/medicina57101022

69. Tunnell PW. Muscle length assessment of tightness-prone muscles. *J Bodyw Mov Ther.* 1998;2(1):21-27. doi:10.1016/S1360-8592(98)80043-0
70. Purslow PP. The structure and role of intramuscular connective tissue in muscle function. *Front Physiol.* 2020;11(May). doi:10.3389/fphys.2020.00495
71. Haff GG, Triplett NT. *Essentials of Strength Training and Conditioning 4th Edition.* 4th ed. Human kinetics; 2015.
72. Cooke R. Force generation in muscle. *Curr Opin Cell Biol.* 1990;2(1):62-66. doi:10.1016/S0955-0674(05)80032-8
73. Taylor DC, Dalton JD, Seaber A V., Garrett WE. Viscoelastic properties of muscle-tendon units. *Am J Sports Med.* 1990;18(3):300-309. doi:10.1177/036354659001800314
74. McHugh M, Magnusson S, Gleim G, Nicholas JA. Viscoelastic stress relaxation in human skeletal muscle. *Med Sci Sport Exerc.* 1992;12(24):1375-1382. <https://europepmc.org/article/med/1470021>.
75. Ryan ED, Herda TJ, Costa PB, et al. Viscoelastic creep in the human skeletal muscle-tendon unit. *Eur J Appl Physiol.* 2010;108(1):207-211. doi:10.1007/s00421-009-1284-2
76. Ikeda N, Yonezu T, Kawakami Y. Minute oscillation stretching: A novel modality for reducing musculo-tendinous stiffness and maintaining muscle strength. *Scand J Med Sci Sports.* 2021;31(1):104-114. doi:10.1111/sms.13830
77. Herbert RD, Bolsterlee B, Gandevia SC. Passive changes in muscle length. *J Appl Physiol.* 2019;126(5):1445-1453. doi:10.1152/japplphysiol.00673.2018
78. Wolfram S, Lipps DB. The in vivo passive stretch response of the pectoralis major is region-specific. *J Biomech.* 2023;161(October):111856. doi:10.1016/j.jbiomech.2023.111856

79. Gennisson J-L, Deffieux T, Fink M, Tanter M. Ultrasound elastography: Principles and techniques. *Diagn Interv Imaging*. 2013;94(5):487-495. doi:10.1016/j.diii.2013.01.022
80. Drakonaki E. Ultrasound elastography for imaging tendons and muscles. *J Ultrason*. 2012;12(49):214-225. doi:10.15557/JoU.2012.0008
81. Melo ASC, Cruz EB, Vilas-Boas JP, Sousa ASP. Scapular dynamic muscular stiffness assessed through myotonometry: A narrative review. *Sensors*. 2022;22(7):2565. doi:10.3390/s22072565
82. Page P. Current concepts in muscle stretching for exercise and rehabilitation. *Int J Sports Phys Ther*. 2012;7(1):109-119.
83. Lima CD, Ruas C V., Behm DG, Brown LE. Acute effects of stretching on flexibility and performance: A narrative review. *J Sci Sport Exerc*. 2019;1(1):29-37. doi:10.1007/s42978-019-0011-x
84. Arntz F, Markov A, Behm DG, et al. Chronic effects of static stretching exercises on muscle strength and power in healthy individuals across the lifespan: a systematic review with multi-level meta-analysis. *Sport Med*. 2023;53(3):723-745. doi:10.1007/s40279-022-01806-9
85. Harvey L, Herbert R, Crosbie J. Does stretching induce lasting increases in joint ROM? A systematic review. *Physiother Res Int*. 2002;7(1):1-13. doi:10.1002/pri.236
86. Shah R, Samuel MW, Son J. Acute and chronic effects of static stretching on neuromuscular properties: A meta-analytical review. *Appl Sci*. 2023;13(21):11979. doi:10.3390/app132111979
87. Witvrouw E, Mahieu N, Danneels L, McNair P. Stretching and injury prevention. *Sport Med*. 2004;34(7):443-449. doi:10.2165/00007256-200434070-00003

88. Bradley PS, Portas MD. The relationship between preseason range of motion and muscle strain injury in elite soccer players. *J Strength Cond Res.* 2007;21(4):1155. doi:10.1519/R-20416.1
89. Swain DP. *ACSM's Resource Manual for Guidelines for Exercise Testing and Prescription.* Seventh ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2014.
90. Sands WA, McNeal JR, Murray SR, et al. Stretching and its effects on recovery. *Strength Cond J.* 2013;35(5):30-36. doi:10.1519/SSC.0000000000000004
91. Afonso J, Clemente FM, Nakamura FY, et al. The effectiveness of post-exercise stretching in short-term and delayed recovery of strength, range of motion and delayed onset muscle soreness: A systematic review and meta-analysis of randomized controlled trials. *Front Physiol.* 2021;12:677581. doi:https://dx.doi.org/10.3389/fphys.2021.677581
92. Henschke N, Lin CC. Stretching before or after exercise does not reduce delayed-onset muscle soreness. *Br J Sports Med.* 2011;45(15):1249-1250. doi:10.1136/bjsports-2011-090599
93. Herbert RD. Effects of stretching before and after exercising on muscle soreness and risk of injury: systematic review. *BMJ.* 2002;325(7362):468-468. doi:10.1136/bmj.325.7362.468
94. Riccetti M, Opplert J, Durigan JLQ, Cometti C, Babault N. Acute Static stretching results in muscle-specific alterations amongst the hamstring muscles. *Sports.* 2020;8(9):119. doi:10.3390/sports8090119
95. Guissard N, Duchateau J. Neural aspects of muscle stretching. *Exerc Sport Sci Rev.* 2006;34(4):154-158. doi:10.1249/01.jes.0000240023.30373.eb

96. Moore MA, Hutton RS. Electromyographic investigation of muscle stretching techniques. *Med Sci Sport Exerc.* 1980;12(5):322-329. doi:10.1249/00005768-198025000-00004
97. Avela J, Finni T, Liikavainio T, Niemelä E, Komi P V. Neural and mechanical responses of the triceps surae muscle group after 1 h of repeated fast passive stretches. *J Appl Physiol.* 2004;96(6):2325-2332. doi:10.1152/japplphysiol.01010.2003
98. Winn N, Lalam R, Cassar-Pullicino V. Sonoelastography in the musculoskeletal system: Current role and future directions. *World J Radiol.* 2016;8(11):868. doi:10.4329/wjr.v8.i11.868
99. Voerman G, Gregorič M, Hermens H. Neurophysiological methods for the assessment of spasticity: The Hoffmann reflex, the tendon reflex, and the stretch reflex. *Disabil Rehabil.* 2005;27(1-2):33-68. doi:10.1080/09638280400014600
100. Guissard N, Duchateau J, Hainaut K. Mechanisms of decreased motoneurone excitation during passive muscle stretching. *Exp Brain Res.* 2001;137(2):163-169. doi:10.1007/s002210000648
101. Nakamura M, Ikezoe T, Takeno Y, Ichihashi N. Time course of changes in passive properties of the gastrocnemius muscle–tendon unit during 5 min of static stretching. *Man Ther.* 2013;18(3):211-215. doi:10.1016/j.math.2012.09.010
102. Takeuchi K, Nakamura M, Fukaya T, Konrad A, Mizuno T. Acute and long-term effects of static stretching on muscle-tendon unit stiffness: A systematic review and meta-analysis. *J Sport Sci Med.* 2023;(June):465-475. doi:10.52082/jssm.2023.465
103. Konrad A, Tilp M. The acute time course of muscle and tendon tissue changes following one minute of static stretching. *Curr Issues Sport Sci.* 2020;5:1-7. doi:10.15203/CISS_2020.003

104. Ingram LA, Tomkinson GR, D'Unienville NMA, et al. mechanisms underlying range of motion improvements following acute and chronic static stretching: A systematic review, meta-analysis and multivariate meta-regression. *Sport Med.* 2025;55(6):1449-1466. doi:10.1007/s40279-025-02204-7
105. Pei-Hsin Kuo, Deshpande AD. Contribution of passive properties of muscle-tendon units to the metacarpophalangeal joint torque of the index finger. In: *2010 3rd IEEE RAS & EMBS International Conference on Biomedical Robotics and Biomechatronics*. IEEE; 2010:288-294. doi:10.1109/BIOROB.2010.5626918
106. Eby SF, Song P, Chen S, Chen Q, Greenleaf JF, An K-N. Validation of shear wave elastography in skeletal muscle. *J Biomech.* 2013;46(14):2381-2387. doi:10.1016/j.jbiomech.2013.07.033
107. Voglar M, Vatovec R, Kozinc Ž, Šarabon N. The effects of eccentric exercise on passive hamstring muscle stiffness: Comparison of shear-wave elastography and passive knee torque outcomes. *Eur J Transl Myol.* 2022;32(2). doi:10.4081/ejtm.2022.10567
108. Nojiri S, Yagi M, Mizukami Y, Ichihashi N. Static stretching time required to reduce iliacus muscle stiffness. *Sport Biomech.* 2021;20(7):901-910. doi:10.1080/14763141.2019.1620321
109. Kusano K, Nishishita S, Nakamura M, Tanaka H, Umehara J, Ichihashi N. Acute effect and time course of extension and internal rotation stretching of the shoulder on infraspinatus muscle hardness. *J Shoulder Elb Surg.* 2017;26(10):1782-1788. doi:10.1016/j.jse.2017.04.018
110. Umegaki H, Ikezoe T, Nakamura M, et al. Acute effects of static stretching on the hamstrings using shear elastic modulus determined by ultrasound shear wave

- elastography: Differences in flexibility between hamstring muscle components. *Man Ther.* 2015;20(4):610-613. doi:10.1016/j.math.2015.02.006
111. Nakao G, Taniguchi K, Katayose M. Correction: Acute effect of active and passive static stretching on elastic modulus of the hamstrings. *Sport Med Int Open.* 2018;02(06):E200-E200. doi:10.1055/a-0822-3474
112. Nakamura M, Ikezoe T, Kobayashi T, et al. Acute effects of static stretching on muscle hardness of the medial gastrocnemius muscle belly in humans: An ultrasonic shear-wave elastography study. *Ultrasound Med Biol.* 2014;40(9):1991-1997. doi:10.1016/j.ultrasmedbio.2014.03.024
113. Taniguchi K, Shinohara M, Nozaki S, Katayose M. Acute decrease in the stiffness of resting muscle belly due to static stretching. *Scand J Med Sci Sports.* 2015;25(1):32-40. doi:10.1111/sms.12146
114. Nakamura M, Takeuchi K, Fukaya T, Nakao G, Konrad A, Mizuno T. Acute effects of static stretching on passive stiffness in older adults: A systematic review and meta-analysis. *Arch Gerontol Geriatr.* 2024;117(October 2023):105256. doi:10.1016/j.archger.2023.105256
115. Fowles JR, Sale DG, MacDougall JD. Reduced strength after passive stretch of the human plantarflexors. *J Appl Physiol.* 2000;89(3):1179-1188. doi:10.1152/jappl.2000.89.3.1179
116. Ryan ED, Beck TW, Herda TJ, et al. Do practical durations of stretching alter muscle strength? A dose-response study. *Med Sci Sport Exerc.* 2008;40(8):1529-1537. doi:10.1249/MSS.0b013e31817242eb
117. Behm DG. *The science and physiology of flexibility and stretching: Implications and applications in sport performance and health.* Routledge. 2024

118. Copay AG, Eyberg B, Chung AS, Zurcher KS, Chutkan N, Spangehl MJ. Minimum clinically important difference: Current trends in the orthopaedic literature, part II: Lower extremity. *JBJS Rev.* 2018;6(9):e2-e2. doi:10.2106/JBJS.RVW.17.00160
119. Dobija L, Pereira B, Cohen-Aknine G, Roren A, Dupeyron A, Coudeyre E. Immediate effect of passive hamstring stretching on flexibility and relationship with psychosocial factors in people with chronic low back pain. *Heliyon.* 2023;9(9):e19753. doi:10.1016/j.heliyon.2023.e19753
120. Atkinson G, Williamson P, Batterham AM. Issues in the determination of ‘responders’ and ‘non-responders’ in physiological research. *Exp Physiol.* 2019;104(8):1215-1225. doi:10.1113/EP087712
121. Herold F, Müller P, Gronwald T, Müller NG. Dose–Response Matters! – A Perspective on the exercise prescription in exercise–cognition research. *Front Psychol.* 2019;10(November):1-17. doi:10.3389/fpsyg.2019.02338
122. Buford TW, Pahor M. Making preventive medicine more personalized: Implications for exercise-related research. *Prev Med (Baltim).* 2012;55(1):34-36. doi:10.1016/j.ypmed.2012.05.001
123. Booth FW, Laye MJ. The future: genes, physical activity and health. *Acta Physiol.* 2010;199(4):549-556. doi:10.1111/j.1748-1716.2010.02117.x
124. Swinton PA, Hemingway BS, Saunders B, Gualano B, Dolan E. A statistical framework to interpret individual response to intervention: Paving the way for personalized nutrition and exercise prescription. *Front Nutr.* 2018;5(May). doi:10.3389/fnut.2018.00041
125. Pickering C, Kiely J. Do non-responders to exercise exist—and if so, what should we do about them? *Sport Med.* 2019;49(1):1-7. doi:10.1007/s40279-018-01041-1

126. Montero D, Lundby C. Refuting the myth of non-response to exercise training: ‘non-responders’ do respond to higher dose of training. *J Physiol.* 2017;595(11):3377-3387. doi:10.1113/JP273480
127. Hopkins WG. Measures of reliability in sports medicine and science. *Sport Med.* 2000;30(1):1-15. doi:10.2165/00007256-200030010-00001
128. Ferreira MLV, Castro A, de Oliveira Nunes SG, et al. Hypotensive effects of exercise training: are postmenopausal women with hypertension non-responders or responders? *Hypertens Res.* 2024;47(8):2172-2182. doi:10.1038/s41440-024-01721-8
129. Domaradzki J, Koźlenia D. Cardiovascular and cardiorespiratory effects of high-intensity interval training in body fat responders and non-responders. *Sci Rep.* 2024;14(1):14631. doi:10.1038/s41598-024-65444-z
130. Alvarez C, Ramírez-Campillo R, Ramírez-Vélez R, Izquierdo M. Effects of 6-weeks high-intensity interval training in schoolchildren with insulin resistance: Influence of biological maturation on metabolic, body composition, cardiovascular and performance non-responses. *Front Physiol.* 2017;8(JUN). doi:10.3389/fphys.2017.00444
131. Bonafiglia JT, Rotundo MP, Whittall JP, Scribbans TD, Graham RB, Gurd BJ. Inter-Individual variability in the adaptive responses to endurance and sprint interval training: a randomized crossover study. Calbet JAL, ed. *PLoS One.* 2016;11(12):e0167790. doi:10.1371/journal.pone.0167790
132. Gurd BJ, Giles MD, Bonafiglia JT, et al. Incidence of nonresponse and individual patterns of response following sprint interval training. *Appl Physiol Nutr Metab.* 2016;41(3):229-234. doi:10.1139/apnm-2015-0449
133. Domaradzki J, Koźlenia D, Popowczak M. The prevalence of responders and non-

- responders for body composition, resting blood pressure, musculoskeletal, and cardiorespiratory fitness after ten weeks of school-based high-intensity interval training in adolescents. *J Clin Med*. 2023;12(13):4204. doi:10.3390/jcm12134204
134. Ross R, de Lannoy L, Stotz PJ. Separate effects of intensity and amount of exercise on interindividual cardiorespiratory fitness response. *Mayo Clin Proc*. 2015;90(11):1506-1514. doi:10.1016/j.mayocp.2015.07.024
 135. Bouchard C, Blair SN, Church TS, et al. Adverse metabolic response to regular exercise: is it a rare or common occurrence? Li S, ed. *PLoS One*. 2012;7(5):e37887. doi:10.1371/journal.pone.0037887
 136. Bagger M, Petersen PH, Pedersen PK. Biological variation in variables associated with exercise training. *Int J Sports Med*. 2003;24(6):433-440. doi:10.1055/s-2003-41180
 137. Stanforth PR, Gagnon J, Rice T, et al. Reproducibility of resting blood pressure and heart rate measurements. *Ann Epidemiol*. 2000;10(5):271-277. doi:10.1016/S1047-2797(00)00047-8
 138. Scharhag-Rosenberger F, Walitzek S, Kindermann W, Meyer T. Differences in adaptations to 1 year of aerobic endurance training: individual patterns of nonresponse. *Scand J Med Sci Sports*. 2012;22(1):113-118. doi:10.1111/j.1600-0838.2010.01139.x
 139. Karavirta L, Häkkinen K, Kauhanen A, et al. Individual responses to combined endurance and strength training in older adults. *Med Sci Sport Exerc*. 2011;43(3):484-490. doi:10.1249/MSS.0b013e3181f1bf0d
 140. Ahtiainen JP, Walker S, Peltonen H, et al. Heterogeneity in resistance training-induced muscle strength and mass responses in men and women of different ages. *Age (Omaha)*. 2016;38(1):10. doi:10.1007/s11357-015-9870-1

141. Erskine RM, Jones DA, Williams AG, Stewart CE, Degens H. Resistance training increases in vivo quadriceps femoris muscle specific tension in young men. *Acta Physiol.* 2010;199(1):83-89. doi:10.1111/j.1748-1716.2010.02085.x
142. Davidsen PK, Gallagher IJ, Hartman JW, et al. High responders to resistance exercise training demonstrate differential regulation of skeletal muscle microRNA expression. *J Appl Physiol.* 2011;110(2):309-317. doi:10.1152/jappphysiol.00901.2010
143. Parr EB, Camera DM, Burke LM, Phillips SM, Coffey VG, Hawley JA. Circulating MicroRNA Responses between ‘high’ and ‘low’ responders to a 16-wk diet and exercise weight loss intervention. Song Y, ed. *PLoS One.* 2016;11(4):e0152545. doi:10.1371/journal.pone.0152545
144. Galaasen Bakken A, Eklund A, Oksanen A, Axén I. The response to individualized treatment after a standardized treatment protocol among neck pain sufferers: a secondary analysis of a randomized controlled trial. *Chiropr Man Therap.* 2025;33(1):13. doi:10.1186/s12998-025-00579-y
145. Karlsson L, Takala E-P, Gerdle B, Larsson B. Evaluation of pain and function after two home exercise programs in a clinical trial on women with chronic neck pain - with special emphasises on completers and responders. *BMC Musculoskelet Disord.* 2014;15(1):6. doi:10.1186/1471-2474-15-6
146. Bloom DA, Kaplan DJ, Mojica E, et al. The minimal clinically important difference: a review of clinical significance. *Am J Sports Med.* 2023;51(2):520-524. doi:10.1177/03635465211053869
147. Hays RD, Peipert JD. Between-group minimally important change versus individual treatment responders. *Qual Life Res.* 2021;30(10):2765-2772. doi:10.1007/s11136-021-

02897-z

148. Cook JA, Julious SA, Sones W, et al. DELTA2 guidance on choosing the target difference and undertaking and reporting the sample size calculation for a randomised controlled trial. *Trials*. 2018;19(1):606. doi:10.1186/s13063-018-2884-0
149. Dankel SJ, Loenneke JP. A method to stop analyzing random error and start analyzing differential responders to exercise. *Sport Med*. 2020;50(2):231-238. doi:10.1007/s40279-019-01147-0
150. Atkinson G, Batterham AM. True and false interindividual differences in the physiological response to an intervention. *Exp Physiol*. 2015;100(6):577-588. doi:10.1113/EP085070
151. Morais N, Cruz J. The pectoralis minor muscle and shoulder movement-related impairments and pain: Rationale, assessment and management. *Phys Ther Sport*. 2016;17:1-13. doi:10.1016/j.ptsp.2015.10.003
152. Fani M, Ebrahimi S, Ghanbari A. Evaluation of scapular mobilization and comparison to pectoralis minor stretching in individuals with rounded shoulder posture: A randomized controlled trial. *J Bodyw Mov Ther*. 2020;24(4):367-372. doi:10.1016/j.jbmt.2020.07.021
153. Kay AD, Husbands-Beasley J, Blazeovich AJ. Effects of contract-relax, static stretching, and isometric contractions on muscle-tendon mechanics. *Med Sci Sport Exerc*. 2015;47(10):2181-2190. doi:10.1249/MSS.0000000000000632
154. Tu Y, Gilthorpe MS. Revisiting the relation between change and initial value: a review and evaluation. *Stat Med*. 2007;26(2):443-457. doi:10.1002/sim.2538
155. Snider SE, Quisenberry AJ, Bickel WK. Order in the absence of an effect: Identifying rate-dependent relationships. *Behav Processes*. 2016;127:18-24.

doi:10.1016/j.beproc.2016.03.012

156. Sasaki K, Toyama S, Ishii N. Length-force characteristics of in vivo human muscle reflected by supersonic shear imaging. *J Appl Physiol.* 2014;117(2):153-162.
doi:10.1152/jappphysiol.01058.2013
157. Mullaney MJ, McHugh MP, Johnson CP, Tyler TF. Reliability of shoulder range of motion comparing a goniometer to a digital level. *Physiother Theory Pract.* 2010;26(5):327-333. doi:10.3109/09593980903094230
158. Prentice WE, Arnheim D. *Principles of Athletic Training: A Competency-Based Approach.* McGraw-Hill New York, NY, USA.; 2011.
159. Laudner K, Thorson K. Acute effects of pectoralis minor self-mobilization on shoulder motion and posture: A blinded and randomized placebo-controlled study in asymptomatic individuals. *J Sport Rehabil.* 2020;29(4):420-424. doi:10.1123/jsr.2018-0220
160. Bohunicky S, Rutherford L, Harrison K-L, Malone Q, Glazebrook CM, Scribbans TD. Immediate effects of myofascial release to the pectoral fascia on posture, range of motion, and muscle excitation: A crossover randomized clinical trial. *J Man Manip Ther.* 2024;00(00):1-11. doi:10.1080/10669817.2024.2316414
161. Roman J. Short term effects of a pectoralis minor positional release in collegiate swimmers. *Clin Pract Athl Train.* 2018;1(2):42-48. doi:10.31622/2018/0002.7
162. Williams JG, Laudner KG, McLoda T. The acute effects of two passive stretch maneuvers on pectoralis minor length and scapular kinematics among collegiate swimmers. *Int J Sports Phys Ther.* 2013;8(1):25-33.
163. Muraki T, Aoki M, Izumi T, Fujii M, Hidaka E. Lengthening of the pectoralis minor motions and stretching techniques. *Phys Ther.* 2009;89(4):333-341.

164. Stiver ML, Mirjalili SA, Agur AMR. Measuring shear wave velocity in adult skeletal muscle with ultrasound 2-d shear wave elastography: A scoping review. *Ultrasound Med Biol.* 2023;49(6):1353-1362. doi:10.1016/j.ultrasmedbio.2023.02.005
165. Yoo W. Comparison of shoulder muscles activation for shoulder abduction between forward shoulder posture and asymptomatic persons. *J Phys Ther Sci.* 2013;25(7):815-816. doi:10.1589/jpts.25.815
166. Reiner M, Gabriel A, Sommer D, Bernsteiner D, Tilp M, Konrad A. Effects of a high-volume 7-week pectoralis muscle stretching training on muscle function and muscle stiffness. *Sport Med - Open.* 2023;9(1):40. doi:10.1186/s40798-023-00582-8
167. Wohllann T, Warneke K, Kalder V, Behm DG, Schmidt T, Schiemann S. Influence of 8-weeks of supervised static stretching or resistance training of pectoral major muscles on maximal strength, muscle thickness and range of motion. *Eur J Appl Physiol.* January 2024. doi:10.1007/s00421-023-05413-y
168. Panidi I, Donti O, Konrad A, et al. Muscle architecture adaptations to static stretching training: A systematic review with meta-analysis. *Sport Med - Open.* 2023;9(1):47. doi:10.1186/s40798-023-00591-7
169. Miyazaki M, Maeda S. Changes in hamstring flexibility and muscle strength during the menstrual cycle in healthy young females. *J Phys Ther Sci.* 2022;34(2):2021-2147. doi:10.1589/jpts.34.92
170. Lim W, Park H. No significant correlation between the intensity of static stretching and subject's perception of pain. *J Phys Ther Sci.* 2017;29(10):1856-1859. doi:10.1589/jpts.29.1856
171. Medeiros DM, Cini A, Sbruzzi G, Lima CS. Influence of static stretching on hamstring

- flexibility in healthy young adults: Systematic review and meta-analysis. *Physiother Theory Pract.* 2016;32(6):438-445. doi:10.1080/09593985.2016.1204401
172. Konrad A, Budini F, Tilp M. Acute effects of constant torque and constant angle stretching on the muscle and tendon tissue properties. *Eur J Appl Physiol.* 2017;117(8):1649-1656. doi:10.1007/s00421-017-3654-5
173. Blazeovich AJ. Adaptations in the passive mechanical properties of skeletal muscle to altered patterns of use. *J Appl Physiol.* 2019;126(5):1483-1491. doi:10.1152/japplphysiol.00700.2018
174. McHugh MP, Cosgrave CH. To stretch or not to stretch: The role of stretching in injury prevention and performance. *Scand J Med Sci Sports.* 2009;20(2):169-181. doi:10.1111/j.1600-0838.2009.01058.x
175. Takeuchi K, Nakamura M. The optimal duration of high-intensity static stretching in hamstrings. Lavender AP, ed. *PLoS One.* 2020;15(10):e0240181. doi:10.1371/journal.pone.0240181
176. Freitas SR, Vaz JR, Gomes L, et al. A new tool to assess the perception of stretching intensity. *J Strength Cond Res.* 2015;29(9):2666-2678. doi:10.1519/JSC.0000000000000926
177. Cook RJ, Dickens BM, Fathalla MF. World medical association declaration of helsinki: ethical principles for medical research involving human subjects. In: *Reproductive Health and Human Rights.* Vol 81. Oxford University Press; 2003:428-432. doi:10.1093/acprof:oso/9780199241323.003.0025
178. Milenkovic S, Dragovic M. Modification of the Edinburgh Handedness Inventory: A replication study. *Laterality Asymmetries Body, Brain Cogn.* 2013;18(3):340-348.

doi:10.1080/1357650X.2012.683196

179. Peterson DE, Blankenship KR, Robb B, et al. Investigation of the validity and reliability of four objective techniques for forward shoulder. *JOSPT J Orthop Sport Phys Ther.* 1997;25.
180. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. 2nd ed. Hillsdale, N.J.: L. Erlbaum Associates; 1988.
181. Portney LG. *Foundations of Clinical Research : Applications to Practice*. 3rd editio. Philadelphia: F.A. Davis Company
182. Fukaya T, Matsuo S, Iwata M, et al. Acute and chronic effects of static stretching at 100% versus 120% intensity on flexibility. *Eur J Appl Physiol.* 2021;121(2):513-523.
doi:10.1007/s00421-020-04539-7
183. Law RY, Harvey LA, Nicholas MK, Tonkin L, De Sousa M, Finniss DG. Stretch exercises increase tolerance to stretch in patients with chronic musculoskeletal pain: a randomized controlled trial. *Physical Therapy.* 2009 Oct 1;89(10):1016-26.
184. Folpp H, Deall S, Harvey LA, Gwinn T. Can apparent increases in muscle extensibility with regular stretch be explained by changes in tolerance to stretch? *Aust J Physiother.* 2006;52(1):45-50. doi:10.1016/S0004-9514(06)70061-7
185. Hoge KM, Ryan ED, Costa PB, et al. Gender differences in musculotendinous stiffness and range of motion after an acute bout of stretching. *J Strength Cond Res.* 2010;24(10):2618-2626. doi:10.1519/JSC.0b013e3181e73974
186. Mizuno T. Is there a sex difference in the minimum number of static stretch repetitions that should be recommended to improve flexibility? *Int J Sport Heal Sci.* 2019;17(0):80-87. doi:10.5432/ijshs.201836

187. Franklin R, Bohunicky S, Wang S, Scribbans T. Pectoral muscle stiffness, length, and range of motion does not differ between males and females following static pectoral stretching at different intensities. *Can J Appl Physiol.* 2025;20(4):S79. doi:10.1139/h95-041
188. Miyamoto N, Hirata K, Miyamoto-Mikami E, Yasuda O, Kanehisa H. Associations of passive muscle stiffness, muscle stretch tolerance, and muscle slack angle with range of motion: individual and sex differences. *Sci Rep.* 2018;8(1):8274. doi:10.1038/s41598-018-26574-3
189. Freitas SR, Andrade RJ, Larcoupaille L, Mil-homens P, Nordez A. Muscle and joint responses during and after static stretching performed at different intensities. *Eur J Appl Physiol.* 2015;115(6):1263-1272. doi:10.1007/s00421-015-3104-1
190. Hirata K, Miyamoto-Mikami E, Kanehisa H, Miyamoto N. Muscle-specific acute changes in passive stiffness of human triceps surae after stretching. *Eur J Appl Physiol.* 2016;116(5):911-918. doi:10.1007/s00421-016-3349-3
191. Zhou J, Liu C, Zhang Z. Non-uniform stiffness within gastrocnemius-achilles tendon complex observed after static stretching. *J Sports Sci Med.* 2019;18(3):454-461. <http://www.ncbi.nlm.nih.gov/pubmed/31427867>.
192. Akagi R, Takahashi H. Acute effect of static stretching on hardness of the gastrocnemius muscle. *Med Sci Sport Exerc.* 2013;45(7):1348-1354. doi:10.1249/MSS.0b013e3182850e17
193. Fukaya T, Morishita K, Yokoi Y, Takeuchi K, Nakamura M. Acute and chronic effects of static stretching of different target muscles on shear elastic modulus: A narrative review. *J Electromyogr Kinesiol.* 2024;79(October):102939. doi:10.1016/j.jelekin.2024.102939

194. Hirata K, Kanehisa H, Miyamoto-Mikami E, Miyamoto N. Evidence for intermuscle difference in slack angle in human triceps surae. *J Biomech.* 2015;48(6):1210-1213. doi:10.1016/j.jbiomech.2015.01.039
195. Hug F, Lacourpaille L, Maïsetti O, Nordez A. Slack length of gastrocnemius medialis and Achilles tendon occurs at different ankle angles. *J Biomech.* 2013;46(14):2534-2538. doi:10.1016/j.jbiomech.2013.07.015
196. Hug F, Tucker K, Gennisson J-L, Tanter M, Nordez A. Elastography for muscle biomechanics. *Exerc Sport Sci Rev.* 2015;43(3):125-133. doi:10.1249/JES.0000000000000049
197. Fathy A, Abdelmajeed SF, Mohamed ME. Correlation between aberrant upper body posture and shoulder function in subacromial impingement syndrome. *Egypt J Hosp Med.* 2023;91(1):5045-5053. doi:10.21608/ejhm.2023.303623
198. Ludewig PM, Reynolds JF. The association of scapular kinematics and glenohumeral joint pathologies. *J Orthop Sport Phys Ther.* 2009;39(2):90-104. doi:10.2519/jospt.2009.2808
199. Higuchi T, Inoue S, Kawakami T, Kawamura K, Yokoyama S. Effects of two pectoralis minor stretching methods on pectoralis minor length, scapular position and glenohumeral range of motion. 2015;101:560-561.
200. Roddey TS, Olson SL, Grant SE. The effect of pectoralis muscle stretching on the resting position of the scapula in persons with varying degrees of forward head/rounded shoulder posture. *J Man Manip Ther.* 2002;10(3):124-128. doi:10.1179/106698102790819247
201. Lutz A, Thigpen C. What factors contribute to forward head and rounded shoulder posture, and why is it important to assess? In: *Quick Questions in the Shoulder*. New York: Routledge; 2024:3-5. doi:10.4324/9781003526148-2

202. Ellenbecker T, Roetert EP, Petracek K, Kovacs M, Barajas N, Bailie D. Bilateral comparison of anterior shoulder position in elite tennis players. *Int J Sports Phys Ther.* 2022;17(5):863-869. doi:10.26603/001c.36629
203. Dehcheshmeh TF, Majelan AS, Maleki B. Correlation between depression and posture (A systematic review). *Curr Psychol.* 2023;43(33):27251-27261. doi:10.1007/s12144-023-04630-0
204. Korooshfard N, Ramezanzade H, Arabnarmi B. Relationship of self esteem with forward head posture and round shoulder. *Procedia - Soc Behav Sci.* 2011;15:3698-3702. doi:10.1016/j.sbspro.2011.04.358
205. Batistão MV, Moreira R de FC, Coury HJCG, Salasar LEB, Sato T de O. Prevalence of postural deviations and associated factors in children and adolescents: A cross-sectional study. *Fisioter em Mov.* 2016;29(4):777-786. doi:10.1590/1980-5918.029.004.ao14
206. Maciańczyk-Paprocka K, Stawińska-Witoszyńska B, Kotwicki T, et al. Prevalence of incorrect body posture in children and adolescents with overweight and obesity. *Eur J Pediatr.* 2017;176(5):563-572. doi:10.1007/s00431-017-2873-4
207. Balthillaya M G, H S, Reddy I, et al. Comparison of Scapulohumeral rhythm in individuals with and without Forward Shoulder Posture. *F1000Research.* 2024;13:1421. doi:10.12688/f1000research.158183.1
208. Wiranatha MBK, Putra IPYP, Saraswati PAS, Kinandana GP. The relationship between upper cross syndrome posture and shoulder disability. *Phys Ther J Indones.* 2024;5(2):137-141. doi:10.51559/ptji.v5i2.209
209. Greenfield B, Catlin PA, Coats PW, Green E, McDonald JJ, North C. Posture in patients with shoulder overuse injuries and healthy individuals. *J Orthop Sport Phys Ther.*

- 1995;21(5):287-295. doi:10.2519/jospt.1995.21.5.287
210. Sarabadani Tafreshi E, Nodehi Moghadam A, Bakhshi E, Rastgar M. Comparing scapular position and scapular dyskinesis in individuals with and without rounded shoulder posture. *Phys Treat Specif Phys Ther*. 2015;5(3):127-136. doi:10.15412/j.ptj.07050302
211. Griegel-Morris P, Larson K, Mueller-Klaus K, Oatis CA. Incidence of common postural abnormalities in the cervical, shoulder, and thoracic regions and their association with pain in two age groups of healthy subjects. *Phys Ther*. 1992;72(6):425-431. doi:10.1093/ptj/72.6.425
212. Harrison AL, Barry-Greb T, Wojtowicz G. Clinical measurement of head and shoulder posture variables. *J Orthop Sports Phys Ther*. 1996;23(6):353-361. doi:10.2519/jospt.1996.23.6.353
213. Ferreira EA, Duarte M, Maldonado EP, Bersanetti AA, Marques AP. Quantitative assessment of postural alignment in young adults based on photographs of anterior, posterior, and lateral views. *J Manipulative Physiol Ther*. 2011;34(6):371-380. doi:10.1016/j.jmpt.2011.05.018
214. Sepehri S, Sheikhhoseini R, Piri H, Sayyadi P. The effect of various therapeutic exercises on forward head posture, rounded shoulder, and hyperkyphosis among people with upper crossed syndrome: A systematic review and meta-analysis. *BMC Musculoskelet Disord*. 2024;25(1). doi:10.1186/s12891-024-07224-4
215. Magnusson SP, Simonsen EB, Aagaard P, Boesen J, Johannsen F, Kjaer M. Determinants of musculoskeletal flexibility: Viscoelastic properties, cross-sectional area, EMG and stretch tolerance. *Scand J Med Sci Sports*. 1997;7(4):195-202. doi:10.1111/j.1600-0838.1997.tb00139.x

216. Takeuchi K, Takemura M, Nakamura M, Tsukuda F, Miyakawa S. Effects of active and passive warm-ups on range of motion, strength, and muscle passive properties in ankle plantarflexor muscles. *J Strength Cond Res.* 2021;35(1):141-146.
doi:10.1519/JSC.0000000000002642
217. Konrad A, Stafilidis S, Tilp M. Effects of acute static, ballistic, and PNF stretching exercise on the muscle and tendon tissue properties. *Scand J Med Sci Sports.* 2017;27(10):1070-1080. doi:10.1111/sms.12725
218. Franklin, R., Bohunicky, S., Wang, S., Scribbans TD. The impact of acute stretching on shear wave velocity (stiffness) of the pectoralis major and minor: Individual responses using technical error. *Appl Physiol Nutr Metab.* 2023;48(10 (Suppl. 2)):S102.
doi:10.1139/apnm-2023-0378
219. Sparks LM. Exercise training response heterogeneity: Physiological and molecular insights. *Diabetologia.* 2017;60(12):2329-2336. doi:10.1007/s00125-017-4461-6
220. Mann TN, Lamberts RP, Lambert MI. High responders and low responders: Factors associated with individual variation in response to standardized training. *Sport Med.* 2014;44(8):1113-1124. doi:10.1007/s40279-014-0197-3
221. Oldham PD. On the analysis of repeated measurements of the same subjects. *J Chronic Dis.* 1963;16(5):449-450. doi:10.1016/0021-9681(63)90125-5
222. Bonafiglia JT, Preobrazenski N, Gurd BJ. A systematic review examining the approaches used to estimate interindividual differences in trainability and classify individual responses to exercise training. *Front Physiol.* 2021;12(November):1-18.
doi:10.3389/fphys.2021.665044
223. Olivencia O, Godinez GM, Dages J, et al. The reliability and minimal detectable change

- of the ely and active knee extension tests. *Int J Sports Phys Ther.* 2020;15(5):776-782.
doi:10.26603/ijspt20200776
224. Donti O, Gaspari V, Papia K, Panidi I, Donti A, Bogdanis GC. Acute effects of intermittent and continuous static stretching on hip flexion angle in athletes with varying flexibility training background. *Sports.* 2020;8(3). doi:10.3390/sports8030028
225. Donti O, Tsolakis C, Bogdanis GC. Effects of baseline levels of flexibility and vertical jump ability on performance following different volumes of static stretching and potentiating exercises in elite gymnasts. *J Sports Sci Med.* 2014;13(1):105-113.
<http://www.ncbi.nlm.nih.gov/pubmed/24570613>.
226. Fraulin L, Holzgreve F, Haenel J, et al. A device-based stretch training for office workers resulted in increased range of motion especially at limited baseline flexibility. *Work.* 2021;68(2):353-364. doi:10.3233/WOR-203273
227. Kay AD, Blazeovich AJ. Moderate-duration static stretch reduces active and passive plantar flexor moment but not Achilles tendon stiffness or active muscle length. *J Appl Physiol.* 2009;106(4):1249-1256. doi:10.1152/jappphysiol.91476.2008
228. Morse CI, Degens H, Seynnes OR, Maganaris CN, Jones DA. The acute effect of stretching on the passive stiffness of the human gastrocnemius muscle tendon unit. *J Physiol.* 2008;586(1):97-106. doi:10.1113/jphysiol.2007.140434
229. Yoshitake Y, Takai Y, Kanehisa H, Shinohara M. Muscle shear modulus measured with ultrasound shear-wave elastography across a wide range of contraction intensity. *Muscle Nerve.* 2014;50(1):103-113. doi:10.1002/mus.24104
230. Borstad JD. Measurement of pectoralis minor muscle length: Validation and clinical application. *J Orthop Sport Phys Ther.* 2008;38(4):169-174. doi:10.2519/jospt.2008.2723

231. Finley M, Goodstadt N, Soler D, Somerville K, Friedman Z, Ebaugh D. Reliability and validity of active and passive pectoralis minor muscle length measures. *Brazilian J Phys Ther.* 2017;21(3):212-218. doi:10.1016/j.bjpt.2017.04.004
232. Bang MD, Deyle GD. Comparison of supervised exercise with and without manual physical therapy for patients with shoulder impingement syndrome. *J Orthop Sport Phys Ther.* 2000;30(3):126-137. doi:10.2519/jospt.2000.30.3.126
233. Ingram LA, Tomkinson GR, D'Unienville NMA, et al. Optimising the dose of static stretching to improve flexibility: A systematic review, meta-analysis and multivariate meta-regression. *Sport Med.* 2025;55(3):597-617. doi:10.1007/s40279-024-02143-9