THE EFFECTS OF PULSED SHORTWAVE DIATHERMY AND STRETCH ON THE TORQUE-ANGLE RELATION OF THE CALF (PLANTAR FLEXOR) MUSCLES ASSOCIATED WITH PASSIVE STRETCH BOTH DURING AND AFTER TREATMENT

by

MEGAN JANETTE SCHORR

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ABSTRACT

THE EFFECTS OF PULSED SHORTWAVE DIATHERMY AND STRETCH ON THE

TORQUE-ANGLE RELATION OF THE CALF (PLANTAR FLEXOR)

MUSCLES ASSOCIATED WITH PASSIVE STRETCH

BOTH DURING AND AFTER TREATMENT

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Supervising Professor: Mark D. Ricard

Pulsed shortwave diathermy (PSWD) is an electromagnetic thermal modality used in the clinical setting. It is believed that temperature increases associated with PSWD in combination with stretch may reduce stiffness and increase tissue compliance. Our objective was to assess the short-term effects of PSWD and stretch on the torque-angle relation of the triceps surae muscles when passive stretch is applied both during and after PSWD treatment. We used a 3 X 4 (Time X Treatment) and a 2 X 4 (Time X Treatment) crossover repeated measure designs in this study. The independent variables were condition (stretching during diathermy, stretching after

diathermy, and stretching during and after diathermy treatment) and time (pre, post, 15-minutes post treatment). Alpha was set at 0.05. Data was collected at the University of Texas at Arlington, Department of Kinesiology's Neuromuscular Exercise Science and Research Laboratory. Sixteen males (height, 175.86 ± 9.13 centimeters; weight 82.30 ± 17.16 kilograms; age, 22.94 ± 3.75 years) completed a health history form and signed an informed consent. Dependent variables were energy absorbed, energy returned, peak torque, average stiffness, intramuscular temperature, and average range of motion (ROM). PSWD treatments showed an average increase of 3.51 ± 0.27° C in intramuscular temperature after 20 minutes of PSWD treatment. Whereas, the control treatment's intramuscular temperature decreased 0.85 ± 0.30° C after 30 minutes of resting on a plinth. Low-load long duration stretching in combination with PSWD seems to have an effect on and significantly increases tissue compliance. Peak torque and average stiffness decreased (12% and 10%, respectively) from pre to post treatment for all heating and stretch conditions. However, there was a greater increase in tissue compliance during treatments when stretch was applied during tissue cooling. From the results of our study, we now believe that stretch combined with heat does affect tissue compliance and that the best time to stretch is after the tissue has been heated sufficiently and while the tissue is cooling.

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CHAPTER 1

INTRODUCTION

The practice of heating prior to stretching is common among rehabilitative clinicians. The combination of heating and stretch is believed to improve flexibility and tissue compliance necessary for optimal athletic performance and rehabilitative treatment. Current research has not established guidelines for the clinical application of heating and stretch. To maximize our time as clinicians and coaches, we should determine the best protocol (amount and type) of therapeutic heating and the timing of stretch. For example, how warm does the tissue need to be so that it will respond best to stretch. And should the stretch be applied during tissue warming, at peak tissue temperature, or when the temperature of the tissue is cooling?

Many researchers have explored the temperature/stretch relationship by investigating rat tail tendon *in vitro*. Warren et al. [51, 52] and Lehmann et al.'s [20, 21] studies demonstrated that stretching while the tissue was heated to temperatures between 39° and 45° C (3° to 8° C increase above baseline temperatures) resulted in lasting elongation of the tissue. [6, 20, 51, 52] Pulsed shortwave diathermy (PSWD) is one type of deep heating therapeutic modality that causes increased intramuscular temperature within the therapeutic range of 3° to 8° C. Increases in intramuscular temperature of the gastrocnemius by PSWD has been verified by Draper et al. [8,] Trowbridge et al.[48, 49], and Garrett et al.[12] Draper et al [8] observed a 3.78 ± 1.19°C increase in intramuscular temperature of the gastrocnemius at a depth of 3cm below the surface of the skin after 20

minutes of PSWD (parameters: 27.12 MHz frequency, 800 burst per second, 400 μsecond burst duration, average power = 40 W, total power = 150 W). Using the same parameters Trowbridge et al [48], at a depth of 2.5 cm below the surface of the skin, observed a 2.75 ± 1.39°C (range 1.46°C to 4.69°C) increase in intramuscular temperature of the gastrocnemius (average skin fold ≤ 13 mm). Again using the same parameters as Draper et al. [8] and Trowbridge et al. [48], Garrett et al.[12] observed a 3.02 ± 1.02°C, 4.58 ± 0.87°C, and a 3.28 ± 1.64°C at three sites 3 cm below the surface of the skin. Therefore, PSWD does increase intramuscular tissue temperature of the gastrocnemius to therapeutic levels defined by Lehmann et al. [20, 21] which may be sufficient to cause lasting tissue compliance.

Lehmann et al. [20, 21] also observed that less tissue damage occurs if stretch is applied at peak temperatures. Trowbridge et al. [48, 49] and Draper et al. [8] found that peak tissue temperature with PSWD occurred after 15 minutes of treatment (parameters: 27.12 MHz frequency, 800 burst per second, 400 μsecond burst duration, average power = 40 W, total power = 150 W). A PSWD treatment, with the above parameters, typically lasts 20 minutes. During the last five minutes of PSWD treatment peak temperature levels are maintained or slightly decrease. Lehmann et al. [20, 21] also emphasized that for a lasting elongation of tissue to occur, a stretch should be applied during heating and while the tissue temperature is cooling. Therefore, the best time to apply stretch during PSWD would be after 15 minutes of treatment or when peak temperatures have been reached and continued during cooling of the tissue.

Draper et al. [10, 43] and Rose et al. [10, 43] theoretically designed a stretch-while-heating-and-cooling technique for 1-MHz and 3-MHz continuous ultrasound. Draper et al. [10, 43] and Rose et

al. [10, 43] defined the stretching window as a time period of vigorous heating when tissue undergoes its greatest extensibility and elongation. [10, 40, 43] [10, 43] In theory the stretching window, designed from the results of Lehmann's studies [20, 21] of rat tail tendons, occurred after intramuscular temperatures were elevated to > 3° C. The stretch needed to be maintained during both the vigorous ultrasonic heating and during tissue cooling in order to achieve the greatest increase in tissue compliance. [10, 40, 43]

Brucker et al. [4] and Peres et al. [38] implemented a stretch-while-heating-and-cooling protocol for PSWD. However, these studies were concerned with the effects of PSWD and stretch on ROM changes over time. Neither study compared the effects of stretch-while-heating-and-cooling technique to other techniques in order to define an optimal stretching window for PSWD. [4, 10, 38] They did observe a significant increase in ROM over time with the stretch-while-heating-and-cooling protocol, leading us to believe that this technique is affecting tissue compliance. [4, 10, 38] To this date, there is no known information regarding an optimal stretching window for PSWD.

Therefore, the purpose of this study is to determine the effects of stretching applied both during and/or after pulsed shortwave diathermy on the torque-angle relation of the triceps surae muscles. We want to determine if there may be an optimal stretching window when using therapeutic PSWD treatments to heat muscle tissue. Our primary research questions are 1) should stretch be applied during the PSWD, after the PSWD, or both; and 2) what are the effects of diathermy and stretch 15 minutes after treatment when the tissue temperature has cooled toward baseline values?

1.1 Hypotheses

We hypothesized that for stretching to be most effective it should be applied both during and after PSWD. By applying 20 minutes of PSWD with stretch during the last 5 minutes and stretch during the 5 minutes after treatment (PSWD 5/5 condition) we will induce the greatest changes in the torque-angle relationship of the triceps surae. We also expect that at 15 minutes post treatment the PSWD 5/5 condition will still exhibit the greatest changes in the torque-angle relationship of the triceps surae due to permanent changes in the mechanical properties of the tissue.

1.2 Definition of Terms

Pulsed Shortwave Diathermy (PSWD) – a high frequency (27.12 MHz) electromagnetic deep heating therapeutic modality that creates kinetic energy within the tissue and causes an increase in intramuscular temperature.

Surface Electromyography (EMG) – a recording of the neural activity of muscle fibers via surface electrodes applied to the skin.

Intramuscular Thermocouple – a fine wire thermometer that is inserted into the muscle to measure changes in tissue temperature.

Passive Stretch – an elongation of a relaxed muscle or tendon by an external force.

Range of Motion (ROM) - maximal amount of mobility of a joint

Stiffness – the slope of the torque angle relationship or the change in force per change in radians or degrees.

Energy Absorbed – the energy absorbed by the load applied to the tissue (Figure 2.1)

Energy Returned – the energy returned from the tissue after removal of the load (Figure 2.1)

Viscosity – fluid-like resistance to an external force that is velocity and temperature dependent.

Elasticity – the ability to return to an original form after an external force is applied, temporary deformation

Plasticity – permanent changes that occur after an external force is applied

Thixotropy – the ability of a material to show more resistance with small forces and less resistance to larger forces.

1.3 Delimitations

The delimitations of this study were: 1) subjects included UTA staff, students, and non-UTA students, 2) male subjects were between the ages of 18-40 years, 3) males had subcutaneous fat less than 25 mm (0.98 in), and 4) males did not have a previous history of lower leg injury that would affect the passive tension properties of surrounding tissues during ankle dorsiflexion and plantarflexion.

1.4 Assumptions

The following assumptions were considered throughout the study: 1) the subjects accurately filled out the health history questionnaire, 2) subjects followed instructions throughout

application of modalities, 3) and the thermocouple inserted into the medial gastrocnemius was not significantly affecting the muscle tissue properties.

1.5 Limitations

The limitations of this study were: 1) type of stretch, 2) location of heat application at the musculotendoneous junction, 3) use of only male subjects, 4) the use of the Biodex® for low-load long duration stretching, 5) timing of treatment conditions, 6) limited familiarization, 7) maximal ROM measurements for the Biodex®, 8) no temperature measurements during treatment, and 9) time of day that data was collected.

CHAPTER 2

REVIEW OF THE LITERATURE

2.1 Introduction to Stretch

Stretching is used by clinicians to decrease muscle spasms, increase joint range of motion (ROM), and increase elongation of ligaments, tendons, and muscular tissue.[4, 7, 9, 14, 20, 21, 38, 54] Stretching causes both short term changes and long term adaptations of tissue structures.[37] The short term changes in tissue after stretching may be due to neurophysiological factors and/or mechanical factors of the tissues.[29, 54]

2.1.1 Neurophysiological Factors of Stretching

Two neurophysiological factors of tissue extensibility include muscle stretch reflex and stretch tolerance. The muscle stretch reflex is controlled by the intrafusal fibers within a muscle group. When stretched these intrafusal fibers can cause either a monosynaptic phasic stretch reflex or a multisynaptic tonic stretch reflex. [23] Facilitation of the intrafusal muscle spindles causes a reflexive muscle contraction. This reflexive muscle contraction occurs with high velocity lengthening or a sustained stretch; which initially limits increased ROM.[23]

Weiss et al. [53] looked at the relationship between the angle of the ankle and the stretch reflex of the triceps surae. The ankle was passively stretched from 20.6° of plantar flexion into 12° of extreme dorsiflexion. The stretch reflex was monitored through changes in EMG

amplitude. EMG recordings remained constant until the ankle passed a neutral position. A neutral position was defined as the ankle angle at which no net passive joint torque was generated. If the ankle was dorsiflexed past the neutral position electromyography (EMG) amplitudes would increase. Weis et al. [53] concluded that the stretch reflex increased as the ankle was moved toward its maximal ROM. Other studies [11, 39, 42] have looked at the velocity of stretch on the magnitude of the stretch reflex. These studies[11, 39, 42] found an increase in EMG activity with increased velocity of stretch. Theoretically, the initial neurophysiological limitations of the stretch reflex during passive stretch may be eliminated by controlling the velocity and the depth of stretch. For example, a clinician can apply stretches at speeds less than 5.0°/sec and/or not stretch to depths that would cause excessive pain or discomfort.

Stretch tolerance is another neurophysiological factor that may affect short term tissue changes and ROM measurements. Stretch tolerance is one's ability to tolerate various stretch loads. Repeated doses of maximal stretch appears to increase stretch tolerance.[31] Magnusson et al [31] attributed a 10° increase in hamstring ROM to an increased stretch tolerance. [31] They looked at the effects of maximal stretch on increasing stretch tolerance. Each group received one of two sampling protocols, before and after three weeks of stretch training. Protocol 1 received 90 seconds of static stretch to the hamstrings via a KinCom® dynamometer and was stretched through the same predetermined ROM at a velocity of 5.0°/sec. Protocol 2 was stretched at a velocity of 5.0°/sec to the point of a self selected uncomfortable stretch. They concluded that the mechanical properties of the tissue must have remained unchanged because in protocol 2 the

passive torque necessary to move the hip through the ROM stayed the same even though total hip ROM had increased.

In contrast to changes in ROM being explained by an increase in stretch tolerance, McNair et al. [34] found a significant decrease in passive torque with the application of stretch. They found a decrease in stiffness with cyclical stretching for 60 seconds (16.0 % decrease) and a decrease in peak tension with 60 seconds of static stretching (21.5% decrease). Thus, the conclusion that stretch tolerance accounts for increased ROM measurements post stretching is not completely substantiated. Changes in ROM and passive tension may also be due to changes in mechanical factors of tissue.

2.1.2 Mechanical Factors of Stretching

The mechanical properties of tendons, ligaments and muscle tissue during passive stretch are affected by the thixotropic, viscoelastic, and the plastic behavior of tissue.[14] Changes in the mechanical properties have been equated to changes in tissue stiffness, energy absorbed, energy returned, and changes in ROM. [2, 6, 7, 18, 24-32, 34, 35] These measurements may help to explain the temporary and potentially permanent tissue changes associated with stretching tissue.

Muscle tissue and associated tendons and ligaments of a joint exhibit viscoelastic behavior. Viscoelastic materials are characterized by hysteresis, stress relaxation, and creep. [36] Hysteresis is defined as the area between the energy load and unload curves or the energy loss as

heat due to internal damping.[17] (Figure 2.1)

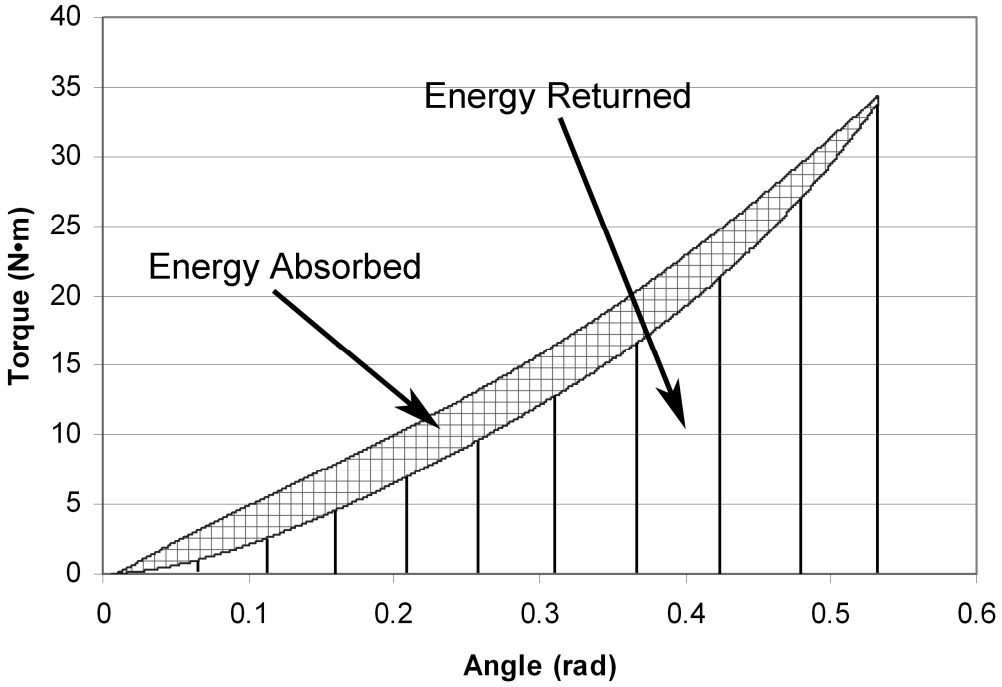


Figure 2.1: A typical load and unload graph. Energy returned was calculated as the area under the unload curve (vertical lines). Energy absorbed or hysteresis was calculated as the area between the load and unload curves (crosshatched area).

Kubo et al. [17] looked at the effect of stretch training on the viscoelastic properties (i.e. hysteresis and stiffness) of the muscle tendon complex of the gastrocnemius. Eight men were stretch for three weeks. One leg was randomly assigned to stretch training and the opposite leg served as a control. Each subject stretched twice a day for twenty consecutive days, but did not stretch on days when pre and post measurements were collected. During each session the subjects performed five stretches for 45 seconds with 15 second rests in between. The gastrocnemius was stretched at 35°s of dorsiflexion while standing. The stretch training produced no significant differences in stiffness ($p = 0.621$), but hysteresis did decrease

significantly ($p = 0.009$, $-37.2 \pm 22.2\%$) .[17] These results suggest a decrease in energy dissipation when tissue is stretched. In contrast, Kubo et al. [16] in a later study observed the affects of 20 days of bed rest on the stiffness and hysteresis of the knee extensors and ankle plantar flexors. Ultrasonic isometric measurements were taken of the vastus lateralis and medial gastrocnemius before and after the 20 days of bed rest. Stiffness decreased for the vastus lateralis (70.3 ± 27.4 *v* 50.1 ± 24.8 N/mm, before and after bed rest, respectively; $p = 0.003$) and medial gastrocnemius (29.4 ± 7.5 *v* 25.6 ± 7.8 N/mm, before and after bed rest, respectively; $p = 0.054$); the vastus lateralis stiffness being significantly different before and after bed rest and the medial gastrocnemius showing no significant difference. After bed rest hysteresis increased for the vastus lateralis ($16.5 \pm 7.1\%$ *v* $28.2 \pm 12.9\%$, before and after bed rest, respectively; $p = 0.017$), but not for the medial gastrocnemius ($17.4 \pm 4.4\%$ *v* $17.7 \pm 6.1\%$, before and after bed rest, respectively; $p = 0.925$). [16] Thus, bed rest causes a decrease in stiffness and an increase in hysteresis in the vastus lateralis of the knee extensors, but not in the medial gastrocnemius of the plantar flexors. Although hysteresis is a defining element of viscoelastic tissues, the mechanical significance behind the area within the hysteresis loop is unknown.

In addition, stress relaxation, another property of viscoelastic materials, is observed when a constant stress is applied. Stress relaxation occurs when tissue is held at a set length or continuously moved through a full ROM; the tension or strain will decrease in a non-linear fashion with time. [24, 34] (Figure 2.2)

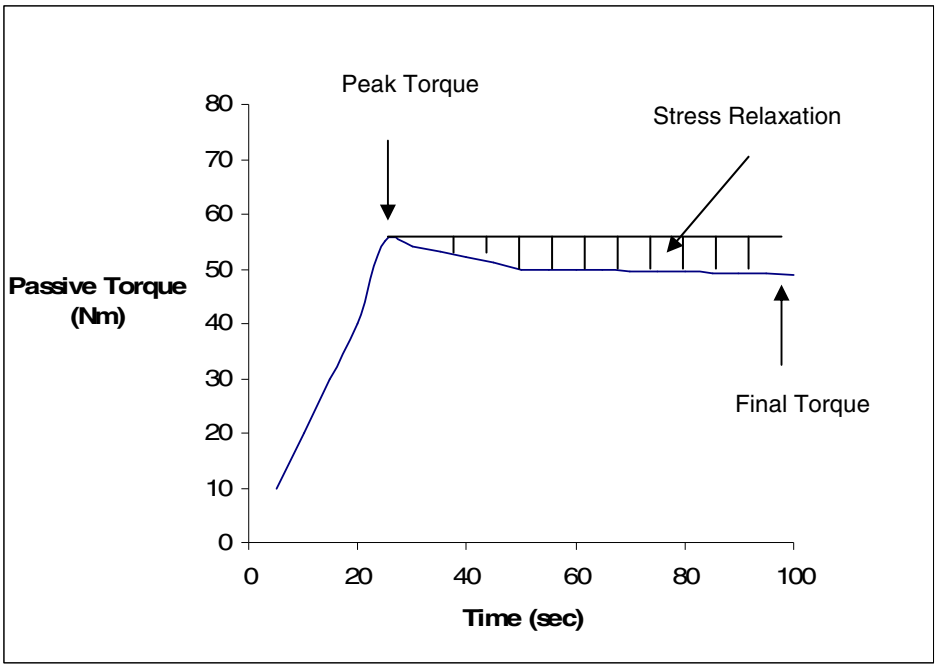


Figure 2.2: Viscoelastic stress relaxation

McNair et al. [34] compared the affects of cyclical stretching to static stretching on 24 subjects. In a 2 X 4 (Time X Treatment) repeated measures design, each subject received all four treatments (continuous passive motion, 1 X 60-s hold, 2 X 30-s hold, and 4 X 15-s hold). One treatment was taken each week the same time each day for four weeks. During treatments the ankle joint was moved through 80% of maximal range of motion. A significant difference was found between initial and final stiffness for continuous passive motion only ($p < 0.05$), but all groups showed a decrease in stiffness over time. Across time force decreased significantly ($p < 0.05$) but was not significantly different between treatments. The hold stretches had the greatest decrease in force over time (continuous passive motion, 10.5%; 1 X 60-s hold, 19%; 2 X 30-s hold, 21.7%; and 4 X 15-s hold, 21.5 %,). [34] McNair et al. [34] concluded that if decreasing stiffness is the purpose of stretch training, continuous motion is more effective, but if relaxation

of peak torque is the goal, holds are the most effective. McNair et al. [34] also eluded that the change in peak torque after passive stretch may be explained by the thixotropic behavior of biological tissue.

Thixotropy is the initial stiffness of the muscle tissue during passive stretch. It is believed to be long-term bonds, called cross-bridges, which have formed between the actin and the myosin heads. The number and location of cross-bridges are dependent upon the history of the tissue.[41] For example, if a muscle has been stretch or agitated prior to movement there will be a decrease in stiffness due to detachment of the cross bridges. In biology this decrease in stiffness was originally observed by Peterfi in 1927 after he agitated the cytoplasm of sea urchin eggs with a needle.[41] Thixotropy has since been used to describe the dry friction-like behavior of muscle tissue, differentiating it from the viscous (temperature and velocity dependent) and elastic (load dependent) behavior of muscle tissue.[1] This friction-like behavior exhibits more resistance to small forces and less resistance to larger forces, which occurs due to smaller forces breaking fewer cross-bridges when compared to larger forces. Hagbarth et al. [13] investigated the inherent changes in stiffness after transient finger flexion and extension. Finger flexor stiffness measures increased after finger flexion and decreased after finger extension or isometric contractions. After resting periods of several minutes, finger flexor stiffness returned to previous levels. Lakie and Robson[19] found similar thixotropic behavior when measuring passive stretch of a frog sartorius muscle *in vivo*. The sartorius muscle was secured proximally by the frog's pelvis to a stainless steel hook and distally to a stainless steel wire. The wire was connected to a miniature electric motor to measure changes in resistance. Three stretches and one 5 second

perturbation were applied to the muscle. Muscle stiffness was reduced after being stretched and agitated, but returned to pre-treatment levels after ten minutes of rest.[19] Short term mechanical changes of tissue stiffness after stretch or activity may be due to a thixotropic effect.

In addition, the viscoelastic property of creep has been associated with the short term adaptations of tissue after application of stretch. [22] Creep is the tendency of viscoelastic material to move or change shape in order to relieve the strain from an applied stress that is below it's yielding or fracture point. [46] Creep is affected by the magnitude of the stress, volume of time the stress is applied, and temperature of the material. Measuring creep in biological tissues is difficult due to the complexity of the combined influence of the series elastic components, parallel elastic components, and the contractile components during stretch, but changes in ROM have been associated with creep within this viscoelastic model. [22] In contrast, Spernoga et al. [46] looked at the short term effects of PNF stretching on ROM and the retention of increased ROM. Thirty males with limited hamstring flexibility were randomly assigned to either the control or experimental stretch group. All subjects performed six active knee extensions. The sixth extension was used for the pre-stretch measurement. The experimental stretch group received 5 hold-relax stretches and the control group rested in supine on a table for five minutes. Post-test measurements were taken at 0, 2, 4, 6, 8, 16, 32 minutes. A significant increase in ROM within the experimental stretch group lasted 6 minutes post stretching. Due to ROM returning to pre-test angles, Spernoga et al. [46] concluded that increase in ROM was not due to creep, but rather elastic components within the tissues.

2.2 Introduction Therapeutic Heating Modalities

The application of heat is believed to alter the viscous property of connective tissue. Decreases in the viscous properties of ground substance within connective tissue may augment the depth of tissue stretch.[38, 44] Increases in the temperature of tissue are believed to increase tissue extensibility and improve ROM. Active and passive techniques have been used to heat tissue and include active warm up, hot packs, paraffin baths, continuous ultrasound, and pulsed shortwave diathermy.[4, 7, 9, 15, 20, 44, 48] To determine the effectiveness of these therapeutic heating modalities Knight et al.[15] compared the influence of different types of heating combined with stretch on dorsiflexion ROM. Therapeutic heating protocols included no heating, an active warm up of 40^{+} heel raises, superficial moist heat pack for 15 minutes, or 1.0 MHz continuous ultrasound at 1.5 W/cm^2 for seven minutes. The stretching protocol included four 20-second runner's stretches three times per week for six weeks. Although all groups showed increases in active range of motion (AROM) and passive range of motion (PROM) after six weeks of treatment, the continuous ultrasound protocol AROM and PROM had the largest increases in ROM (no heat, 4.10°/6.11°; active warm up, 4.16°/4.21°; heat pack, 4.38°/4.90°; and 6.2°/ 7.35°, respectively).[15]

Chan et al. also found that the deep heating modality of 3 MHz ultrasound at 1 W/cm^2 significantly increased tendon temperature. Thermisters were inserted in the medial aspect of the right patellar tendon. Ultrasound treatments were applied at two and four times the effective radiating area (ERA) over the patellar tendon. The thermisters recorded 8.7°C ± 1.7°C and 5.0°C ± 1.0°C increase in two and four times the ERA, respectively. These increases in tendon

tissue temperature are similar to the increased temperatures used by Warren et al.[51, 52] and Lehmann et al. [20] for increasing the tissue extensibility of rat tendons.

2.2.1 Introduction to Diathermy

Pulsed shortwave diathermy (PSWD) is a deep heating therapeutic modality that causes an increase in intramuscular temperature. The increase in temperature occurs when an electromagnetic field is created. The magnetic field resonates from the machine and into the underlying tissues, which creates localized eddy currents that oscillate tissue and produce kinetic energy. The electrical field also causes a dipole effect among free ions which produces kinetic energy because of free ion movement. The increase in tissue temperature is caused by increased kinetic energy.

Increases in intramuscular temperature by PSWD has been verified by Draper et al.[2] and Trowbridge et al.[3,4] Draper et al.[2] found a 3.78 ± 1.19°C increase in intramuscular temperature of the gastrocnemius after a 20 minute treatment of PSWD (parameters: 27.12 MHz frequency, 800 burst per second, 400 μsecond burst duration, average power = 40 W, total power = 150 W). Using the same parameters Trowbridge et al.[3] found a 2.75 ± 1.39°C (range 1.46°C to 4.69°C) increase in intramuscular temperature of the gastrocnemius (average skin fold $\leq$ 13 mm).

Trowbridge et al.[4] also reported the effects of subcutaneous adipose tissue on temperature change in the low back and quadriceps. Subjects with a subcutaneous adipose tissue of $\leq$ 18 mm exhibited 3.26 ± 0.08°C and 1.47 ± 0.07°C increases in temperature for the quadriceps and low back, respectively. Subjects with a subcutaneous adipose tissue of $\geq$ 18 mm exhibited 2.76 ± 0.09 and 1.10 ± 0.05 increases for quadriceps and low back, respectively.[3]

There was a significant difference between the two levels of subcutaneous adipose tissue for the quadriceps ($p = 0.006$) and the low back ($p = 0.001$).[4] These findings suggest that intramuscular temperature change may be affected by amount of subcutaneous fat and muscle location.

2.3 Effects of Diathermy and Stretch on Tissue Extensibility

To alter the factors affecting flexibility of a joint many clinicians use deep heating therapeutic modalities and static stretch [3, 4, 7, 9, 14, 20, 21, 38, 54] Current research has not established guidelines for the clinical application of heating and stretch. For example, how warm does the tissue need to be so that it will respond best to stretch; should the stretch be applied during tissue warming, peak tissue temperature, or when the temperature of the tissue is decaying?

Pulsed shortwave diathermy (PSWD), a deep heating therapeutic modality, is applied in the clinical setting to increase intramuscular temperature. Several studies have assessed the chronic effects of pulsed shortwave diathermy (PSWD) and stretch on changes in ROM.[4, 7, 9, 38, 44] However, these studies have been inconclusive and have not been able to identify the mechanisms behind the changes in ROM. For example, Seiger and Draper[44] demonstrated an increase in ROM by using PSWD in conjunction with joint mobilizations in several case studies of post surgical ankle contractures. In one subject, 2 years post injury, dorsiflexion and plantarflexion was improved by 8° and 22°, respectively [44] Peres et al [38] and Brucker et al [4] also performed studies evaluating the effects of PSWD, but in combination with 3 weeks of low-load, long-duration stretching. Both studies showed increases in ankle ROM.[4, 38] When PSWD was applied during stretching, Peres et al.[38] found greater gains in ankle ROM than stretching alone, but they did not evaluate ankle ROM retention. Brucker et al.[4] used the same stretch protocol as

Peres et al.[14] and reassessed ankle ROM gains during treatment and after 3 weeks. Both groups exhibited ankle ROM gains but PSWD application did not affect the retention of flexibility gains.[4] Although, these studies have provided us with empirical evidence in regards to ROM improvement, no theories have been developed as to why these changes were exhibited. For example, were the ROM changes because of neurophysiological changes, like a resetting of the muscle spindle's sensitivity, or were they because of mechanical factors, like viscous or plastic changes in the tissue structure brought on by the increase in temperature from the PSWD treatments.

Our lab has already begun to investigate the changes in passive tension after stretch, intramuscular heating, and the combination of both. Recently, we collected passive tension data for the triceps surae using the protocol outlined in the methods section. Our four treatment conditions were control (no treatment), 10 minutes stretch, 20 minutes PWSD and no stretch, and 20 minutes PWSD plus 10 minutes stretch. No significant decreases in average stiffness (Nm/rad) were found for control group or the diathermy only group between any of the time points (pre, post, 15 mins post, 30 mins post) ($p>0.05$).[3] There was a significant decrease in average stiffness between pre and post treatment within the stretch group (pre: 72.49 ± 1.08 Nm/rad, post: 61.96 ± 1.08 Nm/rad) and within the diathermy and stretch group (pre: 72.02 ± 1.13 Nm/rad, post: 62.01 ± 1.13 Nm/rad).[3] Because there was no change in the diathermy only group and no significant differences between the two stretch groups, we believe that the stretching was the primary stimulus for changes in passive tension. However, we do not know if the timing of the stretch is an important determinant in changes in passive tension. We applied

the stretch during the PSWD, but not during tissue cooling. Lehmann et al.[8,9] emphasized that for a lasting elongation of tissue to occur, heating (as seen with PSWD) and stretch should be combined and stretch should be continued while the tissue temperature is cooling.

2.4 Stretching Window

Draper et al. [10, 43] defined a stretching window for 1-MHz and 3-MHz continuous ultrasound. The stretching window was defined as the time period of vigorous heating when tissue undergoes its greatest extensibility and elongation. [40] Draper et al. [10, 43] stretching window was theoretical in nature and did not compare the stretch-while-heating-and-cooling technique to other heating and stretching techniques. In addition, Draper et al. [10, 43] did not define an optimal stretching window for other therapeutic heating modalities such as PSWD. [4, 10, 38] To this date, there is no known information regarding an optimal stretching window for PSWD.

Therefore, the purpose of this study is to determine the effects of stretching applied both during and/or after pulsed shortwave diathermy on the torque-angle relation of the triceps surae muscles. In addition, we want to determine if there may be an optimal stretching window when using therapeutic PSWD treatments to heat muscle tissue.

CHAPTER 3

METHODS

3.1 Experimental Design

This study incorporated two study designs. For passive tension and intramuscular temperature measurements a 3 X 4 (time X treatment) repeated measures design and for ROM a 2 X 4 (time X treatment) repeated measures design were used to guide this study. The independent variable of time for passive tension measurements consisted of 3 time points, pretreatment, post treatment, and posts 15 minutes treatment (Appendix A).

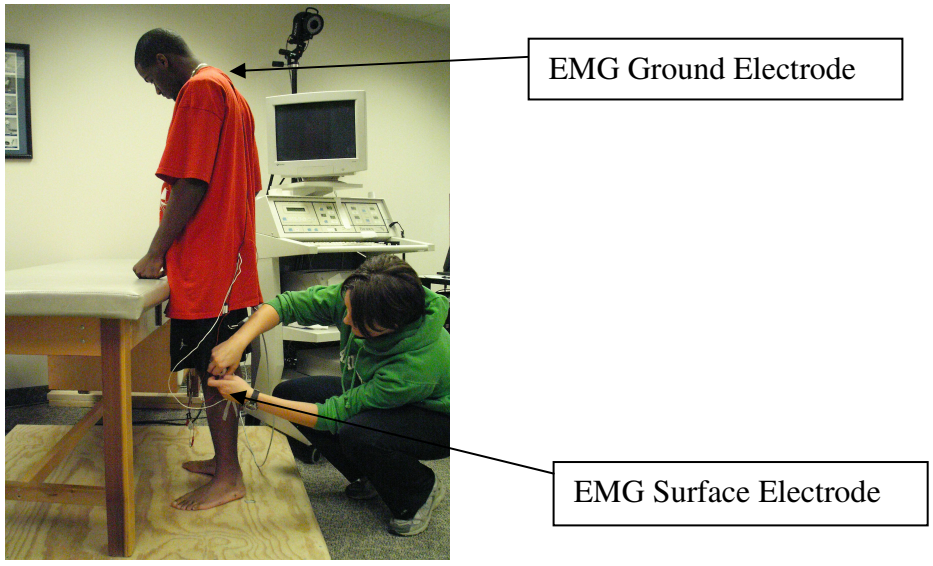


Figure 3.1: External electrode attachment: lateral gastrocnemius, anterior tibialis, and soleus.

The total volume of time for each protocol was 45 minutes; 30 minutes of treatment and 15 minutes post treatment. The first independent variable was treatment, which consisted of 4 conditions (see below), a control and three 20 minute pulsed shortwave diathermy (PSWD) treatment conditions that all included 10 minutes of stretch; however, the timing of the stretch was dispersed differently in each condition.

1. Control – no diathermy or stretch (CON)
2. 20 minutes of PSWD with stretch during the last 5 minutes and stretch during the 5 minutes after treatment (PSWD 5/5)
3. 20 minutes of PSWD with stretch during the last 10 minutes (PSWD 10)
4. 20 minutes of PSWD with stretch during the 10 minutes after treatment (PSWD/10)

The study design

There were three main dependent variables: passive tension, EMG amplitude of the gastrocnemius, soleus, and anterior tibialis, and gastrocnemius intramuscular temperature changes from baseline. The passive tension dependent variables were measured using a technique developed in a prior study[48] and were peak torque, peak stiffness, average stiffness, energy absorbed, energy returned, and ROM.

3.2 Subject Familiarization

Prior to participation in the study all subjects read and signed an informed consent in accordance with the institutional review board guidelines. Subjects filled out a health history questionnaire, establishing freedom from lower leg injury or medications that would interfere with the measurement of passive tension or the application of pulsed shortwave diathermy.

Subjects were randomly assigned to one of four counterbalanced orders of the four treatment conditions.

Subjects reported to the Neuromuscular Exercise Science and Research Laboratory for a 45 minute familiarization session. While lying prone, the same researcher (MS) measured three skinfolds of the left calf. Then portions of the left lower leg were shaved, prepped with an alcohol prep-pad and non-sterile gauze, and marked at the midpoint of the muscle belly for the lateral gastrocnemius, anterior tibialis, and soleus. EMG dual electrodes (Noraxon #272) were externally applied to the muscle and a single EMG electrode was applied over the spinous process of the 7th cervical spine. The EMG electrodes were aligned parallel to the muscle fibers of the gastrocnemius, soleus, and anterior tibialis. After application of EMG electrodes, EMG signals of the gastrocnemius, medial soleus, and the anterior tibialis were verified as the subject performed an active toe raise and a forward step on a wooden platform (Figure 3.1).

Then the subject laid prone on a second plinth with the left knee in full extension. In order to obtain full knee extension, we placed a layer of foam padding under the left knee. The Biodex® axis of rotation was aligned with the lateral malleolus and we secured the left ankle to the dorsiflexion/plantar flexion footplate with one large strap applied over the subject's mid-foot (Figure 3.2). Prior to the ROM measurement and pre measurement, anatomical zero (footplate positioned perpendicular to floor) and limb weight were determined and recorded. ROM measurements were collect with the Biodex®. The ankle was passively dorsiflexed at a velocity of 5 °/sec (0.087 rad/sec) from anatomical zero to the onset of a slightly painful stretch. At this point the subjects were instructed to press the emergency stop button which instantaneously

stopped the Biodex® level arm, angular position was collected, and the ankle was returned to anatomical zero. Subjects were thoroughly instructed in this maneuver and were allowed to try the procedure several times to a point below the pain threshold prior to data collection.[31] A total of three ROM measurements were collected within a 30 second time frame.

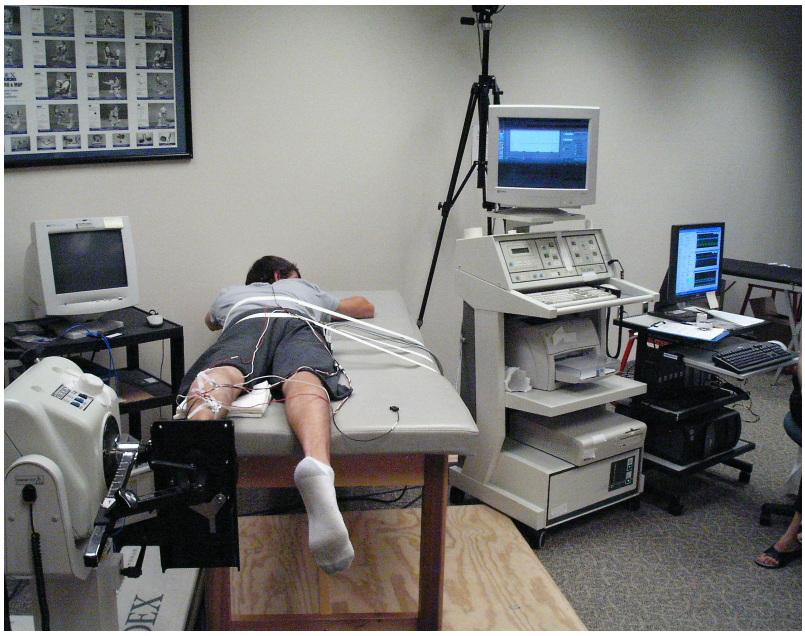


Figure 3.2: Subject secured to plinth. Left ankle secured to the dorsiflexion/plantar flexion biodex® footplate and the subject secured to the plinth with 1.5-in white athletic tape.

We then recorded ten seconds of relaxed EMG activity. Following this baseline EMG measurement we asked the subject to generate a 10 N•m plantarflexion torque, which was used to verify the level of muscular activity during Biodex® passive tension measurements. The subject was secured to the plinth with 1.5" white athletic tape below the inferior angle of the

scapula, and superior and inferior to the sacrum to decrease extraneous movement of the body as the ankle was passively moved by the Biodex® (Figure 3.2).

We determined the subject's maximum dorsiflexion by passively positioning the ankle and having the subject indicate when a non-painful maximal stretch was felt. Then ROM was set back 30º from this position and we recorded both end points of the ROM. These end points helped determine the ROM used during treatment sessions. To measure passive tension, the Biodex® was set to a velocity of 0.087 rad/s (5°/sec). Two 180 second measurements separated by 5-10 minutes were taken as the Biodex® moved the subject's ankle through 30º of dorsiflexion/plantarflexion. During measurements subjects were asked to relax, and not resist as the Biodex® passively moved the ankle through the predetermined 30º ROM. After sampling of the passive tension, three ROM measurements were again collect as previously described. Then, the midpoint of the muscle belly and borders of the EMG electrode were marked, measured, and recorded to improve the day to day reliability of the EMG electrode recordings. Between familiarization and the two testing sessions we provided a black marker to each subject to maintain EMG location between sessions.

3.3 Treatment Session

Subjects reported to the Neuromuscular Exercise Science and Research Laboratory on 4 different occasions each separated by at least 48 hours and no more than 96 hours. Each treatment session lasted for 90 minutes. Skinfold measurements, EMG preparation, and subject positioning occurred as described in the familiarization session.

3.3.1 Thermocouple Insertion and Data Collection

Before we secured the subject to the plinth, the medial belly of the subject's gastrocnemius was marked and cleansed with Poviodine-Iodine Swabstick (Professional Disposables, Inc., Orangeburg, NY). We inserted the intramuscular thermocouple perpendicular to the skin surface via a 20-gauge 1.25-in (3.15-cm) sterile intravenous catheter (Model 3056; Medex, Carlsbad, CA). After the catheter and needle insertion, we removed the needle and threaded the implantable thermocouple through the catheter tube into the muscle belly. When the 5 cm mark on the thermocouple reached the top of the catheter, we withdrew the catheter and we measured the distance between the skin surface and the 5 cm mark on the thermocouple. We intentionally inserted the thermocouple deeper than necessary, so we could retract it until the 5 cm mark was 2.5 cm above the skin surface. This ensured placement of the thermocouple 2.5 cm within the muscle belly. We prevented removal of the thermocouple (Figure 3.3) during the study by securing it to the skin with Transpore clear tape (3M Healthcare, St Paul, MN). We determined baseline temperature (less than 0.5º C change over 10 consecutive 30-second readings) prior to data collection.

While waiting for intramuscular baseline, the left ankle was secured to the dorsiflexion/plantar flexion Biodex® footplate. Anatomical zero, limb weight, ROM, ten second EMG activity, and a 10 N▪m contraction were again determined and recorded as described in the familiarization session. The subject was secured to the second plinth with 1.5" white athletic tape (Figure 3.2).

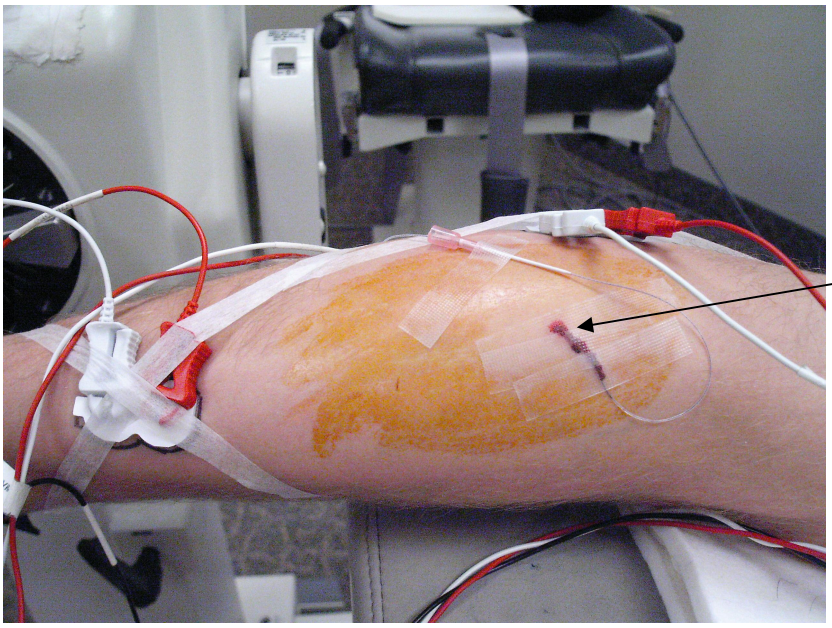


Figure 3.3: Electrode attachments and secured intramuscular thermocouple

3.3.2 Passive Tension Measurements

We determined the subject's maximum dorsiflexion by passively positioning the ankle and having the subject indicate when a non-painful maximal stretch was felt. Then ROM was set back 30º from this position. To increase day to day reliability, the ROM end points were within ±3^{o} of those obtained in the familiarization session. The subject's specific end points were used to determine the treatment ROM. One 180 second measurement (pretest) was taken as the Biodex® moved the subject's ankle through 30º of dorsiflexion/plantarflexion at a velocity of 0.087 rad/s (5°/sec). During the passive tension measurements subjects were asked to relax, and not resist as the Biodex® passively moved the ankle through the predetermined 30º ROM. After the pretest, one of four treatment protocols was applied. Order was determined by counterbalancing the treatment groups.

1. Control – no diathermy or stretch (CON)
2. 20 minutes of PSWD with stretch during the last 5 minutes and stretch during the 5 minutes after treatment (PSWD 5/5)
3. 20 minutes of PSWD with stretch during the last 10 minutes (PSWD 10)
4. 20 minutes of PSWD with stretch during the 10 minutes after treatment (PSWD/10)

Following all treatment applications, two Biodex® passive tension measurements were collected for 180 seconds at a velocity of 0.087 rad/sec (5°/sec). One measurement was collected post treatment and the second was collected 15 minutes post treatment. Subject compliance to previous relaxation guidelines was emphasized. Three ROM measurements were again collected after the 15 minute post-treatment passive tension measurements.

3.3.3 Diathermy Application

Diathermy treatment was applied for 20 minutes (Frequency of 27.12 MHz; 800 pps and pulse width of 400 microseconds and an average power output of 40 W with a peak power output of 150 W) with the ankle in a neutral position for three of the four conditions (PSWD 5/5, PSWD 10, and PSWD/10).

3.3.4 Thermocouple Removal

Single use sterile intravenous catheters were also used for each treatment session and universal precautions were followed at all times. Following post treatment measurement the thermocouple was removed and sterilized in Cidex Plus 3.4% w/ activator (Johnson & Johnson, Irvine, CA) for at least 4 hours. We cleaned the insertion site with an alcohol pad and applied

triple antibiotic ointment and a sterile bandage. We explained the signs and management of infections such as fever, swelling, red streaks up the calf, and an increased tenderness around the site of the thermocouple. We instructed the subjects to go to the student health center and call us if any of these sign or symptoms of an infection occurred.

3.4 Statistics

3.4.1 Test-retest Reliability

Test-retest reliability for average stiffness and peak torque in our laboratory was measured on 40 subjects on two separate days. Reproducibility of average stiffness and peak torque was analyzed using SPSS (14.0 for Windows) to compute the intraclass correlation coefficient (ICC) using a two factor mixed effects model and type consistency.[33, 45] A high degree of reliability was found between average stiffness (ICC = 0.961) and peak torque (ICC = 0.967).

3.4.2 Power Analysis

Priori power calculations for this study were done using preliminary data from our laboratory for average stiffness and peak torque. Sample sizes were determined based on predicted power to detect a difference of 10-15% between the groups with an alpha 0.05 and 80% power. We consider a pre to post difference of ≥10-15% to be clinically relevant. Based on the formula of Vincent[50] and Cohen[5], minimal sample sizes of between 3 and 12 subjects were determined from our existing data for these variables. Inclusion of 16 total subjects should

provide adequate power to detect clinically relevant differences and allow for possible subject attrition.

3.4.3 Statistical Analysis

The independent variables were condition (CON, PSWD5/5, PSWD 10, PSWD/10) and time (pre, post, and 15 minutes post treatment). A 4 X 3 repeated measures ANOVA was used to test for differences in the following dependent variables: passive tension (peak stiffness, average stiffness, peak torque, energy absorbed, energy returned), EMG amplitude of the gastrocnemius, soleus, and anterior tibialis, and temperature (temperature change from pre to post treatment, pre treatment to 15 minutes post treatment, and temperature change from post treatment to 15 minutes post treatment). A 4 X 2 repeated measures ANOVA was used to test for differences in ROM. A Tukey-Kramer post-hoc analysis was conducted with alpha set at 0.05.

CHAPTER 4

RESULTS

4.1 Reliability

A (3, 1) ICC was performed on the pre treatment data for day 1 and day 2. The (3, 1) ICC results are presented in Table 4.1.

Table 4.1. Reliability Data between Treatment Day 1 and Day 2

	ICC (3,1)*	
	ICC	Reliability
Average Stiffness	0.836	Moderate
Peak Torque	0.803	Moderate
Energy Absorbed	0.776	Borderline
Energy Returned	0.922	High
ROM	0.552	Poor

* Will Hopkins (3,1) ICC Reliability Analysis

The 4 X 2 and the 4 X 3 repeated measures ANOVA showed two-way interactions for intramuscular temperature, energy returned, and peak torque. Further analysis with the Tukey-Kramer multiple comparison procedure was used to determine the interaction for Condition X Time. The results for intramuscular temperature are presented in Table 4.2, Table 4.3, Table 4.4, and Table 4.5. The results for passive tension are presented in figures 4.1, 4.2, 4.3, and 4.4. The results for ROM are presented in figure 4.5.

4.2 Intramuscular Temperature

All PSWD conditions showed an increase of 3.51 ± 0.27° C in intramuscular temperature from Pre to Post PSWD treatment (Table 4.2). Whereas, the control condition showed a 0.85 ± 0.30° C decrease in intramuscular temperature after 30 minutes of resting on a plinth (Table 4.4). The PSWD conditions intramuscular temperature continued to decrease over time after PSWD treatment (Table 4.3). The intramuscular temperature did not return to baseline for any of the PSWD conditions 15 minutes post PSWD treatment.

Intramuscular temperature varied up to one degree between Post PSWD passive tension measurements and up to half a degree between Post-15 PSWD passive tension measurements (Table 4.4, 4.5). Post Control and Post-15 Control intramuscular temperatures during passive tension measurements were considerably lower than the PSWD intramuscular temperatures during passive tension measurements (Table 4.4, 4.5).

Table 4.2: Intramuscular Temperature Change
Before and After Pulsed Shortwave Diathermy

	Mean Temperature*	
	Pre Diathermy	Post Diathermy
PSWD 5/5	35.39 ± 0.11	38.67 ± 0.21
PS10WD	35.17 ± 0.18	38.90 ± 0.31
PSWD_10	35.64 ± 0.17	39.16 ± 0.26

*Mean temperatures ± standard error of measurement

Table 4.3: Intramuscular Temperature Cooling
After Pulsed Shortwave Diathermy

	Mean Temperature*		
	Post-5 Diathermy	Post-10 Diathermy	Post-15 Diathermy
PSWD 5/5	37.93 ± 0.15	37.48 ± 0.13	37.12 ± 0.12
PS10WD	38.11 ± 0.21	37.51 ± 0.17	37.08 ± 0.17
PSWD_10	38.31 ± 0.11	37.62 ± 0.11	37.40 ± 0.11

*Mean temperatures ± standard error of measurement

Table 4.4: Intramuscular Temperature During Passive Tension Measurements

	Mean Temperature*		
	Pre Passive Tension	Post Passive Tension	Post-15 Passive Tension
Control	35.98 ± 0.18	34.90 ± 0.19	34.56 ± 0.21
PSWD 5/5	35.77 ± 0.12	37.92 ± 0.14	36.86 ± 0.12
PS10WD	35.65 ± 0.18	38.90 ± 0.31	37.12 ± 0.17
PSWD_10	35.84 ± 0.17	37.68 ± 0.11	36.88 ± 0.11

*Mean temperatures ± standard error of measurement

Table 4.5: Change in Intramuscular Temperature
Between Pre and Post, and Pre and Post-15 Passive Tension Measurements

	Mean Temperature*	
	Change Pre to Post	Change Pre to Post-15
Control	-1.08 ± 0.07	-1.43 ± 0.10
PSWD 5/5	2.15 ± 0.14	1.09 ± 0.13
PS10WD	3.25 ± 0.34	1.46 ± 0.20
PSWD_10	1.83 ± 0.13	1.03 ± 0.12

*Mean temperatures ± standard error of measurement

4.3 Energy Absorbed and Returned

Energy absorbed (Figure 4.1) was not significantly different between Pre, Post, and Post-15 passive tension measurements for all four treatments ($p = 0.67$). Energy returned (Figure 4.2) was significantly less ($p < 0.001$) after PSWD treatment and stayed significantly lower ($p < 0.001$) 15 minutes Post PSWD treatment for the PSWD 5/5 and PSWD_10 treatments.

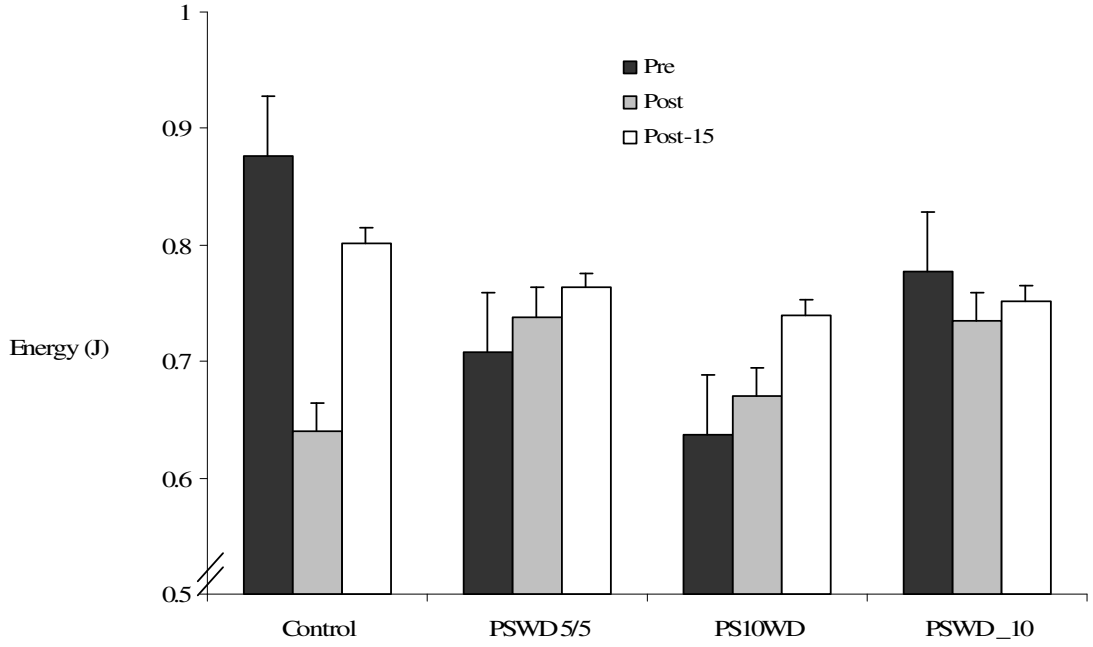


Figure 4.1: Energy absorbed: There was not a significant increase or decrease in energy absorbed for all four treatments across time.

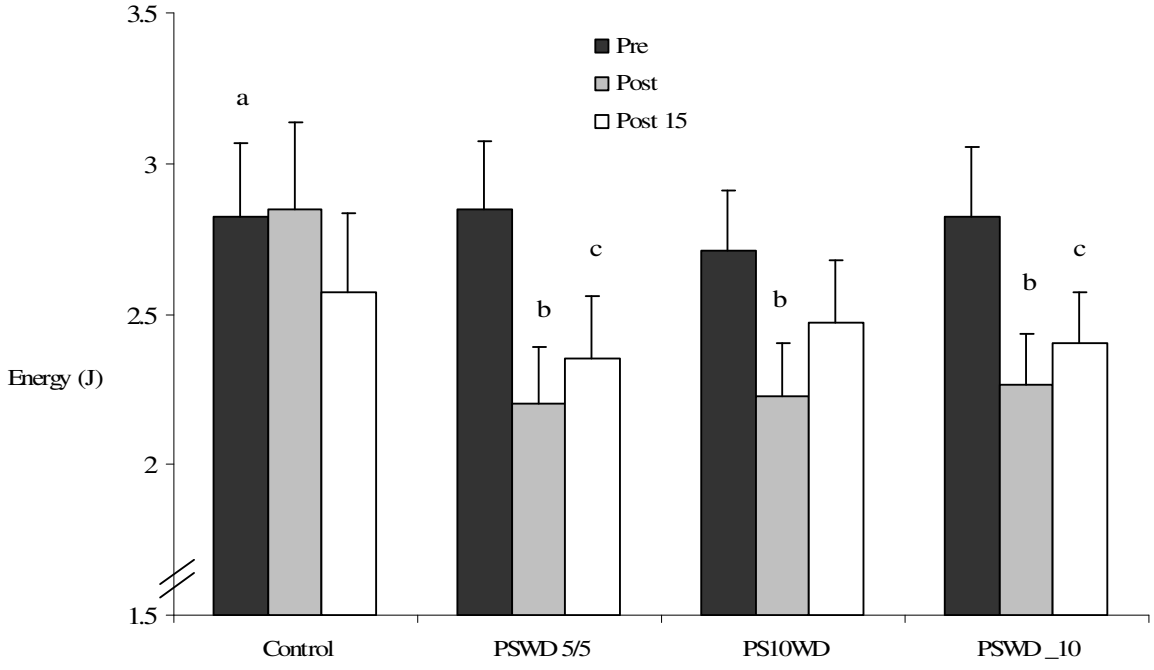


Figure 4.2: Energy returned

[a] No significant difference between Pre Control, Pre PSWD 5/5, Pre PS10WD, and Pre PSWD_10

[b] Significantly less than all Control and all Pre PSWD measurements

[c] Significantly less than all Pre measurements

4.4 Peak Torque

Peak torque (Figure 4.3) significantly decreased ($p = 0.03$) Post PSWD for all PSWD treatments. Peak torque Post-15 minutes remained lower than Pre treatment measurements for all four conditions, but was significantly lower ($p = 0.01$) for the PSWD 5/5 and PSWD_10 treatments.

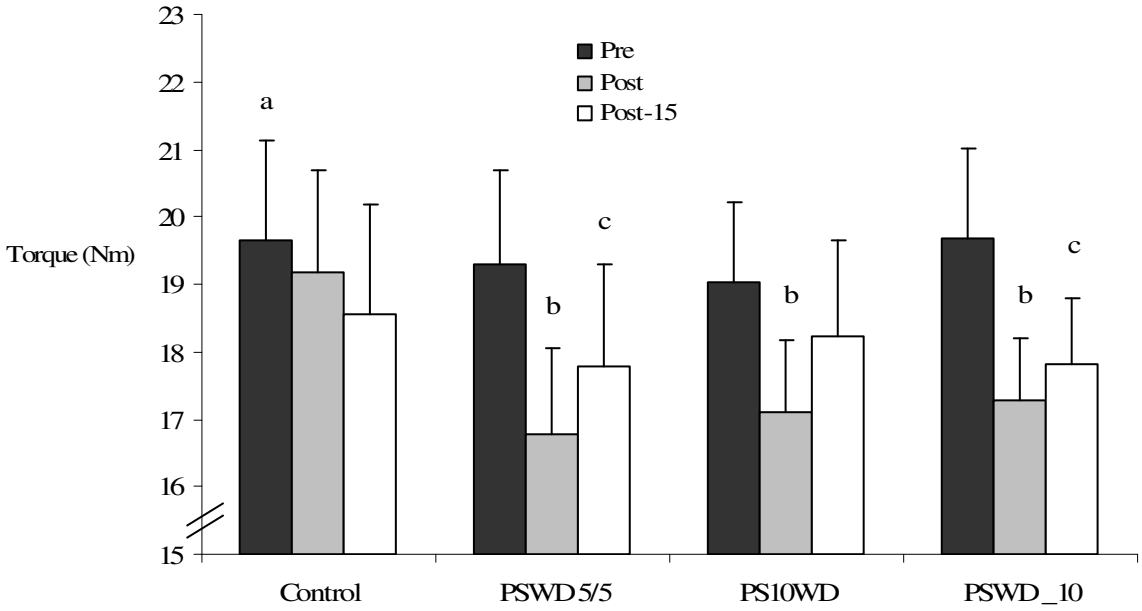


Figure 4.3: Peak torque

[a] No significant difference between Pre Control, Pre PSWD 5/5, Pre PS10WD, and Pre PSWD_10

[b] Significantly less than all Control measurements and all Pre PSWD measurements

[c] Significantly less than Pre Control

4.5 Average Stiffness

Average stiffness (Figure 4.4) was significantly less ($p = 0.01$) for Post PSWD 5/5 and PSWD_10 treatments. Post-15 PSWD treatments and the Post-15 Control average stiffness were lower than the Pre treatments measurements, but not significantly lower.

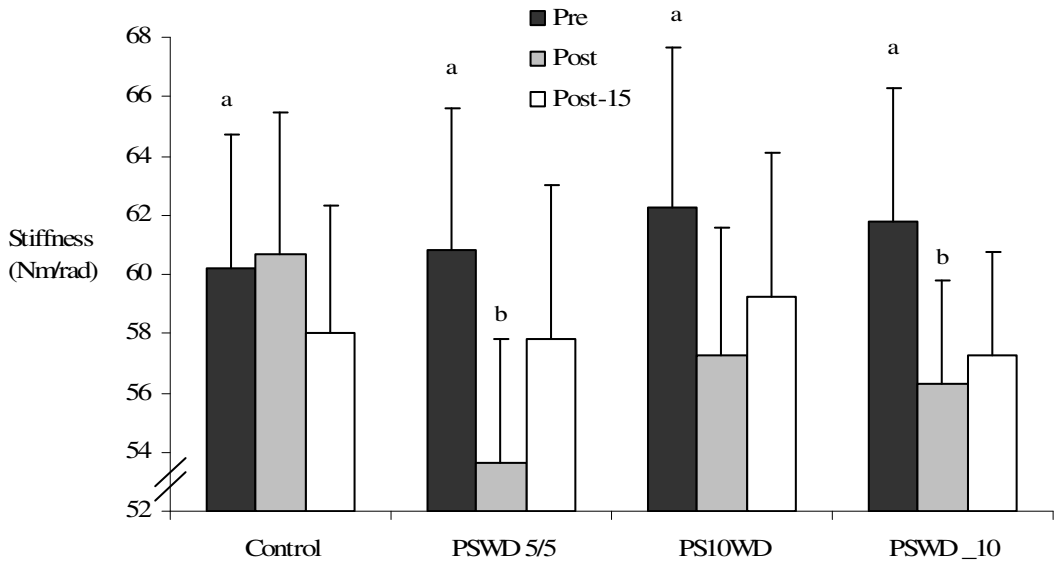


Figure 4.4: Average stiffness

[a] No significant difference between Pre Control, Pre PSWD 5/5, Pre PS10WD, and Pre PSWD_10

[b] Significantly less than Pre Control and their respective Pre PSWD treatments

4.6 Average Range of Motion

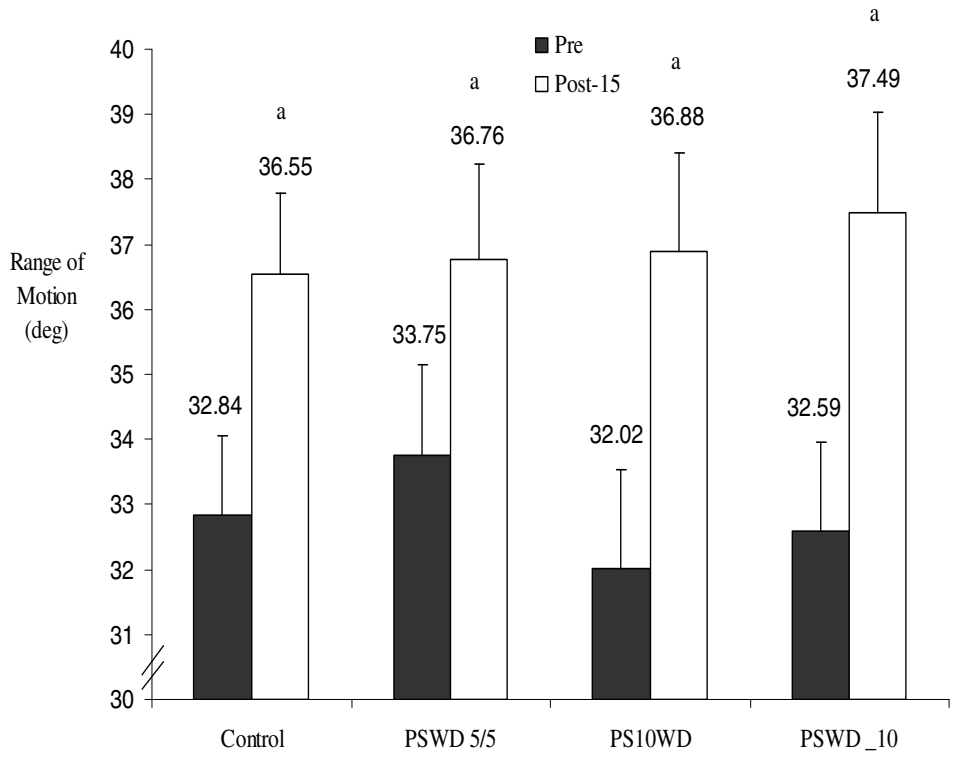


Figure 4.5: Average range of motion (ROM)

[a] Significantly greater than Pre ROM measurements

CHAPTER 5

DISCUSSION

The purpose of this study was to compare the effects of heating and the timing of stretch on the torque angle relation of the triceps surae muscle. We demonstrated that the combination of low-load long duration stretching and deep heating increased tissue compliance. Peak torque and average stiffness decreased 12% and 10%, respectively (Figure 4.3 and 4.4) from pre to post treatment for all PSWD heating and stretch treatments. Therefore, less torque was required to move the ankle complex through the 30 degree range of motion and the tissue exhibited less stiffness with the combination of heat and stretch. The post-15 PSWD and post Control peak torques remained lower than pre PSWD for all four conditions, which may be due to the cumulative nine minutes of cyclical stretch that was applied during all passive tension measurements. In addition, post 15 PSWD_10 and PSWD 5/5 peak torques, which received stretch during the cooling of the muscle, were significantly less than pre Control peak torque measurements (Figure 4.3 and 4.4). These findings support Lehmann et al.[20, 21] who demonstrated that stretch applied during both tissue heating and cooling resulted in greater elongation for rat tail tendons. They [20, 21] recommended that all stretching should be applied for a period of time after the heating has occurred and while the tissue is returning to baseline temperature.

In a previous study we found that stretch alone was as effective as stretching during diathermy in increasing tissue compliance.[3] We looked at four treatment conditions: control (no

treatment), 10 minutes stretch, 20 minutes PWSD and no stretch, and 10 minutes stretch during 20 minutes of PWSD. We found no significant decrease in average stiffness for the control group and the diathermy only group over time (pre, post, post-15 minutes, post-30 minutes) ($p>0.05$).[3] There was a significant decrease in average stiffness between pre and post treatment in the stretch group (pre: 72.49 ± 1.08 Nm/rad, post: 61.96 ± 1.08 Nm/rad) and in the diathermy and stretch group (pre: 72.02 ± 1.13 Nm/rad, post: 62.01 ± 1.13 Nm/rad). [3] Therefore, the addition of heat did not seem to increase tissue compliance. However, this study [3] did not look at the effects of the timing of stretch (stretch during heating, stretch during heating and tissue cooling, and stretch during tissue cooling after heating). In the present study we determined that stretch applied during tissue cooling demonstrated a significant decrease in peak torque and average stiffness (Figures 4.5 and 4.6). Changes in tissue compliance were most pronounced when stretching was applied during the last five minutes of PSWD treatment and continued for five minutes post treatment (PSWD 5/5).

Previous studies [8, 12, 48] of therapeutic heating with PSWD over the gastrocnemius have demonstrated an increase in intramuscular temperatures that are within the therapeutic range defined by Lehmann et al. [20, 21]. Draper et al [8] observed a 3.78 ± 1.19°C increase in intramuscular temperature of the gastrocnemius (3cm below the surface of the skin), Trowbridge et al [48], observed a 2.75 ± 1.39°C increase in intramuscular temperature of the gastrocnemius(2.5 cm below the surface of the skin), and Garrett et al.[12] observed a 3.02 ± 1.02°C, 4.58 ± 0.87°C, and a 3.28 ± 1.64°C at three sites 5cm apart within the gastrocnemius (3 cm below the surface of the skin). Additional studies have investigated the amount of heating required to cause an

increase in tissue compliance; however, they have been done on animals and *in vitro*. Warren et al.[51, 52] and Lehmann et al.[20, 21] *in vitro* studies involving rat tail tendons concluded that stretching after the tissue is heated to temperatures between 3° C to 8° C above baseline temperatures in addition to applying a stretch after heating resulted in lasting elongation of the tissue. These studies [20, 21, 51, 52] agree with our *in vivo* study of healthy human triceps surae. We achieved an average 3.5° ± 0.27° C increase in intramuscular temperature for the pulsed shortwave diathermy conditions and a lasting decrease in peak torque 15 minutes post treatment for the pulsed shortwave diathermy conditions that included stretching during tissue cooling (Figure 4.3).

The lasting increase in tissue compliance in the stretch during heating and tissue cooling (PSWD 5/5), and stretch during tissue cooling after heating (PSWD_10), leads us to believe that stretching during tissue cooling (after reaching therapeutic levels) might be more important than stretching during heating. For example, we expected the PS10WD treatment to be the most effective due to a higher intramuscular temperature (3.25° ± 0.34° C) at the time of passive tension measurements and immediate sampling of passive tension measurements after PSWD treatment. The PS10WD treatment peak torque and average stiffness (Figure 4.3 and 4.4) were significantly higher than the PSWD treatments that included stretch during cooling of the tissue (PSWD 5/5 and PSWD_10). Therefore, the effects of thixotropy and increased intramuscular temperature were not as important as applying stretch during tissue cooling in increasing tissue compliance. Lehmann's rats were right!!

We observed a significant decrease in average stiffness between post PSWD 5/5 and PSWD_10 treatments and the pre Control condition. Again our results emphasized that

stretching applied during the cooling of muscle tissue has a greater effect on tissue compliance (Figure 4.4). Although average stiffness post-15 was less than pre treatment measurements these changes were not significantly lower from the pre-treatment measurements. This observation did not coincide with the lasting decrease in peak torque post-15 observed in the PSWD 5/5 and the PSWD_10 (Figure 4.3 and 4.4). Our average stiffness results again lead us to believe that the timing of stretch does affect tissue compliance and that the best time to stretch is after the tissue has been heated sufficiently and while the tissues are cooling.

In addition, our results showed no significant differences between condition ($p = 0.66$) or time ($p = 0.05$) for energy absorption. Our results found an increase in energy absorption with increasing intramuscular temperature though the increase was not significant (Table 4.4 and Figure 4.3). Strickler et al. [47] *in vitro* study found similar results in New Zealand rabbit tibialis anterior and extensor digitorum longus muscles. They believed the corresponding increase in length and the decrease in torque explained the insignificant increase in energy absorption. In contrast, Magnusson et al [25] observed a decrease in energy absorption in an active increase in intramuscular temperature of the hamstring muscles. In the no-stretch condition no significant decrease was found between passive energy absorption pre-exercise (14.3 ± 2.3 J), post-exercise-10 (14.5 ± 3.2 J), and post-exercise-30 (13.5 ± 2.4 J). Although, in the stretching condition there was a significant decrease in passive energy absorption (10.8 ± 1.8 J) compared to the pre-exercise (14.5 ± 1.7 J) and post-exercise-10 (13.5 ± 1.9 J), but not after 30 minutes of exercise in the post-exercise-30 (13.3 ± 1.8 J). They determined that energy absorption of the hamstrings was insensitive or short-lived to active physiological increases in intramuscular temperature. [25]

Therefore, the relationship between changes in intramuscular temperature and energy absorbed is also dependent on the history of the associated tissues of a joint.

Energy returned post-treatment (Figure 4.2) was significantly lower in the three PSWD treatments. All PSWD treatments were significantly less from post Control treatment (Figure 4.2) and pre Control treatment (Figure 4.2). Kubo et al [17] suggested that an increase in energy returned may be due to an increase in tissue stiffness. From pre to post PSWD treatment conditions energy returned decreased for all three conditions, therefore, the combination of heat and stretch made the muscle-tendon complex more compliant. For all PSWD treatments energy returned increased from post to post-15 passive tension measurements (Figure 4.2). This return toward baseline measurements can be explained by the thixotropic nature of tissue. This increase in energy returned post-15 measurement indicates that the muscle tendon complex increased stiffness is due to time dependent thixotropy. [13, 19]

ROM, though difficult to accurately quantify, is an important clinical measure. In this experiment we measured ROM before the pre passive tension measurement and immediately after the post-15 passive tension measurement. Average ROM (Figure 4.5) was not significantly greater between conditions ($p = 0.26$). The change in ROM observed in the Control treatment suggests the cumulative nine minutes of cyclical stretch applied during all passive tension measurements actually increased ROM. It's possible that due to the time dependent thixotropy nature of tissue the post-15 passive tension measurement reduced muscle-tendon stiffness and may explain the increased ROM. Since the Control treatment and PSWD treatments all gained the same amount of ROM, it appears that the nine minutes of cyclical stretches were as effective

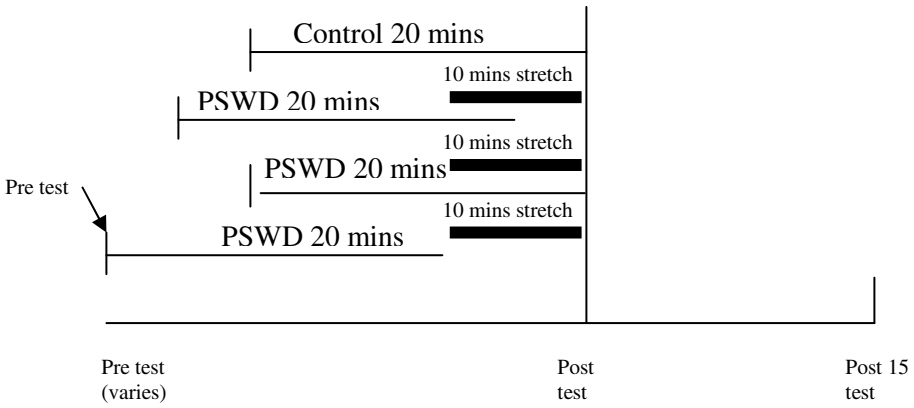
as the heat and stretch applied in the PSWD treatments. Our results are similar to those measured the first day from pre to post treatment by Peres et al. [38], Draper et al. [7], and Brucker et al. [4] The short term increase in ROM in these studies [4, 7, 38] and ours may be due to some combination of thixotropy, stretch tolerance, and mechanical stretch of the tissue.

The results of our study were limited by several factors. First we used a low-load long duration (10 minutes) static stretch and three cyclical stretches (9 minute passive tension measurements). In addition, PSWD was applied at the musculotendoneous junction, although, passive tension measurements and joint ROM are affected by the stiffness of the tendon; skin, fat, ligaments, and fascia. Since we used healthy subjects, different results may be obtained when applied to other populations such as injured tissue and/or scar tissue. Future studies should include 1) a PSWD heating and stretch regimen over time (3-6 weeks), 2) the addition of a stretch only group, 3) looking at the relationship between passive tension measurements and ROM measurements, 4) the effects of PSWD and stretch treatment on scar tissue, and 5) the effects of PSWD and stretch treatment on aged tissue.

The results of this study suggest that the timing of stretch does affect tissue compliance and that the best time to stretch is after the tissue has been heated sufficiently and while the tissues are cooling. Further studies are necessary to determine to what extent these results can be transferred into clinical practice.

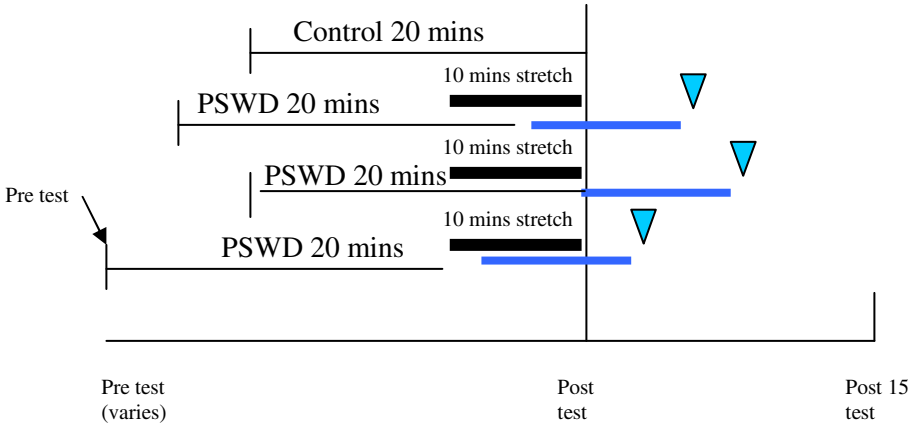
APPENDIX A

PASSIVE TENSION MEASUREMENT TIMELINE


Control 20 mins
10 mins stretch
PSWD 20 mins
10 mins stretch
PSWD 20 mins
Pre test
10 mins stretch
PSWD 20 mins
Pre test
(varies)
Post
test
Post 15
test

APPENDIX B

PASSIVE TENSION MEASUREMENT TIMELINE WITH TRIANGLES


Control 20 mins
10 mins stretch
PSWD 20 mins
10 mins stretch
PSWD 20 mins
Pre test
10 mins stretch
PSWD 20 mins
Pre test
(varies)
Post
test
Post 15
test

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BIOGRAPHICAL INFORMATION

Megan Janette Schorr was born on August 10, 1979 in Richland, Washington to John and Cheri Schorr. She is the youngest of five children (three boys and two girls). When she was nine she moved from Sunnyside, Washington to live in Boise, Idaho. She moved again when she was 14 to Chubbuck, Idaho. In June of 1997 she graduated at the top of her class from Highland High School. Three days after graduation, she moved to Provo, Utah and worked as a live in medical assistant for a paraplegic to while attending Brigham Young University.

Athletic training and research have been the focus of her educational and work experience over the last five years. She graduated from the accredited athletic training program at Brigham Young University (BYU) in April of 2005. Her clinical experience at BYU included working with the nationally ranked women's cross country team, spring football, the dance department, various high school sports, and youth sport camps. As an undergraduate she assisted her biomechanics professor with digitally analyzing the women's hammer throw for athletes who were competing at USA Track and Field events.

She has continued her development as an athletic trainer at The University of Texas at Arlington (UTA), where she is involved in the athletic training education program, clinically as an ATC, and in active research labs. She has had research opportunities in the areas of VO_2 metabolic measurements, body composition, modalities and the viscoelastic properties of muscle tissue. Since attending UTA, she has presented oral presentations at ACES and SWATA, and

poster presentations at TACSM. After graduating she is planning to work as an athletic trainer at a university, college, or high school and continue her research goals within the field of athletic training and rehabilitation.