THE THERMAL EFFECTS OF PULSED SHORTWAVE DIATHERMY ON MUSCLE FORCE PRODUCTION, ELECTROMYOGRAPHY, AND MECHANOMYOGRAPHY

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MUSCLE FORCE PRODUCTION, ELECTROMYOGRAPHY,
AND MECHANOMYOGRAPHY

by

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ABSTRACT

THE THERMAL EFFECTS OF PULSED SHORTWAVE DIATHERMY ON MUSCLE FORCE PRODUCTION, ELECTROMYOGRAPHY, AND MECHANOMYOGRAPHY

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This study investigated the thermal effects of pulsed shortwave diathermy on muscle force production, electromyography (EMG), and mechanomyography (MMG) during isometric contractions. Thirty-five men performed isometric maximal voluntary contractions (MVC) and isometric ramp contractions (10-90% MVC) before and after a 20 min treatment, and were randomly assigned to either the diathermy, sham-diathermy, or control treatment group. Intramuscular temperature was measured during the treatment, while torque, EMG amplitude, EMG instantaneous mean frequency (IMF), MMG amplitude, and MMG IMF were calculated for the MVC and ramp contractions. Temperature of the diathermy group increased 1.75°C, but decreased for the sham-
diathermy (0.43°C) and control (0.51°C) groups. The MMG amplitude and IMF increased after the diathermy treatment. The EMG and MMG amplitude and IMF increased with %MVC for all groups. These finding suggest that surface MMG may track the temperature-related increases in muscle tissue compliance and may be more sensitive to changes in the physiological conditions of the muscle than surface EMG while tracking the intrinsic motor control strategies used to modulate force production.
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CHAPTER 1

INTRODUCTION

Passive heating modalities can be used to produce various physiological effects, such as increasing the extensibility of collagen tissues;\textsuperscript{1-3} relaxing muscles;\textsuperscript{4} providing pain relief;\textsuperscript{5} and increasing blood flow.\textsuperscript{6-9} Since improvement in function is often related to these physiological effects and determined through measures related to the musculotendinous unit, the target area for these passive heating modalities is often the muscle. However, the physiological effects of the heating modality vary based on the depth of penetration. Superficial heating modalities include warm whirlpools, paraffin baths, and hot packs, which are capable of heating the tissues 1 to 2 cm deep, at best; however, deep heating modalities include ultrasound and diathermy and are capable of heating the tissues 2-3 cm deep.\textsuperscript{10} Abramson et al\textsuperscript{9} demonstrated that the application of wet heat for 20 min increased skin temperature of 6.7° C, but intramuscular temperature (3.4 cm deep) only increased 1.4° C. Similarly, Trowbridge et al\textsuperscript{11} reported an increase in skin temperature of 5.0° C and a 1.0° C increase in intramuscular temperature (2 cm deep) with the application of a ThermaCare HeatWrap. The depth of heat penetration needs to be sufficient to heat the muscles; therefore, ultrasound or diathermy should be used to attain deep heating effects. Pulsed shortwave diathermy is the application of high-frequency electromagnetic energy with a frequency of 27.12 MHz. Heat is generated as the energy passes through the tissue due to the friction between the moving
ions and the surrounding tissues.\textsuperscript{10, 12, 13} Garrett et al\textsuperscript{14} recommended pulsed shortwave diathermy over ultrasound due to the fact that it is more effective in treating a large muscle mass and retaining heat longer. Intramuscular temperature 3 cm deep increased 4.58\textdegree\ C with the diathermy treatment but increased only 0.17\textdegree\ C with the ultrasound treatment over the same size treatment area.\textsuperscript{14} Additionally, the rate of temperature decay back to baseline was considerably longer for the diathermy treatment (38.5 min) than for the ultrasound treatment (14.88 min).\textsuperscript{14}

Because heat can increase tissue extensibility and relax muscles, it is often used in conjunction with stretching exercises. The effects of heat and stretch have been investigated by numerous studies. The combination of heat and stretch provides a greater increase in tissue elongation than heat alone or stretch alone,\textsuperscript{1} while the application of a low-load stretch with increased muscle temperature will cause greater tissue elongation.\textsuperscript{3} The application of low-load, short-duration stretching with superficial heat\textsuperscript{15} or pulsed shortwave diathermy\textsuperscript{16} increased hamstring flexibility, but the increase was not significant compared to stretching alone. Peres et al\textsuperscript{17} and Draper et al\textsuperscript{18} applied pulsed shortwave diathermy with low-load, long-duration stretching and found that range of motion increases were greater with the stretching and diathermy treatment than with the control or stretch only groups. Additionally, the increases in range of motion were retained for some time after the end of treatment and stretching.\textsuperscript{17, 18} Therefore, the combination of stretching and diathermy may cause long-lasting changes to the properties of the musculotendinous unit and promote plastic deformation.
Heat studies have used changes in ROM to objectively measure changes in the length of the musculotendinous unit; however, to better understand the “true” changes that occur and assess the properties of the musculotendinous unit, other tools, such as electromyography (EMG) and mechanomyography (MMG) can be used. Surface electromyography (EMG) records the algebraic sum of electrical muscle action potentials that pass within the recording areas of the EMG electrodes. The EMG amplitude provides information about motor unit recruitment and motor unit firing rate, while the EMG frequency reflects the conduction velocity of the action potentials and the frequency of motor unit discharge. In contrast, surface mechanomyography (MMG) records and quantifies the low-frequency sounds caused by the lateral oscillations of contracting skeletal muscles. Together, the EMG and MMG signals can provide information regarding the relationships between the electrical and mechanical events of excitation-contraction coupling.

The EMG and MMG signals respond to changes in force production. Previous studies have reported that EMG amplitude increased linearly with increasing force production, but EMG frequency did not increase significantly as force production increased. Coburn et al reported that MMG amplitude increased with increasing isometric force production up to 80% but then decreased as force production rose to 100%, while MMG frequency increased non-significantly as isometric force production increased. Additionally, muscle temperature affected the EMG and MMG responses. Petrofsky et al reported a linear increase in EMG amplitude during isometric contractions with water bath temperatures above 30° C. During isometric contractions,
EMG frequency decreased with cooling and ischemia,\textsuperscript{30} increased with heat pack application,\textsuperscript{31} and increased with shortwave diathermy treatment.\textsuperscript{32} MMG amplitude decreased with cooling\textsuperscript{33} and increased with heating\textsuperscript{34}. Therefore, muscle temperature may influence the mechanical properties of the muscle and the motor control strategies employed to modulate force production.

Because pulsed shortwave diathermy produces deep heating it may induce changes to the properties of the muscles. EMG and MMG signals can be used to characterize the neurological and mechanical properties. Therefore, the purpose of this study is to investigate the thermal effects of pulsed shortwave diathermy on muscle force production, electromyography (EMG), and mechanomyography (MMG) during isometric contractions.

1.1 Hypotheses

We hypothesized that the following changes in EMG and MMG would occur with increases in temperature: 1) MMG amplitude would not change during the MVC, 2) MMG amplitude would increase during the ramp contraction, 3) EMG frequency would increase, 4) EMG amplitude would not change, and 5) MMG frequency would increase. As force production increases, we hypothesized that EMG amplitude would increase linearly and MMG amplitude would increase up to 80\% MVC and then decrease to 100\%.

1.2 Definition of Terms

Pulsed Shortwave Diathermy – high frequency (27.12 MHz) electromagnetic energy that generates heat as it passes through the tissue due to the friction between the
moving ions and the surrounding tissues. It is created by interrupting the continuous output of shortwave diathermy at consistent intervals.

**Pulse duration** – the length of time that energy is being delivered to the subject, expressed in microseconds (µs).

**Pulse frequency** – the repetition rate or the rate that the pulses are being delivered, usually expressed as pulses per second (pps).

**Surface Mechanomyography (MMG)** – the recording of vibrations produced by contracting muscle fibers. The signal is recorded by miniature accelerometers placed on the surface of the skin.

**Surface Electromyography (EMG)** – the recording of the neural activation of contracting muscle fibers from the surface of the skin.

**Intramuscular Temperature** – the temperature recorded within the muscle via an implantable thermocouple.

### 1.3 Delimitations

The delimitations of this study were: 1) males between the ages of 19 and 35 years of age from The University of Texas at Arlington, 2) males without injury to their knee, thigh, or lower leg within the past 12 months, 3) males that had a subcutaneous skinfold thickness ≤ 30 mm, and 4) males who were able to complete a successful ramp contraction for leg extension within a maximum of five attempts.
1.4 Assumptions

The following assumptions were considered throughout the study: 1) the subjects accurately completed the health history questionnaire and 2) the subjects performed the MVC and ramp contractions to the best of their ability.

1.5 Limitations

The limitations of the study were the differences in the subcutaneous skinfold thickness between the left and right thighs; changes in the room temperature between subjects; the learning effect; subject selection; subject communication; and psychological effects. Measuring skinfold thickness causes some inflammation over the measurement site which may affect the tissue temperature changes or the EMG signals; therefore, during the experimental trial, the skinfold was taken on the left thigh (non-tested limb). During the familiarization trial, the skinfold thickness was measured on both thighs to determine the similarity between the two limbs. The room temperature varied throughout the day causing it to be different for each subject. This may have affected the baseline temperature; however, all subjects sat until the intramuscular temperature did not change by more than 0.5° C over 6 consecutive 30-sec readings. Room temperature and humidity were also recorded for each subject. A familiarization trial was utilized in order to minimize the influence of a learning effect. Due to the nature of the study, it was expected that the subjects willing to participate would be predominately kinesiology students, which may have affected the results due to the subjects possessing some knowledge in the area of this study. Subject communication could have been a limitation because one subject’s experience may be different from that of another subject due to
random group assignment. If a subject communicated their experience to another subject who is in a different treatment group, they may have performed differently because they believed they were/were not receiving a treatment. Finally, there could have been a psychological effect if the subject thought that they were/were not receiving the diathermy treatment causing them to put forth more/less effort for the MVC and ramp contractions.
CHAPTER 2

REVIEW OF LITERATURE

2.1 Thermal Effects of Diathermy

2.1.1. Introduction to Passive Heating Modalities

Therapeutic heating modalities produce various physiological effects, such as increasing the extensibility of collagen tissues;\textsuperscript{1-3} relaxing muscles;\textsuperscript{4} providing pain relief;\textsuperscript{5} and increasing blood flow.\textsuperscript{6-9} Passive heating modalities include hot water baths, various types of heat packs, ultrasound, and diathermy. Hot water baths and heat packs are used for superficial heating,\textsuperscript{9,11} while ultrasound and diathermy provide deep heat.\textsuperscript{14,35-37} Abramson et al\textsuperscript{9} heated the brachioradialis muscle with wet heat for 20 and 30 min. The average increase in skin temperature was 6.7° C (20 min treatment) and 6.4° C (30 min treatment). Additionally, a thermocouple was inserted 3.4 cm deep into the muscle, and intramuscular temperature increased 1.4° C (20 min treatment) and 1.8° C (30 min treatment).\textsuperscript{9} Trowbridge et al\textsuperscript{11} examined the heating effects of the Johnson & Johnson Back Plaster, the ABC Warne-Pflaster, and the ThermaCare HeatWrap on skin and intramuscular (2 cm deep) temperature. The ThermaCare HeatWrap produced an increase in peak skin temperature of 5.0° C, but the intramuscular temperature increase was only about 1.0° C.\textsuperscript{11} These superficial heating modalities may not penetrate deep enough to heat the muscles adequately in order to cause the desired physiological effects; therefore, ultrasound or diathermy are recommended to provide deep heating effects.
Garrett et al.\textsuperscript{14} examined the changes in temperature of a 20 min pulsed shortwave diathermy application (27.12 MHz frequency; 800 bursts per second; 400\,\mu s burst duration; 850 \,\mu s interburst interval; and 48 W per burst average output) and a 20 min continuous ultrasound application (1 MHz frequency and 1.5 W/cm\textsuperscript{2} intensity) over the same size area. Three thermistors were inserted 3 cm deep into the triceps surae with the center one placed at the widest portion of the posterior surface. The remaining two were placed 5 cm superior and inferior of the center thermistor. The diathermy heated the muscle significantly more (4.58\,°C) than the ultrasound treatment (0.17\,°C); in addition, the amount of time for the temperature to decay to baseline was 38.50 min for diathermy and 14.88 min for ultrasound.\textsuperscript{14} The position of the thermistor in the treatment area also had an effect on temperature change, with the center site having a more significant change (4.6\,°C increase) than the superior (3.1\,°C increase) and inferior (3.4\,°C increase) sites; however, the temperature increase was significantly greater for all three sites with diathermy than with ultrasound.\textsuperscript{14} This indicated that diathermy heats a larger area than ultrasound and is capable of providing an equivalent amount of heat throughout the entire treatment area.\textsuperscript{14} Since diathermy is capable of heating a large area while maintaining uniform deep heating throughout the treatment area, it may be the best choice when attempting to heat the muscles to obtain therapeutic effects.

2.1.2. Introduction to Diathermy

Shortwave diathermy is the application of high-frequency (27.12 MHz) electrical energy that is used to generate heat as a result of the resistance of the tissues to the passage of energy.\textsuperscript{12, 35} The heating effects are due to the friction between the moving
ions and the surrounding tissues which is defined by the specific absorption rate (SAR). The SAR is the rate of energy absorbed per unit area of tissue. Pulsed shortwave diathermy is created by interrupting the continuous electromagnetic output of the shortwave diathermy in consistent intervals. Continuous shortwave diathermy is known for its thermal effects, while pulsed shortwave diathermy is known to have both thermal and non-thermal effects. The primary effects of pulsed shortwave diathermy include increased collagen tissue extensibility, increased blood flow, decreased pain, and increased wound healing. Indications for use of pulsed shortwave diathermy are acute and chronic pain; chronic inflammatory conditions; range of motion restrictions; muscle spasms; edema; and wounds. Pulsed shortwave diathermy is contraindicated in subjects having cardiac pacemakers; metal implants; areas of sensory loss; cancerous tissues; fluid filled areas, such as the eyes; over the epiphyseal plate in children; moist wound dressings; and moist skin. During treatment, the patient should be monitored for burns, burning sensations, and paresthesia; however, pulsed shortwave diathermy operating at a frequency of 27.12 MHz does not cause hot spots because it is not reflected by bones, as seen with ultrasound, or at tissue interfaces, as with microwave diathermy.

2.1.3. Thermal Effects of Diathermy

The thermal effects of pulsed shortwave diathermy were demonstrated by Bricknell & Watson who suggested that the thermal effects of each pulse may not dissipate in the time between pulses. They applied a 30 min pulsed shortwave diathermy treatment to the thigh with a pulse duration of 400 µs, pulse rate of 400 pps, and an initial
mean power of 1.6 W. The mean power was increased at a rate of 0.8 W every 30 seconds. Subjects were asked to inform the investigators when they thought they felt a change in temperature and then again when they were absolutely positive they felt a change in temperature. Subjects reported a definite thermal perception at an average of 6:58 minutes with an average power of 10.88 W, and mean skin temperature increase of 2.1° C, indicating thermal effects can be obtained from pulsed shortwave diathermy.\textsuperscript{40} Similarly, Murray & Kitchen\textsuperscript{38} asked the same questions to subjects who were given a 30 min treatment of pulsed shortwave diathermy. Peak power was 190 W and pulse duration was 400 µs, but pulse repetition rate ranged from 26 Hz to 400 Hz, increasing every 2 mins. A definite thermal sensation was apparent at an average pulse repetition rate of 278.8 Hz and a mean power of 21.2 W, while the average skin temperature increase was 2.34° C.\textsuperscript{38} The studies by Murray & Kitchen\textsuperscript{38} and Bricknell & Watson\textsuperscript{40} only measured skin temperature; however, several studies have measured intramuscular temperature during pulsed shortwave diathermy.\textsuperscript{14, 37, 43, 44} Draper et al\textsuperscript{37} examined the rate of temperature rise and decay in human muscle with a pulsed shortwave diathermy treatment (800 bursts per second; 400 µs burst duration; 850 µs interburst interval; and average output of 48 W per burst). The highest average temperature increase occurred at 15 mins where the increase was 3.78° C above baseline, and the temperature decreased 1.78° C over a 10 min period following the diathermy treatment.\textsuperscript{37} Draper et al\textsuperscript{43} measured intramuscular temperature changes 3 cm deep during a 20 min pulsed shortwave diathermy treatment (pulse frequency of 800 Hz, pulse width of 400 µs, and intensity of 150 W). The average temperature increase was the greatest at 15 minutes,
with a 4.0° C increase from baseline.\textsuperscript{43} Castel et al\textsuperscript{44} used a 20 min diathermy treatment, as described for Draper et al,\textsuperscript{43} and examined the rate of temperature decay after the treatment. The measured average increase in intramuscular temperature (3 cm deep) was 4.3° C, and 10 minutes after treatment, the temperature decreased 1.8° C.\textsuperscript{44} The rate of decay for pulsed shortwave diathermy is less than the decay for ultrasound\textsuperscript{14, 37, 44} which is important when heating is followed by stretching or therapeutic exercises. The depth of heat penetration produced by pulsed shortwave diathermy,\textsuperscript{14, 37, 43, 44} in conjunction with the uniform heating\textsuperscript{14} and rate of decay\textsuperscript{14, 37, 44} indicate that diathermy should provide the physiological effects of deep heating the muscles.

\subsection*{2.1.4. Blood Flow}

Increases in temperature are often associated with an increase in blood flow and tissue metabolism.\textsuperscript{6, 7, 8, 36} Akyurekli et al\textsuperscript{8} examined the changes in blood flow distribution in normal porcine skeletal muscle before, during, and after a period of regional microwave hyperthermia. After a 60 min treatment, tissue samples were taken from both hind limbs at a depth of 1.5 and 3.5 cm for microsphere assays of blood flow. The greatest increase in blood flow and temperature was located in the region of the applicator’s peak SAR, in which blood flow increased on average by a factor of 4 (10.5 ± 2.0 ml/min/100g to 43.4 ± 5.4 ml/min/100g) within 15 to 30 minutes. During this time, temperature also peaked at 44.5° C, indicating the greatest increase in blood flow occurred during the greatest increase in temperature.\textsuperscript{8} Blood pressure and pulse did not vary significantly during the treatment, therefore suggesting that the increase in blood flow is a regional phenomenon and is not due to an increase in systemic blood flow.\textsuperscript{8}
However, the initial increase in temperature was followed by a decrease and plateau, which confirms the relationship between tissue temperature and blood flow. This relationship is present when heat application increases blood flow in order to allow the heat in the tissues to dissipate from the body. Abramson et al\textsuperscript{7} examined the effects of temperature on blood flow and motor conduction velocity of the ulnar and median nerves. A 25 min shortwave diathermy treatment was applied to the forearm while placed in a plethysmograph at bath temperatures of 34° C and 4° C. There was a significant rise in median and ulnar motor nerve conduction velocity and tissue temperature during the diathermy treatment in the 34° C and 4° C bath; however forearm blood flow was increased only in the 34° C bath. Abramson et al\textsuperscript{7} suggested that changes in motor nerve conduction velocity are observed in conjunction with changes in tissue temperature but not with changes in local blood flow. Additionally, Sekins et al\textsuperscript{6} examined the local relationship between muscle temperature and the corresponding rates of blood flow with microwave diathermy. There was an approximately linear relationship between temperature and responding blood flow response when the temperatures reached their peak.\textsuperscript{6} Once the temperature peaked, a decrease was observed due to dissipation of heat through the increase in blood flow, which is similar to the results of Akyurekli et al\textsuperscript{8}.

2.1.5. Connective Tissue Extensibility

Several studies have demonstrated that connective tissue extensibility can be increased through passive heating; however, in most cases greater increases were observed when heat was applied in addition to a load or stretch.\textsuperscript{1, 2, 3, 15-18} Lehmann et al\textsuperscript{1} and Warren et al\textsuperscript{3} examined the effects of temperature and load on the rat tail tendon and
found that the length was increased by the greatest amount when the temperature was at 45° C. In addition, Lehmann et al\textsuperscript{1} compared the change in tendon length during heat alone and load alone and found that neither produced a significant increase in length. The application of both heat and low-load, long-duration stretching resulted in the greatest tendon length increase, suggesting that temperature and load may affect the visco-elastic properties of the tendon.\textsuperscript{3} Similarly, Strickler et al\textsuperscript{2} examined the effect of passive heat on the lengthening of rabbit muscles to failure. When muscles were warmed 4° C (35° C to 39° C), a greater increase in length was achieved before the muscle failed; therefore, suggesting that passive warming increases musculotendinous extensibility and may reduce susceptibility to strain injuries.\textsuperscript{2}

Several studies,\textsuperscript{15-18} have demonstrated that stretching combined with passive heating results in increases in human muscle extensibility. Taylor et al\textsuperscript{15} applied a 77° C hot pack or a -18° C gel cold pack to the posterior thigh for 20 minutes followed by one continuous stretch of the hamstring for one minute. Range of motion increased the most with heat and stretching, however, the increase was not significantly greater than stretch alone.\textsuperscript{15} Similarly, Draper et al\textsuperscript{16} examined the effects of low-load, short-duration stretching only; a combination of low-load, short-duration stretching and a pulsed shortwave diathermy treatment; and no treatment (control) on hamstring range of motion. The increases in hamstring flexibility were not significantly greater with stretching and diathermy than with stretching alone.\textsuperscript{16} However, when low-load, long-duration stretching is applied in conjunction with a diathermy treatment, greater ranges of motion have been achieved.\textsuperscript{17, 18} Peres et al\textsuperscript{17} compared 3 weeks of long-duration stretching
alone to 3 weeks of pulsed shortwave diathermy treatment with long-duration stretching and found that range of motion increased more with the stretching and diathermy than with stretching alone. The range of motion remained increased for 6 days after the treatment period for the stretching and diathermy treatment suggesting that low-load, long-duration stretching may affect the plastic deformation of the muscle. Finally, Draper et al examined the changes in hamstring flexibility with pulsed shortwave diathermy and prolonged stretching, sham diathermy, and no treatment (control) over a five day period. The daily changes in range of motion were similar between stretching alone and stretching plus diathermy; however, the cumulative increases in range of motion over the five day period were greater for the stretching plus diathermy group. Therefore, it was suggested that plastic deformation can occur during low-load, long-duration stretching and passive heating. Warren et al demonstrated that the effect of low-load stretch and heat application caused elongation of the connective tissue, while Draper et al and Peres et al found that low-load, long-duration stretching with pulsed shortwave diathermy treatments caused increases in range of motion. These increases in range of motion were still present after a period of rest indicating that this type of stretching and heat application may affect the plastic deformation of the muscles.

2.2 Surface Electromyography and Mechanomyography

2.2.1. Introduction to Surface Electromyography and Mechanomyography

A muscle contraction is caused by the excitation of motor units, which is accompanied by an electrical change across the membranes of the activated muscle cells. Electromyography (EMG) records the algebraic sum of electrical muscle action potentials
that pass within the recording areas of the EMG electrodes. The EMG amplitude quantifies muscle activation and is determined by two processes: 1) the number of motor units recruited and 2) the firing rates of the activated motor units. The frequency of the EMG signal, however, is said to reflect the conduction velocity of the action potentials, and to some degree, the frequency of motor unit discharge. Together, the amplitude and frequency of the EMG signals may provide information regarding motor control strategies and fiber-type recruitment patterns.

As a muscle contracts, it produces tension which causes changes in the muscle fiber geometry related to the sarcomere shortening. The number of motor units recruited affects the amount of muscle surface displacement due to changes in the longitudinal or transverse dimensions of the muscle. Muscle activity is thought to be related to the vibrations or sounds that can be detected as the muscle contracts. In addition, the properties of muscle activity are related to the properties of contraction. Sounds that are generated by muscles as they contract have been referred to by many names, including mechanomyography, acoustic myography, sound myography, phonomyography, vibromyography, and accelerometermyography. The 1995 CIBA Foundation Symposium, however, suggested that the term “surface mechanomyogram” be used to avoid terminological confusion. The surface mechanomyogram (MMG) non-invasively records and quantifies the low-frequency sounds caused by lateral oscillations of contracting skeletal muscles. It has been suggested that these lateral oscillations are generated by a) gross lateral movement of the muscle at the initiation of a contraction that is generated by non-simultaneous activation of muscle fibers, b) smaller subsequent
lateral oscillations occurring at the resonant frequency of the muscle, and c) dimensional changes of the active muscle fibers.\textsuperscript{23} Orizio\textsuperscript{23} suggested that the MMG time and frequency domains may provide information regarding motor unit recruitment and firing rate in contracting muscles.

Studies have shown that the MMG amplitude reflects motor unit recruitment;\textsuperscript{21,23,25,29} however, this information is different from that provided by the EMG amplitude. EMG amplitude reflects muscle activation, which is due to both motor unit recruitment and firing rate (i.e., rate coding), therefore, there is often a linear or curvilinear increase in EMG amplitude from 0 to 100\% of a maximal voluntary contraction (MVC).\textsuperscript{21} Conversely, MMG amplitude generally increases from 0 to 80\% of MVC, and then plateaus or decreases to 100\%,\textsuperscript{25} which may reflect only motor unit recruitment. The MMG amplitude, however, is also affected by muscle temperature,\textsuperscript{34} stiffness, mass, intramuscular pressure, and the viscosity of the fluid media surrounding the muscle fibers.\textsuperscript{24}

It has been suggested that the frequency of the MMG signal reflects the global firing rate of the activated motor units.\textsuperscript{21} This information cannot be segregated from the amplitude or frequency of traditional surface EMG, since EMG frequency is most often related to muscle action potential conduction velocity. The MMG frequency generally exhibits a pattern of increase from 0 to 100\% of MVC,\textsuperscript{21,23,25} with a sharp rise from 80 to 100\%,\textsuperscript{25} which may indicate the use of rate coding to increase muscle force production once the number of recruited motor units reaches its maximal potential. Overall, surface MMG and EMG recorded simultaneously can provide unique information about the
relationships among the electrical and mechanical events of excitation-contraction coupling.\textsuperscript{23, 25-28}

2.2.2. Temperature Responses

Several studies have examined the effects of temperature on EMG amplitude and frequency.\textsuperscript{22, 30-32} Petrofsky & Lind\textsuperscript{22} examined the changes in surface EMG frequency and amplitude during brief and prolonged isometric contractions with changes in temperature as a result of a 30-min water bath (10, 20, 30, and 40° C). A linear relationship was present between the EMG amplitude and tension for brief contractions at temperatures above 30° C; however, the relationship was curvilinear at temperatures below 30° C. Petrofsky & Lind\textsuperscript{22} suggested that the differences may be due to the temperature effect on the muscle which may alter recruitment of the alpha motor neuron pool. In addition, EMG frequency progressively shifted to lower frequencies during the contraction, and there was a linear reduction in the center frequency as muscle temperature decreased.\textsuperscript{22} Merletti et al\textsuperscript{30} investigated the changes in median frequency of the myoelectric signal with local ischemia or reduction of intramuscular temperature during isometric contractions. Initial median frequency (IMF) was decreased with both ischemia and cooling conditions, with IMF recovering quickly after ischemia but gradually after cooling.\textsuperscript{30} Merletti et al\textsuperscript{30} suggested that the decrease was due to the accumulation of byproducts from the lack of blood flow, which may decrease the conduction velocity of the muscle fibers and in turn decrease the median frequency of the myoelectric signal. Since cooling causes vasoconstriction, blood flow was reduced causing the median frequency to respond to cooling similarly to ischemia.\textsuperscript{30} Similarly,
Madigan & Pidcoe\textsuperscript{32} observed an increase in EMG mean power frequency with increasing muscle temperature. Subjects received a shortwave diathermy treatment and then performed a 40\% MVC with each 0.5-1.0\textdegree{} C decrease in muscle temperature. Additionally, subjects cycled at 3 workloads (25, 50, and 75\% \textit{VO}_{2max}) until exhaustion and performed a 5-s isometric contraction at 10 min intervals. With the cycling, the linear increase in EMG mean power frequency was only seen at 25 and 50\% \textit{VO}_{2max}\textsuperscript{32}. Increasing muscle temperature generally increases EMG frequency; however, fatigue testing decreases EMG frequency. Therefore, at the higher intensity cycling (75\% \textit{VO}_{2max}) fatigue can decrease EMG mean power frequency more quickly than a rise in temperature can increase it.\textsuperscript{32} Finally, Krause et al\textsuperscript{31} examined the effects of cooling and heating on EMG amplitude and frequency during isometric contractions (10, 30, 50, and 80\% MVC). Cooling was achieved by an ice pack reducing temperatures to 28\textdegree{} C, while heating was with a heat pack to 45\textdegree{} C. There was an increase in EMG frequency with heat and a decrease with cold, which was significant at 30\% MVC and above.\textsuperscript{31} Additionally, as the MVC increased from 10\% to 80\%, both EMG frequency and amplitude increased during heat and cold.\textsuperscript{31} Therefore, muscle temperature may influence the motor control strategies employed to modulate force production.

Kimura et al\textsuperscript{33} and Barry\textsuperscript{34} examined the effects of temperature on MMG amplitude and frequency. Kimura et al\textsuperscript{33} decreased intramuscular temperature to 15\textdegree{} C using icepacks and stimulated the triceps surae muscles with a supramaximal single twitch once the temperature was at 15, 20, and 25\textdegree{} C. MMG amplitude was significantly reduced as a result of the cooling, while peak force was decreased and contraction time
was prolonged. This demonstrated that hypothermia could reduce the muscle contractile and relaxation properties, therefore, indicating that MMG may be useful in studying the contractile properties of muscles.\textsuperscript{33} Barry\textsuperscript{34} investigated force development and MMG properties during maximal isometric twitches at temperatures of 7.0 to 25.0° C and lengths of 75 to 110% of optimal length. The MMG amplitude increased as muscle temperature increased from 7.0 to 25.0° C and as muscle length increased up to 90% of the optimal length. Since the MMG amplitude decreased at lengths above 90%, Barry\textsuperscript{34} suggested that the lateral movement is restricted by the internal compliance of the muscle, which is reduced by stretching the muscle. This is due to the reduction in muscle’s internal compliance during a stretch of the muscle, in contrast to the increase in muscle compliance immediately following a stretch to the muscle. Therefore, MMG can provide information regarding the mechanical properties of a muscle.\textsuperscript{34}

2.2.3. Force Responses

Several studies have demonstrated that EMG and MMG signals respond differently during isometric contractions than during isokinetic contractions.\textsuperscript{21, 27, 29, 45} Beck et al\textsuperscript{21} examined EMG and MMG amplitude and mean power frequency (MPF) versus torque relationship during isokinetic muscle actions of the biceps brachii at 20, 40, 60, and 80% of peak torque. There was a linear increase for MMG and EMG amplitude with isokinetic force, but no change for MMG and EMG MPF. Since there was no change in the time and frequency domains, Beck et al\textsuperscript{21} suggested that dynamic torque production may be modulated more by recruitment than by increases in the global motor unit firing rate during isokinetic muscle actions. Similarly, Coburn et al\textsuperscript{29} suggested that
different patterns of MMG amplitude and frequency may reflect differences in the motor control strategies that modulate torque production during isometric versus dynamic muscle actions. Subjects performed leg extensions at isokinetic rates of 10, 20, 30, 40, 50, 60, 70, 80, and 90% of peak torque and isometric rates of 10, 20, 30, 40, 50, 60, 70, 80, and 90% MVC. The isometric MMG amplitude increased to 80% MVC, plateaued from 80 to 90% MVC, and then decreased. This plateau or decrease in MMG amplitude at high levels of force production may be due to muscle stiffness, intra-muscular fluid, or fusion of the motor unit twitches at high firing rates. If isometric force production causes muscle stiffness at higher intensities of MVC, the lateral oscillations of the muscle may be reduced causing the decrease in the MMG signal. The MMG amplitude versus torque relationship was linear in isokinetic muscle actions but cubic in isometric muscle actions, therefore indicating that differences may be present in the motor control strategies that modulate submaximal to maximal torque production between the two types of muscle actions. This supports the findings of Beck that dynamic muscle actions may modulate force production by recruitment rather than rate coding. Madeleine et al. compared the EMG and MMG responses during concentric, eccentric, and isometric contractions at 0, 25, 50, 75, and 100% MVC. There were non-linear MMG/force and EMG/force relationships which demonstrate differences in activation strategies during isometric, concentric, and eccentric muscle actions. Additionally, the EMG amplitude increased during isometric and concentric contractions from 0 to 50% MVC and from 0 to 75% MVC during eccentric contractions, while MMG amplitude increased from 0 to 50% MVC with concentric contractions. Madeleine et al. suggested that the type of
contraction, level of contraction, and angular velocity affected the electromechanical efficiency, which can be defined as the amount of mechanical activity per muscle activation or MMG:EMG ratio. Bilodeau et al.\textsuperscript{45} compared EMG frequency during voluntary isometric contractions and electrically stimulated contractions. There was a positive correlation between EMG frequency and the rate of torque development during voluntary contractions; however, during electrically stimulated contractions, the correlation was negative between EMG frequency and the rate of torque development.\textsuperscript{45} Bilodeau et al.\textsuperscript{45} suggested that factors other than muscle fiber composition may influence EMG frequency and the rate of torque production. In addition, the effect of these factors may differ between voluntary and elicited contractions.\textsuperscript{45}

Isometric ramp contractions have been used to evaluate the EMG and MMG signals.\textsuperscript{46, 47} Bilodeau et al.\textsuperscript{46} had subjects perform leg extensions with a gradually increasing force from 0 to 100% MVC over a 6-s period and found that EMG amplitude increased progressively with increasing force. The MMG responses during a ramp contraction of 5 to 80% MVC at a rate of 10% MVC per second were examined by Akataki et al.\textsuperscript{47} The MMG amplitude progressively increased with force levels until 60% MVC was reached at which point, amplitude decreased quickly.\textsuperscript{47} MMG mean power frequency increased rapidly until 30% MVC, followed by a temporary reduction at 50% MVC, and then increased again above 60% MVC.\textsuperscript{47} Akataki et al.\textsuperscript{47} suggested that ramp contractions may provide deeper insights into the motor unit activation strategies of the muscles, and can ensure higher force resolution in the MMG/force relationship. Finally, Akataki et al.\textsuperscript{28} examined the MMG/force relationship of the first dorsal interosseous
muscle and the biceps brachii during ramp contractions of 5 to 70% MVC over a 6.5-s period. MMG amplitude/force and MMG mean power frequency/force relationships differed between muscles, which indicated that motor unit activation strategies vary between muscles. Additionally, the motor unit’s rate coding may have a more prominent role in force production than motor recruitment in the small first dorsal interosseous muscle compared to the relatively large biceps brachii.
CHAPTER 3

METHODS

3.1 Design

This study incorporated three separate experimental designs, one for each condition: (1) intramuscular temperature, (2) the isometric maximal voluntary contractions (MVC), and (3) the isometric ramp contractions. For intramuscular temperature, a $3 \times 7$ (treatment $\times$ time) mixed factorial design was used to examine the effects of pulsed shortwave diathermy on intramuscular temperature. The independent variables were treatment group and time. The treatment group contained 3 factors: diathermy, sham-diathermy, and control. Time had 7 factors: baseline temperature, 5 min of treatment, 10 min of treatment, 15 min of treatment, 20 min of treatment, 5 min post-treatment, and 10 min post-treatment. The dependent variable was intramuscular temperature ($^\circ$C).

For the isometric MVCs, a $2 \times 3$ (time $\times$ treatment) mixed factorial design was used to examine the effects of pulsed shortwave diathermy on muscle torque production, electromyography (EMG), and mechanomyography (MMG). The independent variables for this design were time and treatment group. Time had 2 factors: pre- and post-treatment, and the treatment group contained 3 factors: diathermy, sham-diathermy, and control. The dependent variables were MVC torque (Nm), EMG amplitude ($\mu$Vrms), EMG frequency (Hz), MMG amplitude ($m\cdot s^{-2}$rms), and MMG frequency (Hz).
For the isometric ramp contractions, a $2 \times 3 \times 9$ (time × treatment × %MVC) mixed factorial design was used to examine the effects of pulsed shortwave diathermy on the EMG and MMG signals. The independent variables for this design were time, treatment group, and percent of MVC (%MVC). Time had 2 factors: pre- and post-treatment, and treatment group contained 3 factors: diathermy, sham-diathermy, and control. Percent of MVC contained 9 factors: 10%, 20%, 30%, 40%, 50%, 60%, 70%, 80%, and 90% of MVC. The dependent variables were torque, EMG amplitude, EMG frequency, MMG amplitude, and MMG frequency (all ramp contraction values were expressed as a percentage of the pre-treatment MVC values, respectively)

The treatment procedures, consisting of either diathermy, sham-diathermy, or control conditions, were single-blinded (with the exception of the control group). Custom-made covers were placed over the diathermy machine so that subjects in the diathermy and sham-diathermy groups were unaware of the operational status of the diathermy machine or if they were actually receiving the treatment. For the diathermy group, the diathermy machine was in place and operational and the subjects received a treatment. For the sham-diathermy group, the machine was in place but was not operational, and the subjects did not receive a treatment. For the control group, the diathermy machine was not present during the treatment time.

### 3.2 Subjects

Thirty-five apparently healthy men volunteered for this study (mean age ± SD = 24.06 ± 4.50 yrs; height ± SD = 197.70 ± 6.64 cm; mass ± SD = 82.75 ± 12.86 kg; right thigh skinfold ± SD = 14.62 ± 6.54 mm). Each subject was randomly assigned to one of
the three groups: diathermy (n=13), sham-diathermy (n=12), or control (n=10). During the familiarization trial, each subject completed an informed consent and a health status questionnaire. Subjects were excluded from the study if they indicated health risks due to cardiopulmonary, metabolic, or coronary heart disease. Subjects with a thigh skinfold thickness $\geq 30$ mm, a cardiac pacemaker, injury within the past 12 months, or metal plates or screws in their right knee, thigh, or lower leg were also excluded. This study was approved by the University Institutional Review Board for Human Subject Research.

3.3 General Procedures

Each subject participated in a familiarization and experimental trial. During the familiarization, each subject performed two 3-s isometric MVCs and at least 5 isometric ramp contractions. The isometric ramp contractions were performed until the subject successfully completed the protocol (based on visual inspection) within a maximum of 12-15 attempts. The purpose of the familiarization trial was for the subjects to gain experience with the testing equipment and to practice the MVC and ramp contractions. Within 2-5 days after the familiarization trial, subjects reported to the laboratory for the experimental trial, which consisted of the following procedures (in order): (1) 2 pre-treatment isometric MVCs, (2) 2-3 pre-treatment isometric ramp contractions, (3) thermocouple insertion, treatment, and intramuscular temperature recording, (4) 2-3 post-treatment isometric ramp contractions, (5) 2 post-treatment isometric MVCs, and (6) thermocouple removal (Figure 3.1). All tests and measures were recorded from the right thigh for each subject.
Figure 3.1 Timeline of events for experimental testing

The amount of time required for each step of the procedure (mean ± SD); total time = A – G = 64.49 ± 5.59 min. A = Subject prep (shave leg, clean skin, place electrodes): 10.91 ± 4.05 min. B = Pretreatment MVC and ramp contractions: 7.46 ± 1.07 min. C = Thermocouple insertion and obtaining a baseline intramuscular temperature: 8.23 ± 2.46 min. D = Treatment (determined by group placement: Diathermy, Sham-diathermy, or Control): 20.0 ± 0.0 min. E = Time between treatment and post-treatment ramp and MVC contractions: 0.83 ± 0.75 min. F = Post-treatment ramp and MVC contractions: 7.71 ± 1.36 min. G = Removal of electrodes and thermocouple, cleansing of skin, and application of antibacterial ointment and bandaid: 8.00 ± 2.65 min.

3.4 Intramuscular Temperature Measurements

A 16-channel Isothermex (Isothermex, Columbus, OH) was used to record intramuscular temperature to the nearest ± 0.1°C every 30 seconds for 30 minutes. Prior to all data collection, the Isothermex was factory calibrated (over a range of -20°C to 80°C) as well as custom calibrated using a 3-temperature procedure (center point = 37°C). The Isothermex was interfaced to a desktop computer and temperatures were sampled at 30 second intervals throughout the 30 min session; however, intramuscular temperature was recorded at baseline and at 5 min intervals during the 20 min treatment period and up to 10 minutes post-treatment. Because the diathermy unit interfered with the Isothermex recordings, the diathermy unit was paused for 5 seconds at each 5 min interval to allow the Isothermex to sample intramuscular temperature. An intramuscular-implantable thermocouple (Physitemp Instruments, Type IT-21 (diameter = .41 mm), Clifton, NJ) sensor was used to measure temperature 2.5 cm deep in the vastus lateralis of each subject.
Before each use, all intramuscular-implantable thermocouples were sterilized in Cidex Plus 28 day (Johnson & Johnson, Irivne, CA) for at least 24 hours. Prior to sterilization, the intramuscular-implantable thermocouples were marked at 5-cm from the tip of the sensor so the depth of placement could be accurately estimated.

Prior to the thermocouple insertion, skinfold measurements were taken at the site of insertion, and skinfold thickness (mm) was determined by the average of three measures. Subjects were eligible for inclusion if their skinfold measure was ≤ 30 mm to ensure that the intramuscular thermocouple insertion of 2.5 cm was fully within the vastus lateralis muscle.

After the pre-treatment isometric MVCs and ramp contractions, a 3 × 3 cm area surrounding the site for intramuscular thermocouple insertion was cleansed with providine-iodine. The thermocouple was inserted at 62% of the distance from the anterior superior iliac spine (ASIS) to the superolateral border of the patella. The thermocouple was inserted perpendicular to the skin surface to a depth of 2.5 cm via a 20-gauge 1.25 in (3.15 cm) sterile intravenous catheter (Medex, Inc., Model 3056, Carlsbad, CA). After insertion of the catheter and needle into the vastus lateralis, the needle was removed and the implantable thermocouple was threaded through the catheter tube into the vastus lateralis muscle. Threading the thermocouple into the catheter until the 5 cm mark on the thermocouple reached the top of the catheter controlled the initial insertion depth. The catheter was then removed and the distance between the skin surface and the 5 cm mark on the thermocouple was measured. Because the thermocouple was intentionally inserted deeper than necessary it was withdrawn until the mark is 2.5 cm
above the skin surface. Blenderm surgical tape (3M Healthcare, St. Paul, MN) was used near the insertion site to secure the thermocouple and to prevent extraction of the thermocouple during the experiment. The thermocouple was connected to the Isothermex and the connections were verified through real-time temperature measurement.

Prior to treatment, the baseline temperature was recorded. For this study, baseline was determined when the intramuscular temperature did not change more than 0.5°C over 6 consecutive 30 second readings.

3.5 Group Treatments

Immediately following the baseline intramuscular temperature measurement, the subject received a treatment based on group assignment. The diathermy group received a 20-min pulsed shortwave diathermy treatment over the vastus lateralis muscle. The diathermy unit was a Megapulse II machine (Accelerated Care Plus, Sparks, NV) with a frequency of 27.12 MHz. The diathermy was set to a treatment time of 20 minutes, pulse duration of 400 µs, pulse rate of 800 pps, and average output power of 40 W. The sham-diathermy group sat for 20 min with the diathermy drum in place over the vastus lateralis. The diathermy machine was on, but the treatment was not started, thus no energy was emitted from the drum. The control group sat for 20 min between the pre- and post-treatment measurements.

3.6 Muscle Strength Assessments

Immediately before (pre) and after (post) the treatment conditions, the subjects performed 2 isometric MVCs and 2-3 isometric ramp contractions on a Biodex System 3 isokinetic dynomometer (Biodex Medical Systems, Inc., Shirley, NY). Each subject was
seated on the Biodex chair with restraining straps over the trunk, pelvis, and contralateral thigh in accordance with the Biodex User’s Guide (Biodex Pro Manual, Applications/Operations. Biodex Medical Systems, Inc., Shirley, NY. 1998). Using a hand-held goniometer, the dynamometer lever arm was fixed in a position that was consistent with a knee joint angle of 60° below the horizontal plane. All isometric leg extension contractions (MVC and ramp) were performed with the right leg.

3.6.1. Isometric Maximal Voluntary Contractions

Two 3-s isometric MVCs were conducted before the first and after the last isometric ramp contraction (Figure 3.1). Subjects were asked to produce as much force as possible for 3 sec during which strong verbal encouragement was provided. Two minutes of rest was allowed between each MVC trial. Peak MVC torque was calculated as the highest average torque value that occurred during any 0.5-s duration within the 3-s MVC. The highest MVC torque value between the 2 MVC trials was used as the representative value. The pre-treatment isometric MVC that yielded the highest torque value was used as the 100% torque value during the subsequent isometric ramp contractions at pre- and post-treatment.

3.6.2. Isometric Ramp Contractions

For the isometric ramp contractions, subjects were required to track their torque production on a computer monitor placed in front of them that displayed their real-time torque signal overlayed onto a programmed ramp template. The ramp template consisted of a 5-s horizontal baseline at 5% MVC followed by a linearly increasing ramp line lasting 6 sec and ending with a 2-s horizontal plateau at 100% MVC. The isometric
ramp contractions had the following characteristics: a) pre-treatment: duration = 6.292 ± 0.422 sec, r = 0.9963 ± 0.0026, and SEE = 6.13 ± 2.22 and b) post-treatment: duration = 6.326 ± 0.482 sec, r = 0.9957 ± 0.0033, and SEE = 6.49 ± 2.48. During the experimental trial, subjects were asked to perform 2-3 ramp contractions (5-100% MVC) during which verbal encouragement was provided, and a 2-min rest period was allowed between each ramp contraction. The ramps that best satisfied the following criteria at pre- and post-treatment were used for later analysis: (1) maximal force reaching at least 90% of MVC and (2) a smooth linear increase in force through visual inspection. The isometric ramp template and real-time torque overlay was programmed using LabVIEW 7.1 software (National Instruments, Austin, TX).

3.7 Surface Electromyographic Measurements

A bipolar surface electrode arrangement was placed along the longitudinal axis of the vastus lateralis muscle (Figure 3.2). The center of the bipolar electrode arrangement (20 mm inter-electrode distance) was placed at 75% of the distance from the anterior superior iliac spine (ASIS) to the superolateral border of the patella. One electrode was placed over the spinous process of the seventh cervical vertebrae to serve as a reference electrode. Electrodes were placed according to Hermens et al.\textsuperscript{48} to avoid overlap with the innervation zone and cross-talk between muscles. Local areas of the skin were cleaned with alcohol and lightly rubbed with emery paper to keep the interelectrode impedance below 5,000 Ohms. The EMG signals (recorded in microvolts) were differentially amplified with a bandwidth of 1 to 500 Hz, input impedance of 2MΩ (differential), common mode rejection ratio of 110 dB, maximum input voltage of ±10 V, sampling
rate of 1,000 Hz, and gain of 1,000 (EMG100C, Biopac Systems). Pre-gelled, disposable EMG electrodes containing a 1-cm diameter Ag-AgCl disc (Moore Medical, New Britain, CT) were used for this study.

3.8 Surface Mechanomyographic Measurements

The MMG signal was detected with an active miniature accelerometer (EGAS-FS-10/-VO5, Entran Inc., Fairfield, NJ) that was preamplified with a gain of 200, frequency response of 0 to 200 Hz, sensitivity of \(70 \text{ mV/m.s}^{-2}\), and range of \(\pm 98 \text{ m.s}^{-2}\). The accelerometer was placed over the vastus lateralis muscle at 50% of the distance from the ASIS to the lateral border of the patella, superior to the active EMG electrodes. The accelerometer was fixed to the skin with 3M double-sided foam tape and 3M removable double-coated tape (Figure 3.2).
3.9 Signal Processing

During each isometric MVC and ramp contraction, the EMG, MMG, and torque signals were recorded simultaneously with a Biopac data acquisition system (MP150WSW, Biopac Systems, Inc., Santa Barbara, CA). The analog torque (Nm) signal from the Biodex dynamometer was sampled at 125 points·s\(^{-1}\), while the EMG (µV) and MMG (m·s\(^{-2}\)) signals recorded from the vastus lateralis were sampled at 1,000 points·s\(^{-1}\). All signals were stored on a personal computer (Dell Optiplex GX260, Dell, Inc., Round Rock, TX) for off-line analysis. All digital signal processing was completed off-line using custom written software (LabVIEW v 7.1, National Instruments, Austin, TX). The EMG and MMG signals were bandpass filtered with a zero-phase 8\(^{\text{th}}\)-order Butterworth filter with bandwidths of 10-500 Hz and 5-100 Hz, respectively. The torque signal was low-pass filtered (5 Hz cutoff) with a zero-phase 8\(^{\text{th}}\)-order Butterworth filter. All subsequent analyses in the time and frequency domains were performed on the filtered signals.

MVC torque (Nm) was determined as the highest 0.5-s average torque value that occurred during the 3-s MVC (Figure 3.3). The EMG and MMG signals were expressed as root mean square (rms) amplitude values that corresponded with the 0.5-s window for MVC torque. Average instantaneous mean frequency values (Hz) were also calculated from the time-frequency representations of the EMG and MMG signals that corresponded with the 0.5-s window for MVC torque.
Figure 3.3 EMG, MMG, and torque signals recorded during the MVC.

An example of the surface EMG (top), EMG instantaneous mean frequency (second), MMG (third), MMG instantaneous mean frequency (forth), and torque (bottom) signals during a pre-treatment isometric MVC. The surface EMG, MMG, and torque signals were recorded during the MVC, while the EMG and MMG time-frequency representations were constructed off-line with a continuous wavelet transform (CWT). The gray shaded region represents the 0.5-s period that resulted in the highest MVC torque value, thus, this epoch of each signal was extracted for analysis.
Isometric ramp torque values (Nm) were determined as the 0.1 sec average torque values that best represented the 10%, 20%, 30%, 40%, 50%, 60%, 70%, 80%, and 90% MVC increments during the 6-s ramp contraction (Figure 3.4). EMG and MMG rms amplitude values were calculated for each 0.1-s window that represented the incremental torque values (10-90% MVC). Average instantaneous mean frequency values (Hz) were also calculated from the time-frequency representations of the EMG and MMG signals that corresponded with the 0.1-s windows during the ramp contraction (10-90% MVC).

### 3.9.1. Time-Frequency Analysis

Time-frequency representations of the EMG and MMG signals were constructed using instantaneous mean frequency (IMF) estimates (Hz) provided by a continuous wavelet transform (CWT) function defined as:49

\[
CWT(a,b) = \int m(t)\psi^*_a(b,t)dt
\]

Where \(m(t)\) was the input signal, \(a \in \mathbb{R}^+\) represents the scale parameter, \(b \in \mathbb{R}\) represents the translation parameter, and \(\psi^*_{a,b}(t)\) was obtained by scaling the prototype (mother) wavelet \(\psi(t)\) at time \(b\) and scale \(a\):

\[
\psi^*_{a,b}(t) = \frac{1}{\sqrt{a}}\psi\left(\frac{t-b}{a}\right)
\]

When scale \(a\) becomes large, the prototype wavelet was dilated, yet the shape of the wavelet was maintained. The more dilated the prototype wavelet, the lower its mean frequency, therefore, it is useful in characterizing the low-frequency components of the EMG and MMG signals. Conversely, when scale \(a\) becomes small, the prototype was compressed, yielding higher mean frequencies, thus, better resembling the high-
Figure 3.4 EMG, MMG, and torque signals recorded during the ramp contraction.
An example of the surface EMG (top), EMG instantaneous mean frequency (second), MMG (third), MMG instantaneous mean frequency (forth), and torque (bottom) signals during a pre-treatment isometric ramp contraction. The surface EMG, MMG, and torque signals were recorded during the ramp contraction, while the EMG and MMG time-frequency representations were constructed off-line with a continuous wavelet transform (CWT). The gray shaded regions represent the 0.1-s epochs that were extracted for analysis to represent 10 – 90% MVC.
frequency components of the EMG and MMG signals. This time-scale representation has an equivalent time-frequency expression that was characterized by:

\[ a = \frac{f_0}{f} \]

Based on Karlsson et al.\textsuperscript{49} we used a complex Morlet prototype wavelet with a center frequency of 1,000 Hz at scale \( a = 1 \) and a length of 126 at the coarsest scale (i.e., the minimum length of the wavelet). For the EMG signals, we modulated the prototype wavelet from scale \( a = 1 \) to \( a = 100 \) with 1-scale intervals, which provided 100 scales with center frequencies ranging from 1,000 – 10 Hz, respectively. For the MMG signals, we modulated the prototype wavelet from scale \( a = 1 \) to \( a = 200 \) with 2-scale intervals, which provided 100 scales with center frequencies ranging from 1,000 – 5 Hz, respectively. IMF values were obtained at each instant of the signal by the following equation:

\[
IMF(t) = \frac{\int_0^{F} \omega P(t, \omega) d\omega}{\int_0^{F} P(t, \omega) d\omega}
\]

Where \( F \) was the Nyquist frequency and \( P(t, \omega) \) was the time-dependent power spectral density (i.e., the power spectrum with \textit{scale} on the x-axis and \textit{scalogram coefficient magnitude}\textsuperscript{2} / \textit{scale} on the y-axis for each instant in the time domain of the original signal).
3.10 Statistical Analysis

All data recorded during the isometric ramp contractions (torque, EMG amplitude, EMG frequency, MMG amplitude, and MMG frequency) were normalized to their respective values obtained during the pre-treatment isometric MVC that resulted in the highest mean torque value. Therefore, the torque, EMG amplitude, EMG frequency, MMG amplitude, and MMG frequency values during the isometric ramp contractions were expressed as a percentage of the corresponding pre-treatment MVC values (%MVC), and these normalized values were used in the statistical analyses. The intramuscular temperature and pre- and post-MVC values, however, were expressed in their absolute units for statistical analysis.

A two-way mixed factorial ANOVA (TIME [baseline vs. 5 min vs. 10 min vs. 15 min vs. 20 min vs. 25 min vs. 30 min] × TREATMENT [diathermy vs. sham-diathermy vs. control]) was used for the intramuscular temperature values (°C). Five separate two-way mixed factorial ANOVAs (TIME [pre- vs. post-treatment] × TREATMENT [diathermy vs. sham-diathermy vs. control]) were used to analyze the torque (Nm), EMG amplitude (µVrms), EMG frequency (Hz), MMG amplitude (m·s⁻²rms), and MMG frequency (Hz) values recorded during the pre- and post-treatment isometric MVCs. Five separate three-way mixed factorial ANOVAs (TIME [pre- vs. post-treatment] × TREATMENT [diathermy vs. sham-diathermy vs. control] × %MVC [10% vs. 20% vs. 30% vs. 40% vs. 50% vs. 60% vs. 70% vs. 80% vs. 90%]) were used to analyze the torque, EMG amplitude, EMG frequency, MMG amplitude, and MMG frequency values (all expressed at %MVC) recorded during the pre- and post-treatment isometric ramp
contractions. A step down process\textsuperscript{50} was used to interpret each ANOVA, which began with the highest order interactions. If the interactions were significant, the statistical model was decomposed to analyze the simple effects with Bonferroni corrections. If the interactions were not significant, the significant main effects were analyzed with Bonferroni corrections. In addition, the strength of association (effect size) was estimated with the eta squared ($\eta^2$) statistic.\textsuperscript{51} For statistical interactions and main effects involving a repeated measures independent variable, the partial $\eta^2$ was calculated. For between-subjects main effects, the standard $\eta^2$ was calculated. For convenience, both the partial and standard $\eta^2$ will be represented as $\eta^2$ throughout the study. The a priori type I error rate was set at 5\%, and all statistics were performed with SPSS 11.5 software (SPSS Inc., Chicago, IL).
CHAPTER 4

RESULTS

4.1 Intramuscular Temperature

There was a significant two-way interaction (TIME × TREATMENT, p<0.001, \(\eta^2=0.776\)). Intramuscular temperature increased for the diathermy group ((p<0.001, \(\eta^2=0.883\)), but decreased for the sham-diathermy (p<0.001, \(\eta^2=0.595\)) and control (p=0.045, \(\eta^2=0.343\)) groups across time (baseline, 5 min of treatment, 10 min of treatment, 15 min of treatment, 20 min of treatment, 5 min post-treatment, and 10 min post-treatment) (Figure 4.1). There were no differences in intramuscular temperature among the treatment groups at baseline (p=0.940) or 5 min of treatment (0.055). However, the diathermy group was significantly greater than the sham-diathermy and control groups at 10 min of treatment (p<0.001, \(\eta^2=0.571\), 15 min of treatment (p<0.001, \(\eta^2=0.784\)), 20 min of treatment (p<0.001, \(\eta^2=0.865\)), 5 min post-treatment (p<0.001, \(\eta^2=0.785\)), and 10 min post-treatment (p<0.001, \(\eta^2=0.667\)). During the diathermy treatment, the mean temperature increase was 1.75°C, which occurred at 20 min of treatment.

4.2 Muscle Strength

For the isometric maximal voluntary contraction (MVC) torque (Figure 4.2), there were no two-way interactions (time × treatment; p=0.166) and no main effects for time (p=0.231), but a significant main effect for treatment (p=0.042, \(\eta^2=0.185\)). The
marginal means for torque (collapsed across time) indicated that the control group was greater ($p=0.038$) than the diathermy group.

![Intramuscular Temperature Graph](image)

**Figure 4.1 Intramuscular temperature.**

Intramuscular temperature at 5 min intervals from baseline to 10 min post-treatment for the diathermy, sham-diathermy, and control groups. There were significant differences among time for the diathermy, sham-diathermy, and control group as follows:

**Diathermy:**
- Baseline < 5, 10, 15, 20 min of treatment, and 5 and 10 min of post-treatment ($p=0.001-0.004$)
- 5 min of treatment < 10, 15, 20 min of treatment, and 5 and 10 min of post-treatment ($p<0.001$)
- 10 min of treatment < 15, 20 min of treatment, and 5 and 10 min of post-treatment ($p=0.001-0.004$)
- 15 min of treatment < 20 min of treatment and 5 min of post-treatment ($p=0.001, 0.008$)
- 20 min of treatment > 10 min of post-treatment ($p=0.014$)
- 5 min of post-treatment > 10 min of post-treatment ($p=0.001$)

**Sham-diathermy:**
- Baseline > 5, 10, 15, 20 min of treatment, and 5 min of post-treatment ($p=0.001-0.010$)
- 5 min of treatment > 10, 15, and 20 min of treatment ($p=0.001-0.003$)
- 10 min of treatment < 15, and 20 min of treatment ($p=0.006, 0.001$)
- 15 min of treatment > 20 min of treatment ($p=0.007$)

**Control:**
- Baseline > 5, 10, 15, and 20 min of treatment ($p=0.001$)
- 5 min of treatment > 10, 15, and 20 min of treatment ($p=0.001-0.009$)
- 10 min of treatment < 15, and 20 min of treatment ($p=0.001, 0.010$)

The differences between group for each time period are as follows:

**Baseline:** Diathermy = Sham-diathermy = Control ($p=0.940$)
- 5 min of treatment: Diathermy = Sham-diathermy = Control ($p=0.055$)
- 10 min of treatment: Diathermy > Sham-diathermy and Control ($p<0.001$)
- 15 min of treatment: Diathermy > Sham-diathermy and Control ($p<0.001$)
- 20 min of treatment: Diathermy > Sham-diathermy and Control ($p<0.001$)
- 5 min of post-treatment: Diathermy > Sham-diathermy and Control ($p<0.001$)
- 10 min of post-treatment: Diathermy > Sham-diathermy and Control ($p<0.001$)
Figure 4.2 Torque during MVCs.

Isometric maximal voluntary torque (Nm) from pre- to post treatment. There was a significant difference for torque (collapsed across time) for the groups as follows:

Control > Diathermy (p=0.038)

For the isometric ramp torque (Figure 4.3), the analysis indicated no three-way interaction (time \times treatment \times \%MVC, p=0.197), no two-way interactions (time \times \%MVC, p=0.146; treatment \times \%MVC, p=0.155), but a significant two-way interaction (time \times treatment, p=0.032, η²=0.194) and main effect for \%MVC (p<0.001, η²>0.999). The marginal means (collapsed across time and treatment) for \%MVC indicated that torque increased (p<0.001) as \%MVC increase; 10\% < 20\% < 30\% < 40\% < 50\% < 60\% < 70\% < 80\% < 90\%. Subsequent analysis of the time \times treatment interaction indicated no differences from pre- to post-treatment for the diathermy (p=0.615), sham-diathermy (p=0.232), or control (p=0.110) groups and no differences between treatment groups at pre-treatment (p=0.067) or at post-treatment (p=0.223).
Figure 4.3 Torque during ramp contractions.

Normalized Torque (%MVC) from pre- to post-treatment for the (a) diathermy group, (b) sham-diathermy group, and (c) control group. There were significant differences between %MVC (collapsed across time and group) as follows: 10% < 20% < 30% < 40% < 50% < 60% < 70% < 80% < 90% (p<0.001)
4.3 Surface Electromyographic Amplitude

EMG amplitude for the isometric MVC had no two-way interaction (time × treatment, p=0.077) and no main effect for treatment (p=0.471), but a significant main effect for time (p=0.002, η²=0.273) (Figure 4.4). The marginal means (collapsed across treatment) for EMG amplitude decreased from pre- to post-treatment.

![Figure 4.4 EMG amplitude during MVCs.](image)

EMG Amplitude (µVrms) from pre- to post treatment. There was a significant difference in EMG amplitude (collapsed across group) for time as follows: pre-treatment > post-treatment (p=0.002)

The analysis of isometric ramp EMG amplitude indicated no three-way interaction (time × treatment × %MVC; p=0.091), no two-way interactions (time × %MVC, p=0.929; treatment × % MVC, p=0.497; time × treatment, p=0.288), no main effect for time (p=0.551), but significant main effect for %MVC (p<0.001, η² =0.928). The marginal means (collapsed across time and treatment) for %MVC increased as %MVC increased (Figure 4.5); 10% < 20% < 30% < 40% < 50% < 60% < 70% < 80% < 90% (p<0.001-0.001).
Figure 4.5 EMG amplitude during ramp contractions.

Normalized EMG Amplitude (%MVC) from pre- to post-treatment for the (a) diathermy group, (b) sham-diathermy group, and (c) control group. There were significant differences between %MVC (collapsed across time and group) as follows: 10% < 20% < 30% < 40% < 50% < 60% < 70% < 80% < 90% (p<0.001-0.001).
4.4 Surface Electromyographic Instantaneous Mean Frequency

EMG instantaneous mean frequency for the isometric MVC (Figure 4.6) had no two-way interactions (time × treatment, p=0.653) and no main effect for treatment (p=0.221) or time (p=0.182).

Figure 4.6 EMG IMF during MVCs.
EMG Average Instantaneous Mean Power Frequency (Hz) from pre- to post treatment.

The analysis of isometric ramp EMG instantaneous mean frequency indicated no three-way interaction (time × treatment × %MVC; p=0.211), no two-way interaction (time × %MVC, p=0.253; time × treatment, p=0.841), but a significant two-way interaction (%MVC × treatment; p=0.034, \(\eta^2=0.113\)) and a main effect for time. The marginal means (collapsed across treatment and %MVC) for EMG frequency increased from pre- to post-treatment (p=0.023, \(\eta^2=0.151\)). Results of the analysis of the treatment × %MVC interaction (collapsed across time) indicated that there were differences between %MVC for the diathermy group (p<0.001; \(\eta^2=0.539\)), sham-diathermy group (p<0.001; \(\eta^2=0.644\)), and control group (p<0.001; \(\eta^2=0.587\)). There were also significant differences in EMG frequency among treatment groups (collapsed across time) at the
following %MVCs: 40% (p=0.002; $\eta^2=0.318$), 50% (p=0.001; $\eta^2=0.344$), 60% (p=0.003; $\eta^2=0.300$), and 70% (p=0.020; $\eta^2=0.208$). The specific differences are noted in the caption of Figure 4.7.

Figure 4.7 EMG IMF during ramp contractions.
Normalized EMG Average Instantaneous Mean Power Frequency (%MVC) from pre- to post-treatment for the (a) diathermy group, (b) sham-diathermy group, and (c) control group. There was a significant main effect for time (collapsed across group and %MVC) as follows: Pre-treatment < post-treatment (p=0.023). There were significant differences between groups (collapsed across time) at the following %MVCs:

- **40% MVC:** Sham-diathermy > Control (p=0.002)
- **50% MVC:** Diathermy > Control (p=0.011)
  - Sham-diathermy > Control (p=0.001)
- **60% MVC:** Diathermy > Control (p=0.004)
  - Sham-diathermy > Control (p=0.019)
- **70% MVC:** Sham-diathermy > Control (p=0.026)

(a) There were significant differences among %MVC (collapsed across time) for the diathermy group at the following %MVCs:

- 10% MVC < 40%, 50%, 60%, 70%, 80%, and 90% MVC (p=0.001-0.006)
- 20% MVC < 50%, 60%, and 90% MVC (p=0.013-0.020)

(b) There were significant differences among %MVC (collapsed across time) for the sham-diathermy group at the following %MVCs:

- 10% MVC < 40%, 50%, 60%, 70%, 80%, and 90% MVC (p=0.001-0.010)
- 20% MVC < 40%, 80%, and 90% (p=0.001-0.006)
- 30% MVC < 40%, 50%, 70%, 80%, and 90% (p=0.002-0.042)

(c) There were significant differences among %MVC (collapsed across time) for the control group at the following %MVCs:

- 10% MVC < 90% MVC (p=0.022)
- 20% MVC < 80% and 90% MVC (p=0.016, 0.003)
- 30% MVC < 90% MVC (p=0.004)
- 40% MVC < 90% MVC (p=0.002)
- 50% MVC < 80% and 90% MVC (p=0.038)
- 60% MVC < 90% MVC (p=0.011)
- 70% MVC < 90% MVC (p=0.019)

4.5 Surface Mechanomyographic Amplitude

MMG amplitude for the isometric MVC (Figure 4.8) had no two-way interaction (time × treatment, p=0.064), no main effect for treatment (p=0.072), but a main effect for time (p=0.001, η²=0.318). The marginal means (collapsed across treatment) for MMG amplitude increased from pre- to post-treatment.

The analysis of isometric ramp MMG amplitude indicated no three-way interaction (time × treatment × %MVC; p=0.595), no two-way interaction (time × %MVC, p=0.131; treatment × %MVC, p=0.915), but a significant two-way interaction (time × treatment; p=0.004, η²=0.291) and main effect for %MVC (p<0.001, η²=0.659).
The marginal means (collapsed across time and treatment) for MMG amplitude increased as %MVC increased (Figure 4.9). Analysis of the time × treatment interaction indicated that MMG amplitude increased from pre- to post-treatment for the diathermy group only (p=0.003, $\eta^2=0.530$). Additionally, there were no differences (collapsed across %MVC) between the diathermy, sham-diathermy, or control groups at pre-treatment (p=0.216) or at post-treatment (p=0.139).

![Figure 4.8 MMG amplitude during MVCs.](image)

MMG Amplitude ($m \cdot s^{-2} \cdot rms$) from pre- to post treatment. There was a significant difference in MMG amplitude (collapsed across group) for time as follows: pre-treatment < post-treatment (p=0.001)
Normalized MMG Amplitude (%MVC) from pre- to post-treatment for the (a) diathermy group, (b) sham-diathermy group, and (c) control group. There were significant differences among %MVC (collapsed across time and group) for the following %MVCs: 10% MVC < 40%, 50%, 60%, 70%, 80%, and 90% MVC (p=0.001-0.028)
20% MVC < 30%, 40%, 50%, 60%, 70%, 80%, and 90% MVC (p=0.001-0.005)
30% MVC < 40%, 50%, 60%, 70%, 80%, and 90% MVC (p=0.001-0.005)
40% MVC < 50%, 60%, 70%, 80%, and 90% MVC (p<0.001)
50% MVC < 70%, 80%, and 90% MVC (p=0.001-0.035)
There were significant differences for pre- to post-treatment for each group as follows: Diathermy: pre-treatment < post-treatment (p=0.003)
4.6 Surface Mechanomyographic Instantaneous Mean Frequency

MMG instantaneous mean frequency for the isometric MVC (Figure 4.10) had no two-way interactions (time × treatment, p=0.150), no main effect for treatment (p=0.376) but a main effect for time (p=0.001, $\eta^2=0.297$). The marginal means (collapsed across treatment) for MMG frequency increased from pre- to post-treatment.

For the isometric ramp MMG instantaneous mean frequency, there was no three-way interaction (time × treatment × %MVC; p=0.520), no two-way interactions (time × %MVC, p=0.516; %MVC × treatment, p=0.811), but a significant two-way interaction (time × treatment, p=0.004, $\eta^2=0.292$) and main effect for %MVC (p<0.001, $\eta^2=0.343$). The marginal means (collapsed across time and treatment) for %MVC indicated some significant differences occurred to MMG frequency, which are listed in the caption of Figure 4.11. Analysis of the time × treatment interaction indicated that MMG frequency increased (p=0.003, $\eta^2=0.542$) from pre- to post-treatment for the diathermy treatment only. In addition, there was no difference between the diathermy, sham-diathermy, or control groups at pre-treatment (p=0.865) or at post-treatment (p=0.317).

![Figure 4.10 MMG IMF during MVCs.](image)

MMG average instantaneous mean power frequency (Hz) from pre- to post-treatment. There was a significant difference in MMG frequency (collapsed across group) for time as follows: pre-treatment < post-treatment (p=0.001)
Figure 4.11 MMG IMF during ramp contractions.

Normalized MMG Average Instantaneous Mean Power Frequency (%MVC) from pre- to post-treatment for the (a) diathermy group, (b) sham-diathermy group, and (c) control group. There were significant differences among %MVC (collapsed across time and group) for the following %MVCs:

10% MVC < 60%, 70%, 80%, and 90% MVC (p=0.001-0.009)
20% MVC < 50%, 60%, 70%, 80%, and 90% MVC (p=0.001)
30% MVC < 50%, 60%, 70%, 80%, and 90% MVC (p=0.001)
40% MVC < 50%, 60%, 70%, and 80% MVC (p=0.001-0.008)

There were significant differences from pre- to post-treatment among group as follows: Diathermy: pre-treatment < post-treatment (p=0.003)

There was no significant difference between groups at pre-treatment or at post-treatment. Pre-treatment: Diathermy = Sham-diathermy = Control (p=0.865). Post-treatment: Diathermy = Sham-diathermy = Control (p=0.317).
CHAPTER 5
DISCUSSION

In this study, the 20-min diathermy treatment (27.12 MHz, 400 µs, 800 pps, and 40W) elicited a mean increase in intramuscular temperature (2.5 cm deep) of the vastus lateralis of 1.75 ± 0.39°C. The greatest increase in temperature (0.57 ± 0.14°C) was observed from 15 to 20 min of treatment. However, the 20-min sham-diathermy and control treatments elicited mean decreases in temperature of 0.43°C and 0.51°C, respectively. Two previous intramuscular temperature studies\textsuperscript{14, 37} reported intramuscular temperatures (3 cm deep) in the gastrocnemius muscle during 20 min pulsed shortwave diathermy treatments (27.12 MHz, 800pps, 400 µs, and 48W). Garrett et al\textsuperscript{14} reported a peak temperature increase of 4.58°C, while Draper et al\textsuperscript{37} reported an increase of 3.78°C; however, neither study reported data from a control group.

The magnitude of intramuscular temperature increase (1.75°C °C) in response to the 20 min diathermy treatment of the vastus lateralis muscle in the present study was not as profound as those reported by Garrett et al\textsuperscript{14} and Draper et al\textsuperscript{37} after similar treatments of the gastrocnemius muscle. However, it is possible that four factors, including treatment dosage, blood flow, room temperature, and skinfold thickness, may have contributed to this discrepancy. First, the treatment dosage was different in that the average output power used in our study was 40 W, where as the previous studies\textsuperscript{14, 37} used an average output power of 48 W. This difference in energy output is probably
responsible for the smaller degree of temperature increase. Second, previous studies\(^6-9\) have demonstrated that tissue temperature and blood circulation influence each other. Blood flow plays an important role during therapeutic heat treatments because it allows for the dissipation of heat by delivering cool blood to the area in an attempt to maintain the body’s desired “constant” temperature. Abramson et al\(^7\) demonstrated that skin temperature was dependent on blood flow while applying a heat pack to the thigh. They measured skin temperature while varying the amount of blood supply. While the superficial heat was being applied, the blood supply was occluded causing the skin temperature to increase to a peak value. When the heat was removed, but the blood flow remained occluded, the tissues began to slowly cool.\(^7\) However, when the blood flow was restored the skin temperature decreased at a more rapid rate.\(^7\) Abramson et al\(^7\) suggested that if skin temperature decreases rapidly, it will be unable to conduct heat to the deeper muscles. Therefore, our mean increase in temperature may be lower than those reported by Garrett et al\(^14\) and Draper et al\(^37\) because our target area, the thigh and vastus lateralis, may be more perfused with blood because it is closer to the core and it is a larger muscle than the gastrocnemius. Third, heat may have been lost to the environment in our study. Neither Garrett et al\(^14\) nor Draper et al\(^37\) reported the mean room temperature, nor did they have a control group; therefore, it is difficult to make direct comparisons based on the environmental conditions of the testing laboratories. For our experimental trials, the mean room temperature was 22.19 ± 0.40°C and mean percent humidity was 39.23 ± 9.44%. These conditions may have allowed for dissipation of heat to the environment as demonstrated by the decreases in temperature during the 20
min treatment time for the sham-diathermy and control groups. Additionally, many of the subjects reported feeling cool in the laboratory during testing while wearing shorts and a t-shirt. Therefore, the smaller increase in temperature for the diathermy group and the decrease in temperature for the sham-diathermy and control groups may reflect that the temperature of the diathermy group was affected by heat lost to the ambient air.

Fourth, skinfold thickness can affect the degree of temperature increase because adipose tissue is a poor conductor of electromagnetic energy. Our subjects had a mean thigh skinfold thickness of 15.13 ± 6.67 mm. Hattori et al measured skinfold thicknesses at 15 different sites for male subjects (n=121; age 18-23 years) and reported a mean thigh skinfold thickness of 17.3 ± 6.37 mm and medial calf skinfold thickness of 9.1 ± 4.42 mm. This suggests that the thigh has more subcutaneous fat than the medial calf; therefore, the smaller increase in temperature in our study compared to Draper et al and Garrett et al may be due to the thigh having more adipose tissue than the calf.

In addition to recording temperature during the treatment, we also recorded temperature at 5 and 10- min post-treatment. The subjects in the diathermy group had a temperature increase of 0.06 ± 0.39 °C at 5-min post-treatment but a decrease of 0.49 ± 0.39°C at 10 min post-treatment, while subjects in the sham-diathermy and control groups increased (0.27 ± 0.29°C and 0.05 ± 0.55°C, respectively) at 10 min post-treatment. This amount of temperature decay was smaller than previously reported, where temperature dropped 1.78°C at 10 min post-treatment and 1.0°C in 7.65 min post-treatment. The smaller amount of decay during the 10 min post-treatment period of the present study is probably a result of the post-treatment isometric ramp and MVC.
contractions, which may have raised the intramuscular temperature, or was a result of a smaller increase in temperature at 20 min of treatment.

Surface MMG records and quantifies the lateral oscillations or vibrations that are generated by activated skeletal muscle fibers. These oscillations are highly correlated with the fluctuating dimensional changes of the muscle fibers\(^{25}\) that, in turn, reflect the minute fluctuations in force output (i.e., force ripple).\(^{23, 25, 53, 54}\) The MMG signal has been studied in detail,\(^{23, 53-55}\) and as a result, it has been postulated that the MMG signal reflects the inherent contractile properties of activated motor units.\(^{33}\) It has also been suggested, however, that the amplitude of the surface MMG signal is directly related to the compliance of the active musculotendinous unit.\(^{23, 56-60}\) In theory, increases in physiological temperature will increase the compliance of both the contractile and non-contractile tissues in the muscle. The non-contractile proteins and connective tissues at the levels of the sarcomere and muscle fiber, respectively, provide the structural rigidity necessary for the actin and myosin filaments to generate force in series,\(^{61}\) therefore, temperature-related increases in the compliance of the non-contractile tissues may allow for greater lateral oscillations of the contractile proteins (actin and myosin) during contraction. Furthermore, previous studies have demonstrated temperature-related increases in the extensibility (i.e., compliance) of a musculotendinous unit,\(^{15-18}\) which is thought to be limited by the non-contractile components of the muscle. Therefore, as muscle compliance increases with temperature, it is possible that the muscle fibers would not be as constricted and would be allowed to laterally oscillate to a greater extent. In support of this hypothesis, previous studies have demonstrated decreases in MMG
amplitude caused by experimentally-induced hypothermia in vivo\textsuperscript{33} and in vitro\textsuperscript{34}. Kimura et al\textsuperscript{33} concluded that the surface MMG may be a useful and reliable method for monitoring the contractile properties of active skeletal muscle under a wide range of physiological conditions. However, until now, no previous studies have characterized the responses of the MMG signal under experimentally-induced muscle temperature increases. The results of the present study supported those of Barry\textsuperscript{34} and Kimura et al\textsuperscript{33} and indicated significant increases in MMG amplitude in response to the diathermy treatment but not for the sham-diathermy or control treatments. Therefore, these results suggested that MMG amplitude may reflect changes in the contractile properties of the active motor units that occur as a result of increases in muscle temperature. Specifically, in conjunction with previous studies,\textsuperscript{33, 34} these findings suggested that MMG amplitude may track the changes in muscle compliance that occur with changes in muscle temperature.

From submaximal to maximal isometric force production, it has been demonstrated that MMG amplitude increases with increasing force production up to approximately 80\% MVC, followed by a decrease or plateau to 100\% MVC.\textsuperscript{25} During isometric ramp contractions, however, MMG amplitude may increase to approximately 60\% MVC, followed by a decrease to 80\% MVC.\textsuperscript{62} Orizio et al\textsuperscript{25} suggested that the increase in MMG amplitude and eventual plateau or decrease may reflect the contributions of motor unit recruitment to increases in force production up to approximately 60-80\% MVC until motor unit recruitment is maximized and rate coding takes over to further increase force production. Hence, the plateau or decrease in MMG
amplitude from 60-80% MVC to 100% MVC may reflect the limits of motor unit recruitment and/or the fusion of twitches during the rate coding process. The patterns of response for MMG amplitude in the present study supported those of previous studies\textsuperscript{21, 25, 29, 62, 63} and increased from 10 to 70% MVC, followed by a plateau or decrease to 90% MVC. This pattern was observed for all three groups (diathermy, sham-diathermy, and control) during both the pre- and post-treatment isometric ramp contractions. Interestingly, this pattern of response was not significantly altered in response to the diathermy treatment, despite an upward shift in normalized MMG amplitude (Figure 4.9.a). This finding suggested that although MMG amplitude may have tracked the temperature-related increases in muscle compliance as evident by the upward shift, it was still able to track the potential changes in motor unit recruitment that may have modulated force production under both the normal (pre) and experimentally increased (post) physiological temperatures.

During the isometric MVCs in the present study, MMG amplitude increased from pre- to post-treatment for all groups (diathermy, sham-diathermy, and control) (Figure 4.8). It should be noted, however, that the magnitude of increase for the diathermy group was larger than the increases for the sham-diathermy and control groups, but this difference was not sufficient to elicit a significant interaction (p=0.064). This may be due to the fact that the pre-treatment MVCs were performed first, without any previous muscle contractions, while the post-treatment MVCs were performed last, after the post-treatment ramp contractions (Figure 3.1). Small increases in intramuscular temperature in the sham-diathermy and control groups at 5- and 10-min post-treatment indicated that
the post-treatment ramp and MVC contractions may have caused increases in muscle temperature (Figure 4.1). Therefore, it is possible that the increases in MMG amplitude during the isometric MVCs for the sham-diathermy and control groups from pre- to post-treatment may have resulted from the contraction-induced increases in muscle temperature.

In addition to the increases in MMG amplitude, MMG instantaneous mean frequency (IMF) also increased with intramuscular temperature for the diathermy group, but no changes were observed from pre- to post-treatment for the sham-diathermy or control conditions (Figure 4.11). To our knowledge, this is the first study to examine the effects of increased intramuscular temperature on the frequency domain of the MMG signal. It has been suggested that the frequency domain of the MMG signal reflects the global firing rate of the activated motor units.\textsuperscript{21, 25, 62} It is possible, therefore, that increases in muscle temperature may stimulate increases in the firing rates of the activated motor units. Similar phenomena have been postulated during the study of fatigue. For instance, the “muscle wisdom” hypothesis of Marsden et al\textsuperscript{64} suggested that as a muscle fatigues, the firing rates of the active motor units are reduced to match the relaxation rates of the twitch responses, which results in a more economical motor control strategy.\textsuperscript{65} One of the mechanisms proposed to explain the muscle wisdom hypothesis includes afferent feedback from peripheral receptors that may inhibit the discharge of interneurons or motor neurons at the level of the spinal cord.\textsuperscript{65} It is possible that a similar inverse model is employed during peripheral heating of a muscle, in that afferent feedback from the periphery may augment the discharge rates of interneurons or motor
neurons. Future research should examine the mechanisms underlying the temperature-related increases in the frequency domain of the MMG signal.

In a study using isometric ramp contractions, Akataki et al. reported increases in MMG center frequency from 5 – 80% MVC, despite decreases in MMG amplitude from 60 – 80% MVC. These findings were attributed to the shift in contributions to force production from motor unit recruitment to rate coding from 60 – 80% MVC. Coburn et al. demonstrated a non-significant increase in MMG center frequency with increasing isometric force production for the vastus medialis muscle, while Beck et al. reported significant increases in MMG center frequency for the biceps brachii muscle during submaximal to maximal isometric step contractions. In the present study, MMG IMF increased up to approximately 70-80% MVC, then plateaued or decreased to 90% MVC (Figure 4.11), which was consistent with the findings of Akataki et al. up to 80% MVC. It is possible that the frequency domain of the MMG signal may be useful as a noninvasive tool to estimate the global firing rate of active muscles during isometric ramp contractions. The differences among the findings of the present study and those of Beck et al. and Coburn et al. may be related to differences between isometric step and ramp contractions for elucidating the patterns of response for the surface MMG signal. Future studies should examine the differences between the response patterns of MMG signals during isometric step and ramp contractions.

Previous studies have indicated that EMG frequency parameters may be sensitive to changes in muscle temperature, but EMG amplitude remains unaltered. Krause and colleagues recently demonstrated that increases (via heat pack) and decreases (via
ice pack) in skin temperature resulted in concomitant increases and decreases, respectively, in the turns (i.e. frequency) of a unipolar surface EMG signal with no changes in EMG amplitude. An earlier study by Petrofsky and Lind\textsuperscript{22} reported a direct relationship between muscle temperature and EMG center frequency, but minimal effects of temperature on EMG amplitude at 30°C and above. In addition, Madigan and Pidcoe\textsuperscript{32} demonstrated a linear decrease in EMG center frequency with decreases in intramuscular temperature after a 20-min diathermy treatment. Merletti et al\textsuperscript{30} implied that temperature-related changes in the frequency parameters of the surface EMG may be related to the rate of metabolic byproduct accumulation. The results of the present study indicated sporadic increases in EMG IMF, particularly at 10-40% MVC for the diathermy group, from pre- to post-treatment that were independent of the treatment conditions (Figure 4.7). There were no changes in EMG IMF from pre- to post-treatment during the isometric MVCs (Figure 4.6). Furthermore, no changes in EMG amplitude occurred from pre- to post-treatment for any group during the isometric ramp contractions (Figure 4.5), however, EMG amplitude decreased from pre- to post-treatment during the isometric MVCs for each group (Figure 4.4). It is possible that the decreases in EMG amplitude observed for each group during the MVCs may have been related to the placement of the thermocouple. The pre-treatment MVCs were conducted prior to the placement of the intramuscular thermocouple, whereas the post-treatment MVCs were conducted with the thermocouple in place. Although MVC torque was unaltered (Figure 4.2), inhibitory afferent feedback from the indwelling thermocouple may have caused a decrease in central drive to the muscle, and consequently, cannot be ruled out as an
explanation for the decrease in EMG amplitude. Overall the sporadic changes in EMG amplitude and IMF during the isometric MVC and ramp contractions from pre- to post-treatment may underscore the recent debate\textsuperscript{66} regarding the limitations of surface EMG for examining neuromuscular patterns of response.

In the present study, EMG amplitude increased from 10 to 90\% MVC (Figure 4.5) similar to the increases in torque production (Figure 4.3) during the isometric ramp contractions. Many previous studies have observed similar increases in EMG amplitude with increases in force production during both isometric step\textsuperscript{21, 29, 63, 67} and ramp\textsuperscript{46} contractions. Traditional surface EMG amplitude provides a global indication of muscle activation, which can be affected by motor unit recruitment and the firing rates of the active motor units.\textsuperscript{20, 21, 66} Interestingly, the patterns of response for EMG amplitude during the isometric ramp contractions in the present study were unaltered by any of the treatment conditions (diathermy, sham-diathermy, and control) (Figure 4.5). This finding was consistent with Krause et al.\textsuperscript{31} and the authors concluded that, “…in studies, concerned with thermal effects on the muscle, only amplitude – and not frequency – seems to represent a valid parameter of muscle contraction” (p. 69). In contrast, however, EMG amplitude was not sensitive to the physiological temperature changes that were observed in the study by Krause et al\textsuperscript{31} or in the present study. Therefore, it is possible that based on the temperature-related increases in MMG amplitude observed in the present study without changes in the patterns of response, MMG amplitude may provide more information than EMG amplitude regarding both the potential temperature-
induced increases in muscle compliance as well as the motor unit recruitment strategies used to modulate force production.

Pulsed shortwave diathermy is regarded as a deep heating modality, which can affect muscle tissue. The physiological effects of heat include changes in extensibility of collagen tissue, changes in neuromuscular activity, changes in pain threshold, blood flow, capillary permeability, tissue metabolism, and enzymatic activity. However, the specific type of physiological effects obtained from the heat may be dependent on whether the degree of heat is vigorous or mild. Lehmann indicates that mild heating is associated with reducing muscle spasm and stiffness, but vigorous heating is associated with physiological responses such as increased extensibility of connective tissue, increased blood flow, changes in neuromuscular activity, and changes in pain threshold. To obtain vigorous responses with therapeutic heating modalities, Lehmann et al also suggested that it is necessary to 1) attain the highest temperature at the site of tissue pathology to be treated; 2) elevate the tissue temperature as nearly as possible to the maximally tolerated level; and 3) maintain the effective temperature elevation for an adequate period of time. Each of these three techniques may vary between individual subjects and specific tissue. Therefore, Lehmann does not give specific temperatures associated with mild, moderate, or vigorous heating effects. However, current therapeutic modality textbooks suggest that specific temperature increases are necessary to obtain particular effects. A 1°C increase can reduce mild inflammation and increase metabolism, a 2-3°C increase can decrease pain and muscle spasm, and a 3-4°C increase can increase tissue extensibility. Accordingly, our diathermy treatment to the vastus
lateralis would be qualified as mild to moderate heating because we observed a mean increase of $1.75 \pm 0.39^\circ C$. In addition, the temperature-related increases in MMG amplitude and IMF in the present study suggested that the diathermy-induced increases in intramuscular temperature were sufficient to elicit increases in muscle compliance (i.e., decreases in muscle stiffness). These findings are corroborated by Kimura et al.\textsuperscript{33} and Barry\textsuperscript{34} that have reported decreases in MMG amplitude under experimentally-induced hypothermic conditions, suggesting that the cold may have increased muscle stiffness. Therefore, our diathermy treatment parameters (27.12 MHz, 400 $\mu$s, 800 pps, and 40W) may be effective in creating a physiological decrease in muscle stiffness associated with the mild heating effects described by Lehmann.\textsuperscript{68}

This was the first study to explore the effects of diathermy on the mechanical properties of a muscle. Previous research has focused on the effect of diathermy on joint ROM, which includes muscle, ligament, joint capsule, and tendon properties. Two previous studies\textsuperscript{17, 18} examined the effects of a pulsed shortwave diathermy treatment (800 pps, 400 $\mu$s, and 48W) on the flexibility of the hamstring and ankle dorsiflexors in conjunction with low-load, long-duration stretching. The researchers reported greater increases in range of motion in the groups receiving both stretching and diathermy treatment. These increases were still present 72 hrs\textsuperscript{18} and 6 days\textsuperscript{17} after the stretching and heat treatments were discontinued. This retention of increased ROM, in addition to the fact that ROM increased more in the groups receiving the diathermy treatment and stretching than stretching alone, may suggest that the diathermy treatment provides greater increases in tissue extensibility because diathermy increases intramuscular
Furthermore, the unaltered MVC torque production and patterns of response for EMG amplitude, EMG IMF, MMG amplitude, and MMG IMF, despite the increases in muscle temperature, suggested that the diathermy modality may not adversely affect the neuromuscular motor control strategies used to modulate force production from 10 to 90% MVC. Therefore, with further research, it is possible that the diathermy modality can be used prior to performance events without diminishing the force producing capabilities or motor control strategies of the treated muscle.

Overall, the results of this study suggested that surface MMG may track the temperature-related increases in muscle tissue compliance and may be more sensitive to changes in the physiological conditions of the muscle than surface EMG while simultaneously tracking the intrinsic motor control strategies used to modulate force production. The observed changes in the mechanical properties of the muscle (surface MMG), in conjunction with the improvements in ROM from previous research lend support to the clinical application of diathermy on muscle tissue in a rehabilitation setting. Therefore, future research should include 1) exploring the level of heat increases necessary to obtain maximal changes in the mechanical properties; 2) combining ROM measurements with measures of the muscle’s mechanical properties through surface MMG; and 3) the surface MMG responses to temperature on injured vs. uninjured subjects.
APPENDIX A

STATEMENT OF INFORMED CONSENT
PHASE I

Statement of Informed Consent (Health Status Questionnaire)
IRB Approved Protocol 05.188

Title of Research Study
The thermal effects of pulsed shortwave diathermy on muscle force production, electromyography (EMG), and mechanomyography (MMG).

Invitation to Participate
I have been invited to participate in this research study. This study consists of two phases: Phase I invites me to fill out a Health Status Questionnaire and Phase II invites me to undergo the exercise testing. The following information is provided in order to help me make an informed decision whether or not to participate in either Phase I or II. If I have any questions, I will not hesitate to ask.

Basis for Subject Selection
I have been selected as a potential volunteer because I am between 19 and 35 years of age and in good health. If I wish to participate, I must sign and date two informed consent forms: Phases I and II. This informed consent form invites me to fill out our Health Status Questionnaire. Participation is completely voluntary and will not affect my standing in any class should I choose not to participate.

Assurance of Confidentiality
Any information obtained during this study, which could identify me will be kept strictly confidential as far as possible within state and federal law. The information may be published in scientific journals or presented at scientific meetings, but my identity will be kept strictly confidential. All data collected as a result of my participation will be kept by the investigators in a locked cabinet. My data will receive an identifying number and only the investigators will be able to identify me from my data. My data will be compiled and only group data will be used for dissemination without identifying my name. For the purpose of future reference, my data will be stored indefinitely.

Rights of Research Subjects
My rights as a research subject have been explained to me. If I have any additional questions concerning my rights as a research subject, I may contact the University of Texas at Arlington Institutional Review Board for the Protection of Human Subjects representative, telephone (817) 272-2105.

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Initials

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**Voluntary Participation and Withdrawal**

I am free to decide not to participate in this study or to withdraw at any time without adversely affecting my relationship with the investigators or the University of Texas at Arlington. My decision will not result in any loss of benefits to which I am otherwise entitled.

I AM VOLUNTARILY MAKING A DECISION WHETHER OR NOT TO PARTICIPATE IN THIS RESEARCH STUDY. MY SIGNATURE CERTIFIES THAT THE CONTENT AND MEANING OF THE INFORMATION ON THIS CONSENT FORM HAVE BEEN FULLY EXPLAINED TO ME AND THAT I HAVE DECIDED TO PARTICIPATE HAVING READ AND UNDERSTOOD THE INFORMATION PRESENTED. MY SIGNATURE ALSO CERTIFIES THAT I HAVE HAD ALL MY QUESTIONS ANSWERED TO MY SATISFACTION. IF I THINK OF ANY QUESTIONS DURING THIS STUDY I WILL CONTACT THE INVESTIGATORS. I WILL BE GIVEN A COPY OF THIS CONSENT FORM TO KEEP.

__________________________________________________________________________   ________________
Signature of Subject       Date

MY SIGNATURE AS WITNESS CERTIFIES THAT THE SUBJECT SIGNED THIS CONSENT FORM IN MY PRESENCE AS HIS/HER VOLUNTARY ACT AND DEED.

__________________________________________________________________________   ________________
Signature of Witness       Date

IN MY JUDGEMENT THE SUBJECT IS VOLUNTARILY AND KNOWINGLY GIVING INFORMED CONSENT AND POSSESSES THE LEGAL CAPACITY TO GIVE INFORMED CONSENT.

__________________________________________________________________________   ________________
Signature of Investigator      Date

Investigators:
Sarah M. Marek
work phone (817) 272-7017
mobile phone (254) 541-6352

A. Louise Fincher, Ed.D.
work phone (817) 272-3107
mobile phone (817) 846-9242

Cynthia Trowbridge, Ph.D.
work phone (817) 272-3134
mobile phone (817) 706-5121

Joel T. Cramer, Ph.D.
work phone (817) 272-5784
mobile phone (817) 798-8985
PHASE II

Statement of Informed Consent *(Health Status Questionnaire)*
IRB Approved Protocol 05.188

I have been asked to participate as a subject in the research project entitled The Thermal Effects of Pulsed Shortwave Diathermy on Muscle Force Production, Electromyography and Mechanomyography under the direction of Sarah Marek, A. Louise Fincher, Ed.D., Cynthia Trowbridge, Ph.D., and Joel Cramer, Ph.D.

PURPOSE OF THE STUDY

I understand that the purpose of this study is to examine the effects of heating on muscle function as measured by muscle strength, electromyography (EMG) and mechanomyography (MMG) during isometric ramp contractions.

PROCEDURES

Familiarization Trial: I will report to the lab, read and sign an informed consent, and fill out a health history questionnaire. Three skinfolds will be measured on both legs along a diagonal plane of the distal portion of the thigh, at the site of where the temperature measuring device (thermocouple) will be inserted in the experimental trial. Skin and subcutaneous fat thickness will be determined using the average of the three skinfolds. I will be included in the study if my skin and subcutaneous fat layer is ≤ 30 mm.

I will then be seated on the Biodex with restraining straps over the pelvis. I will perform two maximal voluntary contractions (MVC). MVC will be determined as the maximum force obtained during the 2 MVC trials and will be used as the target maximal force for the ramp contraction. I will then perform ramp contractions that consist of an isometric contraction at 60° of knee flexion for 3 sec at 5% MVC followed by a gradual increase in force production from 5% to at least 85% MVC over a 6 sec period. Ramp contractions will be performed until I can successfully complete a ramp contraction, with a maximum of 8-12 attempts. A successful ramp will meet the following criteria a) a maximal force reaching at least 85% MVC and b) a smooth linear increase in force through visual inspection. All measurements and treatments will be performed on the right leg. A 2 min rest will be given between each trial. After a minimum of 48 hours after the familiarization trial, an experimental trial will be scheduled. The duration of each experimental trial should not exceed two hours.

________________________
Initials
Experimental Trial: The experimental trial will consist of pre- and post-treatment testing. Prior to the trial, three skinfold measurements will be taken on my left thigh. My skin will be cleaned with alcohol swabs and rubbed lightly with emery paper (to remove any dead skin) at one location on my right thigh and one location on the front of my right hip bone. Electrodes will then be placed on the cleaned areas. Two people will be present during the placement of the electrodes. Wires from the electrodes will be hooked to a device, which measures the electrical activity of your muscles. In addition, one small muscle sensor will be placed on the surface of my thigh to measure the vibrations produced by the contracting muscles. I will then be seated in the Biodex to perform pre-treatment testing consisting of 2 MVC and 2 ramp contractions the same as the familiarization trial. If I do not meet the ramp criteria for at least one of the 2 trials, trials will be continued until a successful ramp is completed or a maximum of 5 ramps are performed. If I am unable to complete a successful ramp contraction, I will be dismissed from the study.

A 3 x 3 cm area surrounding the site for intramuscular thermocouple insertion will be cleansed with providine-iodine. The insertion site will be on the right thigh between the EMG and MMG sensors. The thermocouple will be inserted perpendicular to the skin surface with a needle and catheter, the needle will be removed, the implantable thermocouple will be threaded through the catheter tube into the quadriceps muscle (vastus lateralis), and the catheter will be removed. I will be required to sit for a period of time to obtain baseline temperature. Once baseline is reached, I will receive a treatment based on my group assignment.

Immediately following the treatment, the intramuscular thermocouple will be removed for post-treatment testing. I will perform 2 MVCs and 2 ramp contractions the same as the pre-treatment force measurements. If I do not meet the ramp criteria for at least one of the 2 trials, trials will be continued until a successful ramp is completed or a maximum of 5 ramps are performed. At the conclusion of post-treatment testing, the EMG and MMG electrodes will be removed, the skin will be cleaned with Proviodine-Iodine Prep Pads, an antibiotic ointment will be applied to the thermocouple and EMG electrode sites, and an adhesive bandage will be placed over the thermocouple insertion site. I will be given instructions on monitoring the area for potential infection and will be told to report to a local health care center immediately if any signs or symptoms appear.

NUMBER OF SUBJECTS PARTICIPATING

The number of subjects involved in the study is thirty-four.

RISKS OF PARTICIPATION

I understand that I may experience muscle soreness and temporary elevation of blood pressure during the isometric tests. I will be given instructions for stretches, which may aid in the elimination of any muscle soreness as a result of the tests. Throughout the tests, I will be monitored by laboratory personnel trained in CPR. In addition, I will be
asked repeatedly during the tests how I feel in relation to my ability to continue the test. There is also a possibility of infection and soreness from skin exfoliation for the electrode placement and at the site of the thermocouple insertion. Upon completion of all tests, an antibacterial ointment will be applied to the electrode and thermocouple sites and an adhesive bandage will be placed over the thermocouple insertion site to prevent any possible infection.

In the unlikely event that the I should suffer an injury as a direct consequence of the research procedures described above, the acute medical care required to treat the injury can be provided at the University of Texas at Arlington Health Services Center located at 605 S. West St. from the hours of 8:30 a.m.–5 p.m. Monday through Friday, and 10 a.m.–1 p.m. Saturday. The cost of such medical care will be my responsibility, whether at the Health Center or at other local health care facilities. If the health center is unable to treat me, emergency care is available at local community health providers.

BENEFITS TO THE SUBJECT

I understand that the direct benefits to me for participating will be learning about my force production capabilities of the quadricep muscles.

ALTERNATIVE TREATMENT

I understand there is no alternative procedure to be used in lieu of the experimental procedures; therefore I have the option of not participating.

STANDARD CLAUSES

1. I understand that informed consent is required of all persons in this project.

2. The principal and alternate procedures, including the experimental procedures in this project, have been identified and explained to me in language that I can understand.

3. The risks and discomforts from the procedures have been explained to me.

4. The expected benefits from the procedures have been explained to me.

5. An offer has been made to answer any questions that I may have about these procedures. If I have any questions before, during or after the study, I may contact:

   Ms. Sarah Marek at (817) 272-7017 (w) or (254) 541-6352 (c)
   Dr. A. Louise Fincher at (817) 272-3107 (w) or (817) 846-9242 (c)
   Dr. Cynthia Trowbridge at (817) 272-3134 (w) or (817) 706-5121 (c)
   Dr. Joel Cramer at (817) 272-5784 (w) or (817) 798-8985 (c)

_________________________ Initials __________________________

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6. I have been told that I may refuse to participate or stop my participation in this project at any time. All new findings during the course of this research which may influence my desire to continue or not to continue to participate in this study will be provided to me as such information becomes available.

7. If I am injured or have an adverse reaction because of this research, I should immediately contact one of the personnel listed in Clause #5 above. No additional compensation will be provided. Agreeing to this does not mean I am giving up any legal rights that I may have.

8. If I have any questions regarding my rights as a subject participating in this study or research-related injury, I may contact Office of Research Compliance at (817) 272-3723.

9. I have a right to privacy, and all information that is obtained in connection with this study and that can be identified with me will remain confidential as far as possible within state and federal law. However, information gained from this study that can be identified with me may be released to no one other than the investigators. The results of this study may be published in scientific journals without identifying me by name.

I voluntarily agree to participate as a subject in the above named project. I understand that I will be given a copy of the consent form I have signed.

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<thead>
<tr>
<th>Subject Name</th>
<th>Signature of Subject</th>
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Using language that is understandable and appropriate, I have discussed this project and the items listed above with the subject and/or his/her authorized representatives.

<table>
<thead>
<tr>
<th>Principal Investigator Name</th>
<th>Signature of Principal Investigator</th>
<th>Date</th>
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</table>

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<tr>
<th>Witness Name</th>
<th>Signature of Witness</th>
<th>Date</th>
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APPENDIX B

PRE-EXERCISE TESTING HEALTH STATUS QUESTIONNAIRE
Pre-Exercise Testing Health Status Questionnaire

To:  
From:  
Date:  

Name ____________________________________________ Date __________________

Home Address __________________________________________________________

Work Phone _______________________ Home Phone ______________________

Person to contact in case of emergency ______________________________________

Emergency Contact Phone ____________________ Birthday (mm/dd/yy)____/_____/_____

Personal Physician ________________________ Physician’s Phone ______________________

Gender ________ Age ______(yrs)   Height ______(ft)______(in)  Weight______(lbs)

Does the above weight indicate:  a gain____ a loss____ no change____ in the past year?
If a change, how many pounds?___________(lbs)

A. JOINT-MUSCLE STATUS (✓Check areas where you have problems or meet criteria)

1. Do you CURRENTLY have problems with:

Joint Areas

( ) Wrists   ( ) Arms
( ) Elbows   ( ) Shoulders
( ) Shoulders ( ) Chest
( ) Upper Spine & Neck ( ) Upper Back & Neck
( ) Lower Spine ( ) Abdominal Regions
( ) Hips ( ) Lower Back
( ) Knees ( ) Buttocks
( ) Ankles ( ) Thighs
( ) Feet ( ) Lower Leg
( ) Other ____________________________  ( ) Feet

( ) Other___________________________
2. Have you had an injury within the PAST 12 MONTHS to:
   ( ) Knee
   ( ) Thigh
   ( ) Lower Leg
   If any checked, please explain: ____________________________________________

B. HEALTH STATUS (✓ Check if you currently have any of the following conditions)

( ) High Blood Pressure       ( ) Acute Infection
( ) Heart Disease or Dysfunction ( ) Diabetes or Blood Sugar Level Abnormality
( ) Peripheral Circulatory Disorder ( ) Anemia
( ) Lung Disease or Dysfunction       ( ) Hernias
( ) Arthritis or Gout             ( ) Thyroid Dysfunction
( ) Edema                           ( ) Pancreas Dysfunction
( ) Epilepsy                        ( ) Liver Dysfunction
( ) Multiple Sclerosis            ( ) Kidney Dysfunction
( ) High Blood Cholesterol or Triglyceride Levels ( ) Phenylketonuria (PKU)
( ) Loss of Consciousness          ( ) Allergic Reactions to Medication
( ) Hypersensitivity to Needles ( ) Allergic Reactions to Any Other Substance
( ) Others That You Feel We Should Know About __________________________

C. PHYSICAL EXAMINATION HISTORY

Approximate date of your last physical examination __________________________

Physical problems noted at that time _______________________________________

Has a physician ever made any recommendations relative to limiting your level of physical exertion? _______YES _______NO
If YES, what limitations were recommended? __________________________________

D. CURRENT MEDICATION USAGE (List the drug name and the condition being managed)

MEDICATION                          CONDITION
_________________________________ __________________________
_________________________________ __________________________
_________________________________ __________________________
### E. PHYSICAL PERCEPTIONS

(Indicate any unusual sensations or perceptions. 
✓Check if you have recently experienced any of the following during or soon after physical activity (PA); or during sedentary periods (SED))

<table>
<thead>
<tr>
<th>PA</th>
<th>SED</th>
</tr>
</thead>
<tbody>
<tr>
<td>( )</td>
<td>( ) Chest Pain</td>
</tr>
<tr>
<td>( )</td>
<td>( ) Heart Palpitations</td>
</tr>
<tr>
<td>( )</td>
<td>( ) Unusually Rapid Breathing</td>
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<tr>
<td>( )</td>
<td>( ) Overheating</td>
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<tr>
<td>( )</td>
<td>( ) Muscle Cramping</td>
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<tr>
<td>( )</td>
<td>( ) Muscle Pain</td>
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<tr>
<td>( )</td>
<td>( ) Joint Pain</td>
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<td>( )</td>
<td>( ) Other _____________________</td>
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<table>
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<tr>
<th>PA</th>
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<tr>
<td>( )</td>
<td>( ) Nausea</td>
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<td>( )</td>
<td>( ) Light Headedness</td>
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<td>( )</td>
<td>( ) Loss of Consciousness</td>
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<td>( )</td>
<td>( ) Loss of Balance</td>
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<td>( )</td>
<td>( ) Loss of Coordination</td>
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<td>( )</td>
<td>( ) Extreme Weakness</td>
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<tr>
<td>( )</td>
<td>( ) Numbness</td>
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<tr>
<td>( )</td>
<td>( ) Mental Confusion</td>
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### F. FAMILY HISTORY

✓Check if any of your blood relatives . . . parents, brothers, sisters, aunts, uncles, and/or grandparents . . . have or had any of the following)

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<tbody>
<tr>
<td>( ) Heart Disease</td>
<td></td>
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<tr>
<td>( ) Heart Attacks or Strokes (prior to age 50)</td>
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<tr>
<td>( ) Elevated Blood Cholesterol or Triglyceride Levels</td>
<td></td>
</tr>
<tr>
<td>( ) High Blood Pressure</td>
<td></td>
</tr>
<tr>
<td>( ) Diabetes</td>
<td></td>
</tr>
<tr>
<td>( ) Sudden Death (other than accidental)</td>
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### G. CURRENT HABITS

✓Check any of the following if they are characteristic of you current habits)

<p>| | |</p>
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<tbody>
<tr>
<td>( ) Regularly does manual garden or yard work</td>
<td></td>
</tr>
<tr>
<td>( ) Regularly goes for long walks</td>
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<tr>
<td>( ) Frequently rides a bicycle</td>
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<tr>
<td>( ) Frequently runs/jogs for exercise</td>
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</tr>
<tr>
<td>( ) Regularly participates in a weight training exercise program</td>
<td></td>
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<tr>
<td>( ) Engages in a sports program more than once per week. If so, what does the program consist of?</td>
<td></td>
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</tbody>
</table>

If anything above is checked, please estimate the total number of hours per week of regular exercise you engage in: _____________ hours per week

### H. IMPLANTABLE OBJECTS

✓Check if you currently have any of the following implants)

Do you have metal implants (plates or screws) in your:

- ( ) Knee
- ( ) Thigh
- ( ) Lower Leg

Do you have a cardiac pacemaker? ( ) YES ( ) NO

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REFERENCES


25. Orizio C, Gobbo M, Diemont B, Esposito F, Veicsteinas A. The surface mechanomyogram as a tool to describe the influence of fatigue on biceps brachii


BIOGRAPHICAL INFORMATION

Sarah Marie Marek was born on December 5, 1981 in Temple, TX, and in May 2000, she graduated from C.H. Yoe High School in Cameron, TX. Sarah received a Bachelor of Science degree in Kinesiology, with an Athletic Training option and Mathematics minor from Angelo State University in San Angelo, TX in August 2003. She accepted a position as a graduate teaching assistant in the Department of Kinesiology at The University of Texas at Arlington from 2003-2005, where she received a Master of Science degree in Physiology of Exercise in May 2005. She is a Certified Athletic Trainer by the National Athletic Trainers’ Association Board of Certification, and a Licensed Athletic Trainer by the Texas Advisory Board of Athletic Trainers. She plans to work in a university setting as an athletic trainer and an instructor for the athletic training academic courses. Sarah anticipates pursuing a doctorate degree at some point in the future.