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Effect of melt method on thoracolumbar connective tissue

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ABSTRACT

EFFECT OF MELT METHOD ON THORACOLUMBAR CONNECTIVE TISSUE

by
Faria Sanjana

Approximately, 31 million adults in the United States suffer from low back pain (LBP). Altered connective tissue structure has been identified in adults with chronic LBP. Numerous novel treatments are being developed targeting the connective tissue and nervous system to relieve people from LBP. One such self-care treatment is the MELT method. The objective of this study was to determine whether thickness of thoracolumbar connective tissue and other biomechanical and viscoelastic properties of myofascial tissue in the low back region change in subjects with chronic LBP as a result of MELT.

Using ultrasound imaging and an algorithm developed in MATLAB, thickness of thoracolumbar connective tissue was analyzed. A hand-held digital palpation device, called the MyotonPRO, was used to assess biomechanical properties such as stiffness, elasticity, tone and mechanical stress relaxation time of the thoracolumbar myofascial tissue. A forward bending test assessing flexibility and pain scale was added to see if MELT affected subjects with chronic LBP.

A significant decrease in connective tissue thickness and a significant increase in mechanical stress relaxation time in a particular area of the low back was observed in treatment group participants. Significant increase in flexibility and significant decrease in pain was also recorded.

**EFFECT OF MELT METHOD ON THORACOLUMBAR CONNECTIVE
TISSUE**

**by
Faria Sanjana**

**A Thesis
Submitted to the Faculty of
New Jersey Institute of Technology
in Partial Fulfillment of the Requirements for the Degree of
Master of Science in Biomedical Engineering
Department of Biomedical Engineering**

May 2016

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APPROVAL PAGE

**EFFECT OF MELT METHOD ON THORACOLUMBAR CONNECTIVE
TISSUE**

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For my mother who has been a constant source of motivation and inspiration.

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CHAPTER 1

INTRODUCTION

1.1 Background Information

Low back pain (LBP) is the leading cause of work-related disabilities and increase in health costs around the world (De Luca, 1997; Williams et al., 1998). It has been estimated that approximately 31 million adults in the U.S. suffer from LBP (Jensen et al., 1994). Chronic LBP is defined as pain that lasts for three or more months (Wheeler and Berman, 2014). In previous years, chronic LBP was understood from the perspectives of vertebral structural pathologies, neuropsychosocial factors and motor control abnormalities (Langevin et al., 2009). It is not until recently that researchers have associated chronic LBP with thoracolumbar connective tissue (Langevin and Sherman, 2007; Langevin et al., 2009; Langevin et al., 2011; Schilder et al., 2014).

An abnormal thoracolumbar connective tissue structure with increased thickness and disorganization of connective tissue layers have been reported in chronic and recurrent LBP subjects by Langevin et al. (Langevin et al., 2009). Langevin and Sherman have also suggested that alternative treatments such as massage, chiropractic manipulation, movement therapies and acupuncture needle manipulation may be effective in connective tissue remodeling that could reduce LBP (Langevin and Sherman, 2007). These therapies work by changing the biomechanical properties of affected connective tissue such as stiffness, viscoelasticity and density (Smith, 2005).

Myofascial release (MFR) is another such alternative treatment that is said to assist in restoring connective tissue structure. This technique causes a stretch in affected

connective tissue after application of pressure on the tissue barrier for about 120 seconds (Barnes, 1997). As a result, the tissue exhibits histological length changes that are felt as a release. This release is followed up into other affected tissue barriers (Barnes, 1997). After a few releases, the tissue is said to become softer with restored mobility (Barnes, 1997). A similar treatment, fascial unwinding, uses the mechanism of touch and stretch on to connective tissue to relax the tissue and activate the parasympathetic nervous system (Minasny, 2009). The central nervous system is also activated that induces a motor action for the affected muscle to find an easier and more relaxed position (Minasny, 2009). These manual fascial techniques (MFTs) served as basis for the creation of the MELT method.

MELT (previously known as Myofascial Energetic Length Technique) is a hands-off, self-treatment that aims to release tension, decrease pain and restore mobility for LBP patients. Specialized soft treatment balls, soft foam roller and self-care techniques that mimic manual therapy are used to reduce chronic pain by rehydrating connective tissue and rebalancing the regulators of the nervous system (Hitzmann, 2013). Like other MFTs, MELT is proposed to release collagen fibers that causes their reorganization in the underlying substance, whose viscosity changes permit tissue remodeling (Cantu and Grodin, 2001). This change in viscosity allows an increase in hyaluronic acid production, along with flow and drainage of inflammatory mediators and metabolic wastes (Schultz et al., 1996). Till date, no scientific research study has examined whether MELT reduces chronic pain by the aforementioned mechanisms or if there is connective tissue remodeling as a result of treatment.

A method of visualizing whether MELT and other treatments are effective in changing the structure of connective tissue would be done by using an ultrasound. Ultrasound is a non-invasive device that allows us to see anatomical structures from ultrasonic waves that are bounced off from tissue interfaces (Langevin et al., 2009). Multiple studies have shown that ultrasound can be used to assess connective tissue structure in a quantitative manner. For example, Langevin and Yandow performed a B-scan visualization of anatomical details of connective tissue while inserting an acupuncture needle in human arm (Langevin and Yandow, 2002). Langevin et al. also undertook measurements of connective tissue thickness by converting ultrasound raw radio frequency data echoes into B-scan images (Langevin et al., 2009). Moreover, Ophir et al. made use of another technology, elastography, which utilized echo reflections to measure the mechanical response or mechanical property of tissues under stress (Ophir et al., 1999).

Mechanical properties of connective tissue have been reported in a few studies (Langevin et al., 2011). Chaudhry et al. devised a three-dimensional mathematical model that identified the relationship between mechanical forces and deformation of connective tissue under manual therapy (Chaudhry et al., 2008). However, deformation is difficult to evaluate without the use of elastography. An alternative to elastography is the use of a hand-held myometer that measures state of tension, biomechanical and viscoelastic properties of myofascial tissue.

Myometric devices have become popular in research studies because of its non-invasiveness, portability and easy to use qualities. Several studies have shown the reliability of the myometer for biomechanical and viscoelastic properties (Zinder and

Padua, 2011; Chuang et al., 2012; Bailey et al., 2013; Lam et al., 2015). Lam et al. performed stiffness, elasticity and state of tension measurements of 11 muscle sites. They used intra-class correlation coefficient to observe reliability of measurements within the same day and between two consecutive days and found that the within-day measurements were more reliable than inter-day measurements (Lam et al., 2015). Their results also showed asymmetry between left and right sides for elasticity measurements of two classes of muscle, suggesting that this could increase risk of injury (Lam et al., 2015). Zinder and Padua measured myometric stiffness of rectus femoral muscles after subjects were exposed to maximum voluntary isometric contraction of 10%, 20%, 30%, 40% and 50% (Zinder and Padua, 2011). They found that stiffness measurements were consistent with previous literature and were reliable with the use of intra-class correlation coefficients (Zinder and Padua, 2011). Pruyn et al. reported that higher stiffness in the lower body improved physical performance such as jumping, bounding and sprinting, in female athletes (Pruyn et al., 2014). Hence, a myometer along with B-mode ultrasound imaging was used in this study for observing structural change and biomechanical change in myofascial tissue.

1.1.1 Objective

Langevin et al. reported that chronic and recurrent LBP human subjects had a 25% higher than average connective tissue thickness (Langevin et al., 2009). This report was used to find out if the increased connective tissue of LBP subjects would decrease as a result of MELT. The reduction in thickness was hypothesized to occur due to increased fluid (hyaluronic acid) produced in tissues, suggesting rehydration or due to increased stretching caused by elongation of connective tissue in the lateral direction. Furthermore,

if connective tissue remodeling occurred as a result of MELT, then the biomechanical and viscoelastic properties of the tissue would change. Therefore, investigation of a decrease in stiffness and state of tension and increase in elasticity and mechanical stress relaxation time of myofascial tissue, due to MELT was conducted. This change would typically be shown by taking connective tissue thickness and myofascial properties' measurements before and immediately after MELT treatment and after a long-term, 4 week of MELT treatment.

1.1.2 Limitations

The thickness of connective tissue was measured by a program developed in MATLAB (details in "Methods" section). There is uncertainty relating to the accuracy of thickness measurements by this program despite the fact that it converted raw radio frequency data from ultrasound. The investigator had to manually place the cursor of the computer mouse on the areas prompted by the program to obtain connective tissue thickness.

When testing participants after 4 weeks, exact locations that were used for the initial myometer measurements could not be correctly identified. The locations were estimated by hand palpation. In order to produce consistent myometer results, it is important that exact locations be determined. Furthermore, myotonometric measurements are yet incapable to differentiate connective tissue from muscle, fat and skin layers. Therefore, instead of using the term connective tissue stiffness, the term myofascial tissue stiffness is used throughout this thesis.

Finally, the sample size of subjects to show significant myometer changes were too small compared to a sample size calculation that suggested participation of 120 subjects.

1.1.3 Outline

The rest of Chapter 1 reviews connective tissue i.e. fascia and thoracolumbar fascia (TLF) in detail. Chapter 2 discusses methods used for subject recruitment, data acquisition and testing procedures. Chapter 3 presents the results of the study, Chapter 4 discusses the results and Chapter 5 concludes the study.

1.2 Definition and Structure of Fascia

At the First International Fascia Research Congress in 2007, fascia experts defined fascia as “the soft tissue component of the connective tissue system that permeates the human body, forming a continuous, whole-body, three-dimensional matrix of structural support; interpenetrating and surrounding all organs, muscles, bones, and nerve fibers, creating a unique environment for body systems functioning” (Findley and Shalwala, 2013). This definition was advantageous because it meant that fascia “extends to all fibrous connective tissues, including aponeuroses, ligaments, tendons, retinaculae, joint capsules, organ and vessel tunics, the epineurium, the meninges, the periosteal, and all the endomysial and intermuscular fibers of the myofasciae” (Findley and Shalwala, 2013). It also meant that fascia is a single interconnected tensional network that can adjust its fiber arrangement, density and length according to the tensional demands surrounding it (Findley, 2009). The definition was also true to the Latin origin of fascia that means bundle, band or bandage (Findley, 2009).

By the Second International Fascia Research Congress in 2009, the simple definition of fascia was modified by Langevin and Huijing (Langevin and Huijing, 2009). These researchers suggested that fascia should not be defined as a single term by itself because of the uncertainty that the term refers to an anatomic entity or a type of tissue. Further structures or layers should be added to define fascia in order to decrease ambiguity surrounding this term (Langevin and Huijing, 2009). Langevin and Huijing identified twelve aspects of fascia that include dense connective tissue, areolar (non-dense) connective tissue, deep fascia, superficial fascia, intermuscular septa, interosseal membrane, periost, neurovascular tract, epimysium, endomysium, perimysium and intra- and extramuscular aponeurosis (Langevin and Huijing, 2009).

Dense connective tissue consists of closely packed collagen fibers and elastic fibers. According to functional requirements, the dense connective tissue can be irregularly or regularly packed (Langevin and Huijing, 2009). The collagen fibers are arranged in an unwoven mesh in order to resist stretch from all directions (Langevin and Huijing, 2009). This is called the dense irregular connective tissue. When the fibers are arranged in a parallel manner when loading is applied on one side, it is called the dense regular connective tissue (Langevin and Huijing, 2009). Tendons, ligaments, aponeuroses, intermuscular septa are typical dense regular connective tissue (Langevin and Huijing, 2009). Fibroblasts are embedded between and parallel to the collagen fibers of dense irregular and regular connective tissue (Patel, 2014).

Areolar or non-dense connective tissues, on the contrary, are collagen and elastic fibers that are sparingly arranged. It has the misnomer of being called loose connective tissue. Langevin and Huijing pointed out that the term “loose connective tissue” has more

of a morphological meaning than a mechanical one since this type of connective tissue can withstand shearing and bear loads associated with muscle force (Langevin and Huijing, 2009). Neurovascular bundles and sensory nerve branches are contained within areolar connective tissue that end in the dense areolar connective tissue layers (Stecco et al., 2007). However, the orientation of these bundles and branches are not well studied. Since areolar connective tissue allows shear deformation between two adjacent connective tissue layers, it has the ability to demonstrate a gliding phenomenon that has been observed by ultrasound imaging (Fox et al., 2009).

Superficial fascia is made up of areolar connective tissue and fat (Figure 1.1). The superficial fascia layer begins underneath the skin and joins the underlying bones or deep fascia, varying on different species (Abu-Hijleh et al., 2006). The fat layer of superficial fascia is enclosed within the retinacula cutis superficialis, which is orthogonally placed to the surface while connecting with the dermis (Patel, 2014). Connecting on the other side with deep fascia is the retinacula cutis profunda, which is arranged in an obliquely-horizontal manner (Patel, 2014).

The areolar connective tissue layer of superficial fascia consists of fibroelastic tissue that contains elastin fibers in an organized manner, in abundance (Patel, 2014). It is made up from single to multiple interconnecting sub-layers, depending on different organ locations. The gliding phenomenon mentioned earlier is caused in the subcutaneous tissue by the superficial and deep fat layers making the skin above it independently mobile

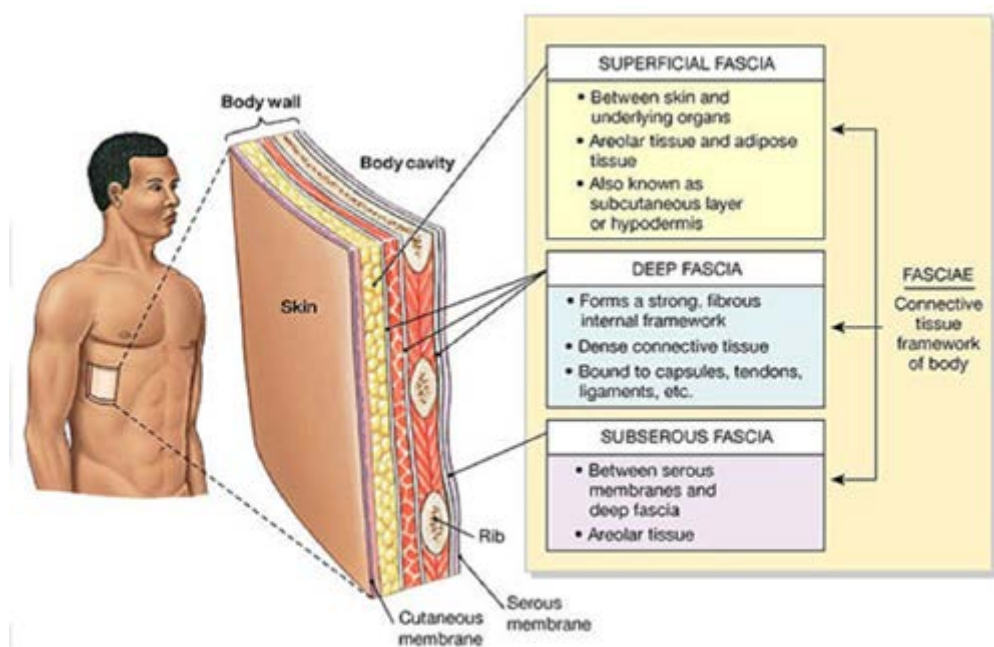


Figure 1.1 Diagram showing superficial, deep and subserous fascia.

Source: Dalton E: Can Bodywork Lubricate Joints and Fascia? *Dalton Myoskeletal*. Web. 16 November 2015.

Deep fascia is the layer directly underneath the superficial fascia (Figure 1.1). It is made up of a sheet of dense irregular connective tissue. The ability of dense irregular connective tissue to resist stretch causes the deep fascia to form a strong, fibrous internal framework. This framework provides the tensile strength to maintain structural form of certain body parts in addition to retaining their exterior outline (Clark, 1949). Deep fascia can resist stretching from underlying muscles within certain limits, after which stretching causes nerve pathway disruptions (Stecco et al., 2006). Such nerve terminations cause the loss of sliding motion of collagen fibers due to trauma, surgery or excessive use (Stecco et al., 2006). As a result, incorrect stimulation of nerve receptors that cause pain, occur even during normal stretching (Stecco et al., 2006).

Deep fascia extends into the muscles lying beneath it. In such cases, the term “investing” can be used to describe deep fascia in some parts of the body like the neck because it extends into the periosteum of bone, epimysium of skeletal muscles, and peritoneum of tendons and ligaments (Langevin and Huijing, 2009; Patel, 2014). The close association of deep fascia and its underlying muscles forms aponeuroses between them (Langevin and Huijing, 2009). The fascia in this region is, therefore, thicker and denser.

According to medical terminology, aponeurosis is a tendinous expansion that forms connections or attachments with connective tissue and muscle. Both extra- and intramuscular parts are contained within an aponeurosis (Langevin and Huijing, 2009). The intramuscular parts are connected to muscle fibers and hence, form an aponeurosis to tendon and to bone (Langevin and Huijing, 2009). Aponeuroses that are made up of partial fascial sheets can be restructured into a tendon closer to the bone by achieving continuous support by hanging from ligaments or crossing over a joint (Langevin and Huijing, 2009). Definitions of the other eight structures mentioned above that are associated with the term fascia according to Langevin and Huijing, can be found in Table 1 of their paper (Langevin and Huijing, 2009).

1.3 Functions of Fascia

In addition to acting as a sheet to support underlying structure, fascia has many musculoskeletal functions. These include its function as ectoskeleton primarily for muscle attachment, allowing return of venous components from lower limbs, providing osteofascial compartments for muscle and dispersing stress in connective tissue between tendon or ligament and bone (Benjamin, 2009).

The role of fascia as a protective sheet helps to shield different structures of the body. In hand palms and foot soles, palmar aponeurosis and plantar aponeurosis guard nerves and vessels that extend deep into them between their proximal and distal parts (Benjamin, 2009). Athwal et al. showed that bicipital aponeurosis, tendinous expansion from the biceps brachii, assists in protecting the underlying structures (Athwal et al., 2007). Force transmission and tendon stabilization is another protective function of the bicipital aponeurosis (Eames et al., 2007). Fascia acts as a mediator between skin and tendons during movement in order to control displacement. However, the inability of dense connective tissue to tackle with compression, hinders it for functions like walking or gripping, for which fat is better suited as it is incompressible (Bojsen-Moller and Flagstad, 1976).

As known by now, fascia connects to tendons, ligaments and bones in addition to muscle. This ability to connect makes the fascia an ectoskeleton. Fascial function as ectoskeleton can be seen in human lower limb where tensor fascia latae and gluteus maximus attach to deep fascia instead of bone, contributing to the upright posture of humans (Jones, 1944). Another function of fascia as ectoskeleton is observed in the attachment of fascia to the tibialis anterior in the upper leg causing the fascia fibers to orient in a longitudinal direction (Benjamin, 2009). On the contrary, absence of ectoskeleton function between deep fascia and muscle can be useful in muscles like the gastrocnemius. This allows the gastrocnemius muscle to work independently and exhibit its capability to bear weight in the face of strong contractions (Grant, 1940).

Deep fascia forms compartments along with intermuscular septa and associated bones, which are called osteofascial compartments. Different sections of the limb have

separate compartments in accordance with their positions or actions performed by their muscles. From the deep fascia, powerful intermuscular septa extend to merge with the periosteum of the tibia and fibula that creates separate compartments for the dorsiflexor, peroneal and plantarflexor muscles (Benjamin, 2009). The dorsiflexor muscles are contained in the anterior compartment and are studied due to their relevance to compartment syndrome. Compartment syndrome occurs due to buildup of pressure in the limited space within the deep fascia (Benjamin, 2009). Abrupt trauma or muscle overuse resulting in disrupted blood flow may be the cause of the pressure build up (Benjamin, 2009). In such cases, removal of the deep fascia may be required to decrease the pressure caused by the blockage (Benjamin, 2009). Example locations where compartment syndrome can occur are in the fascial compartments of hands and feet which are relatively small compared to other fascial compartments (Grant, 1940).

The function of fascia to allow venous return from lower limbs is achieved when the muscles contract against the thick, resistant fascia, causing the thin-walled veins and lymphatics in the muscles to be squeezed so that blood and lymphatic fluid are pumped towards the heart. There is an abundance of deep fascia in the lower limb because of the long distance from the heart to the leg and foot for the purpose of venous return (Jones, 1944). The two sets of veins in the lower limb that are squeezed by the muscle-pumping action are linked by perforated veins, which penetrate the deep fascia (Meissner et al., 2007). These vessels contain valves that allow unidirectional flow of blood and lymphatic fluid; these valves also distribute the hydrostatic pressure build up from blood (Meissner et al., 2007). If the valves do not function properly in the leg, then the muscle pumping

function fails. Meissner et al. points that the calf muscle pump function is the most important due to its large capacitance (Meissner et al., 2007).

1.4 Thoracolumbar Fascia

The thoracolumbar fascia (TLF) is defined as the deep fascia of the back. It spans across the thoracic and lumbar regions of the trunk and is covered by the erector spinae complex (Benjamin, 2009). It forms a thin layer over the extensor muscles in the thoracic region, attaching laterally to the ribs and medially to the spine (Benjamin, 2009). It forms an aponeurosis with abdominal muscles in the lumbar region laterally and attaches to the vertebral spine (Benjamin, 2009). It is divided into anterior, middle and posterior layers medially (Figure 1.2) (Benjamin, 2009). The anterior layer is anterior to the quadratus lumborum, joining the middle layer that passes between the quadratus lumborum and the paraspinal muscles (Patel, 2014). The posterior layer (pTLF) forms an envelope over the erector spinae and the multifidus (Benjamin, 2009).

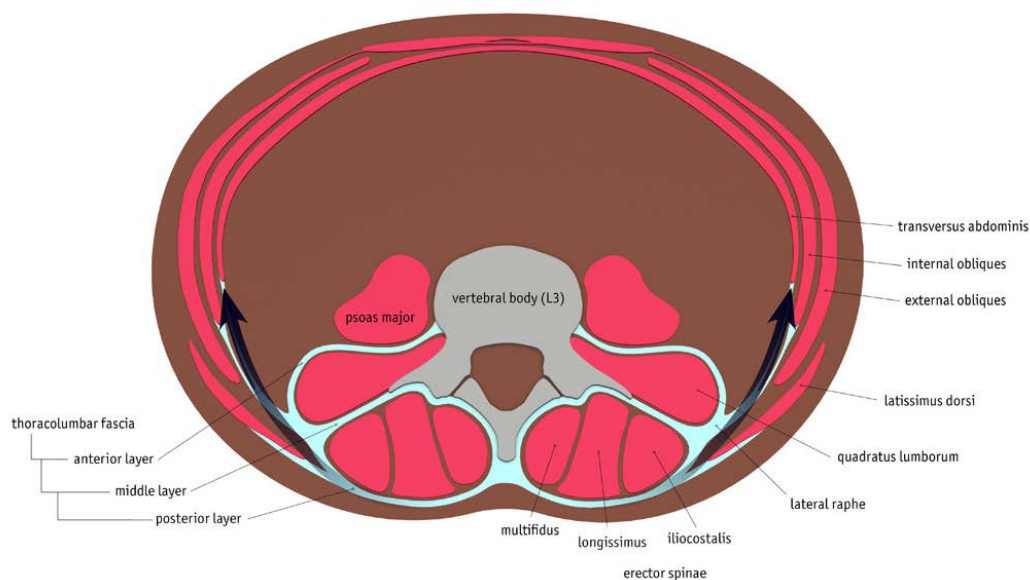


Figure 1.2 Cross-section of the thoracolumbar fascia with its three layers and associated muscles.

Source: Lengthening the torso in forward bends. *The Daily Bandha*. Web. 18 November 2015.

All three layers connect laterally to the lateral raphe (Figure 1.2). The TLF also attaches to the iliac crest, iliolumbar ligament and sacroiliac joint (Benjamin, 2009). Through its attachment to the vertebral spine, TLF joins the supraspinous and interspinous ligaments (Benjamin, 2009).

The layer under investigation via ultrasound in this study is the pTLF. The pTLF is said to be a key component in transferring forces between the spine, pelvis and legs (Vleeming and Stoeckart, 2007). Vleeming et al. reported that the pTLF joins two of the largest muscles of the body, the latissimus dorsi and the gluteus maximus, in a manner that promotes coordinated action of the upper and lower limbs such as the swiveling pendulum-like motion of the arm and walking and running by the legs (Vleeming et al., 1995).

The pTLF can also be subdivided into superficial and deep lamina (Willard et al., 2012). The superficial lamina of the pTLF is made up of aponeuroses with the latissimus dorsi and the serratus posterior inferior (Willard et al., 2012). The aponeuroses of the serratus posterior inferior surpass the twelfth rib, where the muscle ends and extends into the upper lumbar region to form a second layer of the superficial lamina (Benetazzo et al., 2011). The deep lamina of the pTLF forms an enveloping sheath around the paraspinal muscles (Willard et al., 2012). This sheath, known as the paraspinal retinacular sheath, performs as a hydraulic amplifier in order for the paraspinal muscles to support the lumbosacral spine (Willard et al., 2012). The deep lamina passes from the sacrum into the splenius capitis to fuse with the cranial base of the cervical fascia (Willard et al., 2012). The deep lamina joins over the iliac crest along with the aponeurosis of the gluteus medius laterally and inferiorly. Medially and inferiorly, the deep and superficial laminae join together at the PSIS (Willard et al., 2012).

CHAPTER 2

METHODS

This chapter outlines the methods used for subject recruitment, ultrasound imaging, MyotonPRO, flexibility test and pain scale recording.

2.1 Human Subject Recruitment and Recruitment Criteria

The study was approved by New Jersey Institute of Technology Institutional Review Board (HHS FWA 00003246). A total of 61 participants signed consent forms for this study. However, data from 44 participants for both MELT and control group could be used causing a drop in about 28% participant rate for the study. This is because a number of participants did not return for the 4-week retesting day.

The recruitment of 44 subjects, aged 25-65 with non-specific chronic LBP, (22 for MELT treatment group and 22 for control group) occurred via online, phone and in-person advertisements in doctor's offices, pain clinics and associated locations in the New York City area. All subjects provided informed consent. Inclusion criteria consisted of subjects having chronic pain for at least 12 months and pain index of 2 (out of 10 on a Visual Analogue Scale). Exclusion criteria of subjects were: BMI over 28.5, major structural spinal deformity, severe back or low extremity injury or surgery, ankylosing spondylitis or rheumatoid arthritis, neurological disorders, intake of spinal corticosteroid injections, pregnancy and less than 8 months postpartum.

2.2 Ultrasound Imaging

The investigator was blind to the study condition (MELT vs control). Images were taken with Terason T3000 (Terason, Burlington, MA) (Figure 2.1).

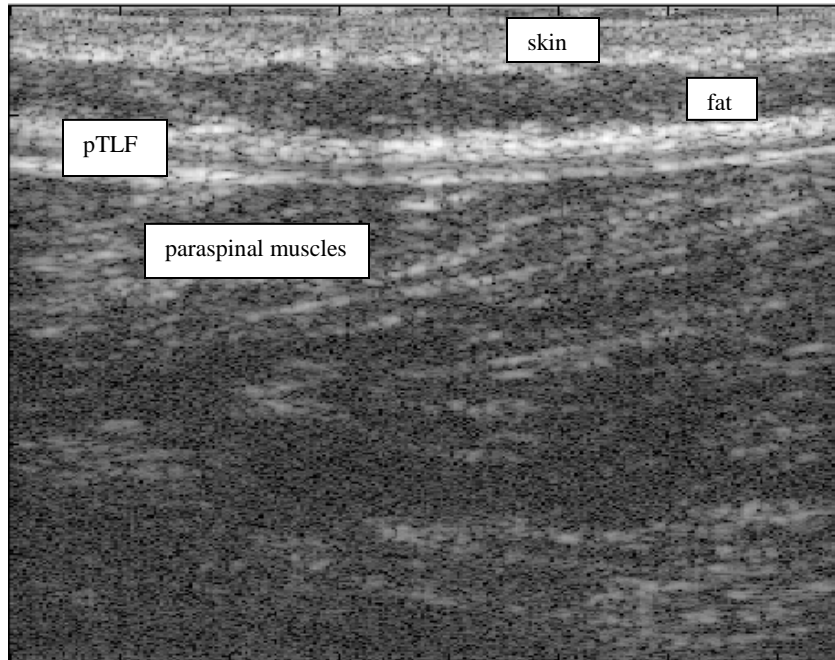


Figure 2.1 Ultrasound image of low back region in the specified transducer location of 2 cm lateral to the spinous process of L2-3. The skin, fat, pTLF and paraspinal muscles are highlighted.

Baseline measures were taken from the subjects' back, at a location where the ultrasound transducer was centered at 2 cm lateral to the middle of L2-3 interspinous ligament on left and right sides, while they lay on a prone position (Figure 2.2). This location was selected according to the prior study of Langevin et al. (Langevin et al., 2009) in which they found that at the L2-3 level, the fascia planes were most parallel to the skin. The testing location was identified in the following steps: 1) Locate pelvic crest on two sides 2) Identify depression on the midpoint of the spine at the level of the pelvic crest, which is the L3-4 interspace 3) Go one level up and locate the depression, which is the L2-3

interspace 4) Validate this location with live ultrasound imaging. 5) Measure 2 cm to the left and right of the depression and mark the midpoint with a surgical pen. Minimal compression to the tissue was applied during image acquisition.

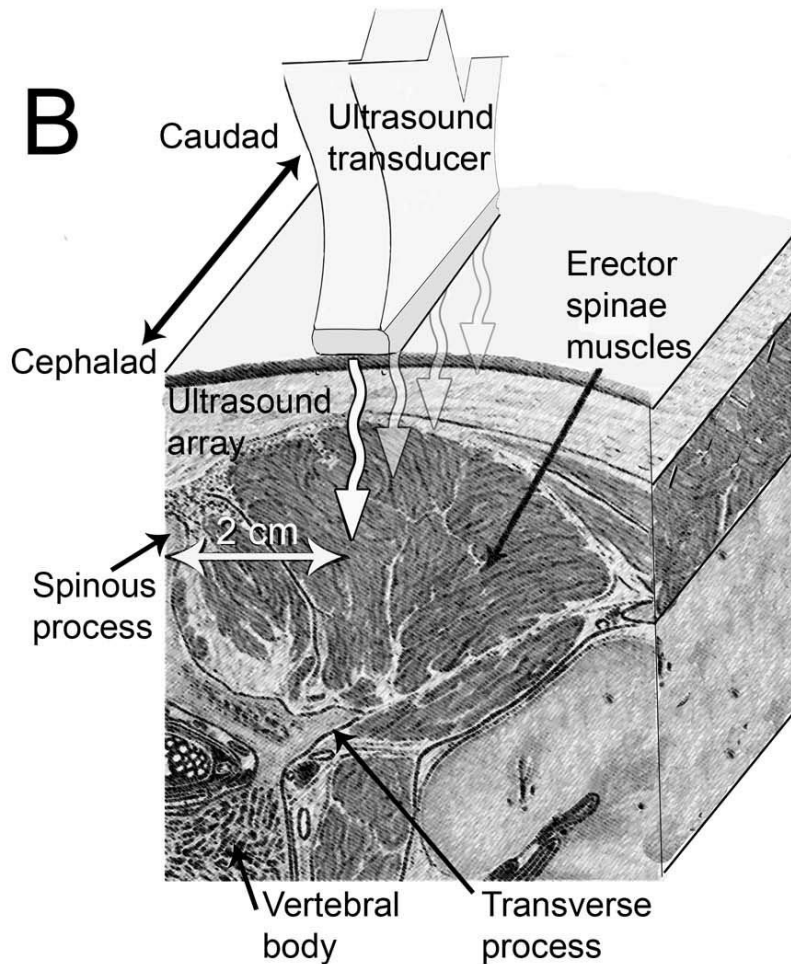


Figure 2.2 Location and placement of ultrasound transducer at 2 cm lateral to the middle of the spinous process of L2-3.

Source: Langevin HM, Stevens-Tuttle D, Fox JR, Badger GJ, Bouffard NA, Krag MH, Wu J, Henry SM: Ultrasound evidence of altered lumbar connective tissue structure in human subjects with chronic low back pain. *BMC musculoskeletal disorders* 2009, 10(1):151.

Thickness of TLF was calculated after converting the raw ultrasound data in MATLAB (The MathWorks, Natick, MA) by a program developed and used by Langevin et al. (Langevin et al., 2009). The program identifies a 1 cm region centered at the middle

of the image, located between the deep border of the dermis and superficial border of the muscle (Figure 2.3). Three areas within the region are prompted to be selected, the skin depth, fascia band top and the muscle depth. After that the program calculates the thickness of the subcutaneous, perimascular and combined zones. Perimascular zone thickness is the thickness between the more echogenic layered structure closest to the muscle separated by the nearest superficial echogenic layer by more than 2 mm (Langevin et al., 2009). Subcutaneous zone thickness is measured between the dermis and superficial border of the perimascular zone (Langevin et al., 2009). Combined subcutaneous and perimascular zone thickness is the thickness between the deep border of the dermis and superficial border of the muscle (Langevin et al., 2009).

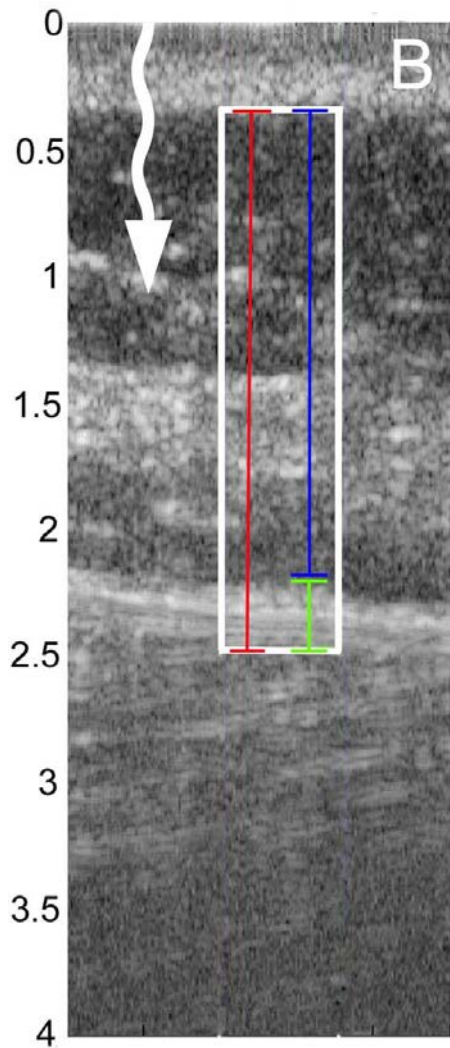


Figure 2.3 Ultrasound image of low back showing how the thickness of TLF was calculated. Small arrow represents the perimuscular zone thickness, medium arrow represents the subcutaneous zone and the big arrow represents the combined subcutaneous and perimuscular zone thickness.

Source: Langevin HM, Stevens-Tuttle D, Fox JR, Badger GJ, Bouffard NA, Krag MH, Wu J, Henry SM: Ultrasound evidence of altered lumbar connective tissue structure in human subjects with chronic low back pain. *BMC musculoskeletal disorders* 2009, 10(1):151.

2.3 MyotonPRO

A hand-held digital palpation device (myometer), called the MyotonPRO (Myoton AS, Tallinn, Estonia), was used to measure the biomechanical and viscoelastic properties of

the myofascial tissue at the low back. The properties measured were tone (oscillation frequency in Hz), elasticity (logarithmic decrement with no units), stiffness (N/m) and mechanical stress relaxation time (ms). Tone represents the state of tension of tissue in its passive state without any voluntary contraction, elasticity represents the ability of tissue to restore back to its original shape after the removal of an external force, stiffness represents the resistance of tissue to a contraction or external force, and mechanical stress relaxation time represents the time of the tissue to restore its original shape after a contraction or external force is removed (Bailey et al., 2013).

The Myoton was placed on low back areas, while the participant lay in a prone position, perpendicular to the target myofascial tissue, which was in a relaxed state. The testing end of the device, which is a probe of 3 mm diameter, applies a constant pre-pressure (0.18 N) on the skin surface that causes the tissue beneath it to be compressed (Lam et al., 2015). The pre-pressure applied by the probe is felt on the subject's skin as a small tap. A mechanical impulse (0.40 N, 15 ms) is released by the Myoton on the compressed subcutaneous tissue (Lam et al., 2015). The tissue responds back to the impulse by a damped oscillation that is recorded by the accelerometer in the Myoton. The damped oscillation from the measured tissue causes co-oscillation of the tissue being measured, subcutaneous tissue layers above the tissue being measured, the probe, measurement mechanism and the accelerometer attached to the measurement mechanism (Figure 2.4). The oscillation signal is processed by the Myoton to give values of the biomechanical properties mentioned above (Lam et al., 2015). The probe taps the skin 5 times (i.e. subcutaneous tissue is pre-compressed 5 times producing 5 mechanical

impulses and therefore 5 damped oscillations) and the average value of the results are given by the Myoton. In order to test whether the Myoton results are consistent, this procedure was done three times and an average value out of the three was calculated.

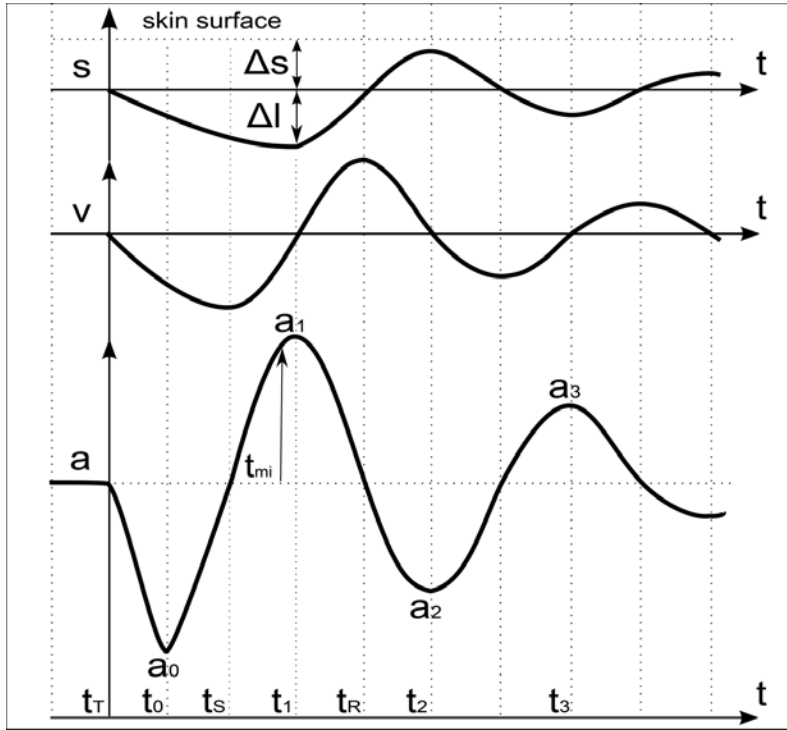


Figure 2.4 Relationship of displacement oscillation (s) and velocity oscillation (v) in relation to acceleration oscillation (a).

Source: Myoton AS: MyotonPRO Digital Palpation User Manual. 2015.

In Figure 2.4, a_0 means maximum acceleration, a_1 means maximum tissue resistance, a_2 means maximum opposite displacement due to residual inertia of tissue oscillation, a_3 means maximum displacement of the second period of oscillation, Δs means pre-compression of the subcutaneous tissue, Δl means maximum displacement of tissue, t_T means start of mechanical impulse, t_{mi} means end of mechanical impulse, t_0 means time when maximum acceleration was reached, t_1 means time when maximum displacement was reached, t_R means time when tissue returns to its original shape, t_2 means time when

maximum opposite displacement was reached and t_3 means time when maximum displacement of the second period of oscillation was reached.

The areas of low back used in this study were 3 cm lateral to the spinous process of L1 that targets the fascia above the paravertebral muscles, 5 cm lateral to the spinous process of L3 that targets the fascia above the quadratus lumborum muscle and the area below the 12th rib that targets the fascia over the latissimus dorsi (Ercole et al., 2010). These particular areas were chosen because previous investigators have found changes in pain in LBP patients after using the Fascial Manipulation techniques (Ercole et al., 2010). The areas were located by looking at a picture of low back anatomy and by hand palpation. After L1 and L3 were found, measurements of 3 cm and 5 cm respectively, to the left and right sides of the vertebrae, was marked with a surgical pen. The lower area was pretty straightforward to locate by counting and identifying the 12th rib.

2.4 Flexibility Test and Pain Scale

In order to assess flexibility, participants were asked to stand on a step tool, bend forward and reach down as much as they could without bending their knees. A measuring tape was used to calculate the distance from the floor to the tip of the participants' fingers to measure their forward flexion. The height of the tool was taken into account during measurement.

An Oswestry Low Back Pain Questionnaire (Fairbank et al., 1980) was given to the participants when they came in for testing. This questionnaire reported their average

intensity of pain on a scale of 0 to 10, with 0 being no pain and 10 being most severe pain, and how it affected their ability to lead their daily life activities.

2.5 Statistical Methods

Paired t-test was used to compare MELT and control group's connective tissue thickness, flexibility tests and all biomechanical properties measured by the Myoton. Significance level was set at $\alpha = 0.05$. Microsoft Excel 2013 (Microsoft, Redmond, WA) was used to perform the t-tests and correlation scatter plots between thickness and pain or flexibility (results not shown). Wilcoxon signed-rank test was used to compare the pain scale between MELT and control group participants.

2.6 Testing Procedure For MELT vs Control Group

All participants had to come in for two days for testing. The first day consisted of ultrasound, Myoton, flexibility and pain scale recording before MELT for MELT group or no activity for control group. MELT group participants did MELT self-treatment while watching a 30 minute video and rested for 5 minutes after which they were re-measured with the ultrasound and Myoton and their flexibility and pain scale were recorded again. These participants were given all the required MELT tools and a 4-week MELT plan. They came back after 4 weeks and re-measured in the same manner as their initial measurement. They were also given a questionnaire to assess if they had been following the MELT plan accordingly.

Control group participants had a 35 minute non-active, wait time before initial and second measurement. They left the testing facility without any MELT tools and were

instructed not to perform MELT for 4 weeks. After 4 weeks they were re-tested following the initial testing procedure.

2.7 MELT Procedures

The 4-week MELT plan for MELT group participants were explained visually in detail in DVDs and the MELT method book provided to them (Figure 2.5).



Figure 2.5 Complete MELT tool kit.

Source: Sue Hitzmann, creator of the MELT method.

The plan consisted of

- a) Mini Soft Ball Foot Treatment and Rebalance Sequence for 4 times in the first week taking 1 day off between the days they MELT
- b) First week's treatment with the addition of Upper and Lower Body Compression for a minimum of 4 times a day in the second week
- c) First two weeks' treatment with the addition of Lower Body Length and Low Back Release Sequence for a minimum of 5 times a day varying the order of sequences each time they MELT in the third week
- d) All the previous weeks' sequences in a specific order in the fourth week

The initial MELT treatment consisted of all these sequences condensed in 30 minutes.

CHAPTER 3

RESULTS

This chapter exhibits the results of ultrasound imaging and MyotonPRO as well as results from the Oswestry Low Back Pain Scale and forward bending tests.

3.1 Ultrasound Imaging

Three zones, the perimuscular zone, the subcutaneous zone and the combined subcutaneous and perimuscular zone thickness of the connective tissue before and after (immediately after and 4 weeks after) MELT treatment or non-MELT for MELT and control group participants were measured by the ultrasound (Tables 3.1 and 3.2). In Tables 3.1 and 3.2, Pre means before MELT or non-active wait time, Post means immediately after MELT or non-active wait time 4Post means 4 weeks after MELT or non-MELT; SUBQ means subcutaneous zone thickness, PM means perimuscular zone thickness and TOT means combined subcutaneous and perimuscular zone thickness.

Table 3.1 Mean, standard deviation and percent change of connective tissue thickness of MELT and control subjects before, immediately after and 4 weeks after MELT and non-MELT on left side; values in bold exhibit statistical significance ($p < 0.05$)

		Thickness (cm)	PRE			POST			4POST		
			SUBQ	PM	TOT	SUBQ	PM	TOT	SUBQ	PM	TOT
LEFT	MELT Group	Mean	0.832	0.036	0.868	0.784	0.026	0.810	0.762	0.026	0.788
		SD	0.382	0.011	0.393	0.329	0.010	0.339	0.337	0.011	0.348
		% Change				-5.77	-27.8	-6.68	-8.41	-27.8	-9.21
	Control Group	Mean	0.939	0.041	0.980	0.918	0.041	0.959	0.941	0.044	0.985
		SD	0.405	0.013	0.418	0.375	0.012	0.387	0.430	0.013	0.443
		% Change				-2.23	0	-2.14	0.21	7.31	0.51

Table 3.2 Mean, standard deviation and percent change of connective tissue thickness of MELT and control subjects before, immediately after and 4 weeks after MELT and non-MELT on right side; values in bold exhibit statistical significance ($p < 0.05$)

		Thickness (cm)	PRE			POST			4POST		
			SUBQ	PM	TOT	SUBQ	PM	TOT	SUBQ	PM	TOT
RIGHT	MELT Group	Mean	0.814	0.034	0.848	0.771	0.025	0.796	0.776	0.025	0.801
		SD	0.395	0.011	0.406	0.387	0.008	0.395	0.397	0.009	0.406
		% Change				-5.28	-26.4	-6.13	-4.67	-26.4	-5.54
	Control Group	Mean	0.884	0.039	0.923	0.889	0.041	0.930	0.856	0.040	0.896
		SD	0.374	0.011	0.385	0.364	0.012	0.372	0.344	0.013	0.357
		% Change				0.57	5.12	0.76	-3.16	2.56	-2.93

3.1.1 Perimuscular Zone Thickness

A statistical significant decrease in perimuscular zone connective tissue thickness was seen on both left ($p=0.004$) and right ($p=0.026$) sides of L2 paraspinal muscles immediately after MELT (Figure 3.1.1a). A significant decrease in perimuscular zone thickness was also seen after subjects were exposed to 4 weeks of MELT on both left ($p=0.004$) and right ($p=0.026$) sides of L2 paraspinal muscles (Figure 3.1.1a).

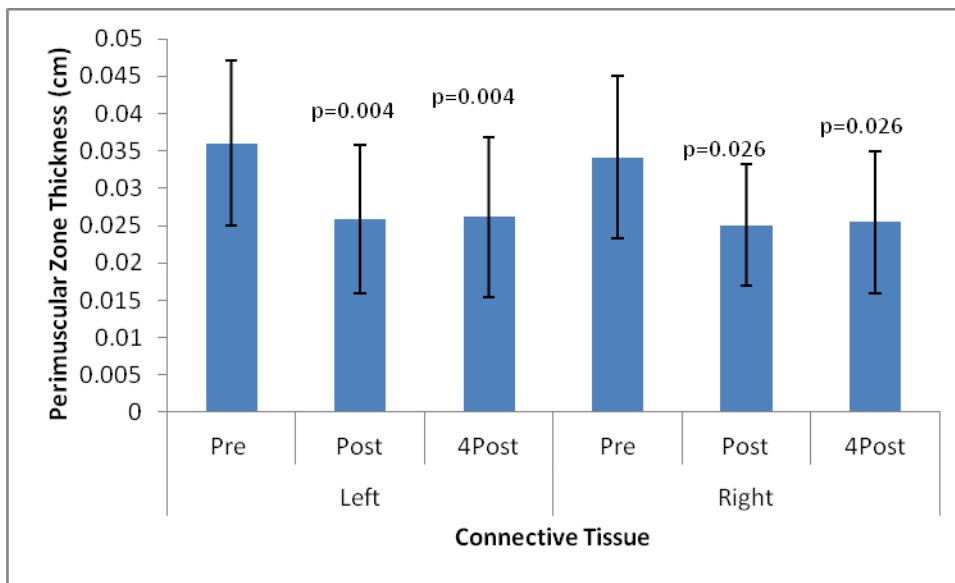


Figure 3.1.1a Perimuscular zone thickness of connective tissue pre-MELT, immediately after MELT and 4 weeks post MELT on left and right sides of L2 paraspinal muscles.

There was no statistical significant change in perimuscular zone connective tissue thickness in control subjects who did not MELT during the initial ultrasound measurement and 4 weeks post measurement (Figure 3.1.1b).

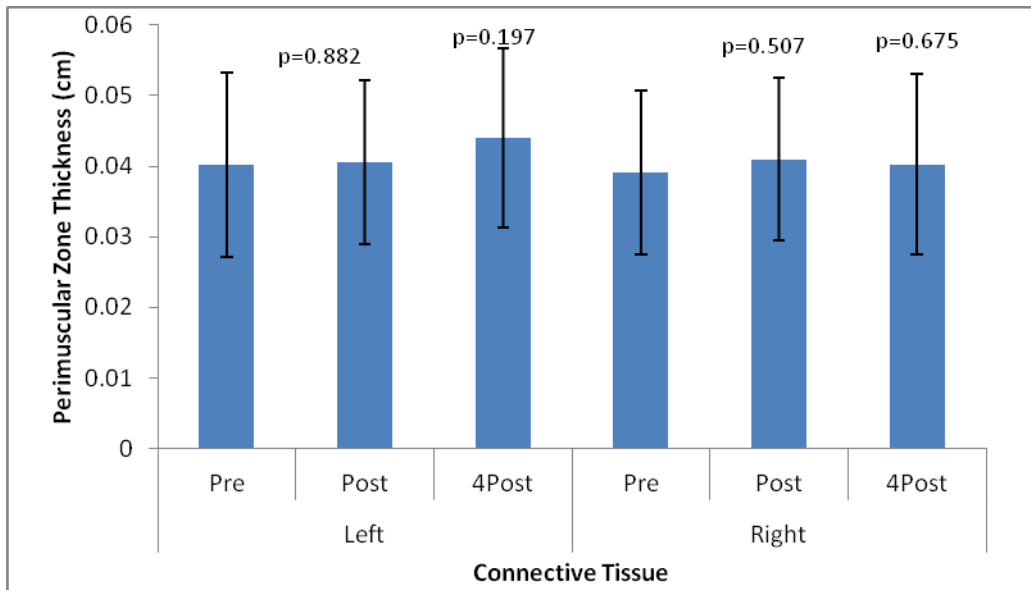


Figure 3.1.1b Perimuscular zone thickness of connective tissue of subjects who did not MELT on left and right sides of L2 paraspinal muscles.

3.1.2 Subcutaneous Zone Thickness

A statistical significant change in subcutaneous zone connective tissue thickness was seen after 4 weeks of MELT on the left ($p=0.026$) side of the L2 paraspinal muscles. Other changes were not significant (Figure 3.1.2a).

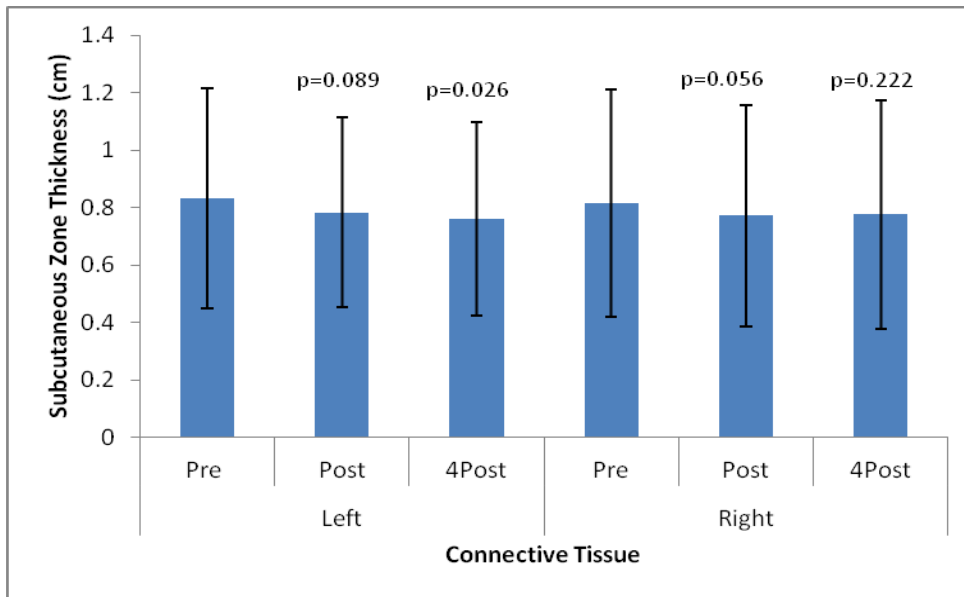


Figure 3.1.2a Subcutaneous zone thickness of connective tissue pre-MELT, immediately after MELT and 4 weeks post MELT on left and right sides of L2 paraspinal muscles.

No statistical significant change was seen in subcutaneous zone thickness of connective tissue in control participants who did not MELT (Figure 3.1.2b).

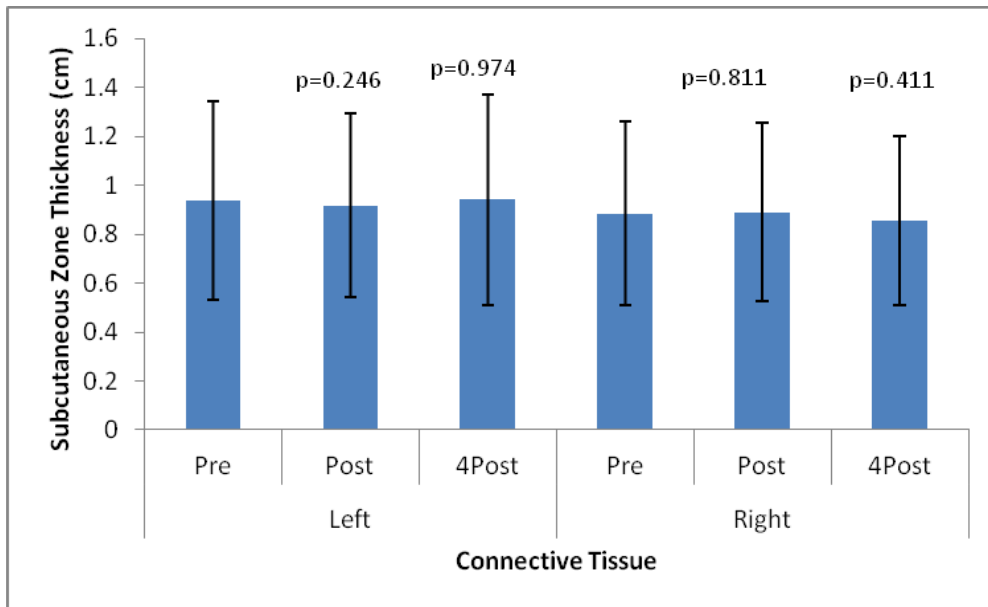


Figure 3.1.2b Subcutaneous zone thickness of connective tissue who did not MELT on left and right sides of L2 paraspinal muscles.

3.1.3 Combined Subcutaneous And Perimuscular Zone Thickness

A statistical significant decrease in thickness of combined subcutaneous and perimuscular zone connective tissue was seen on both left ($p=0.043$) and right ($p=0.026$) sides of L2 paraspinal muscles immediately post MELT (Figure 3.1.3a). There was a statistical significant decrease on the left side ($p=0.013$) but not on the right ($p=0.144$) 4 weeks after MELT (Figure 3.1.3a).

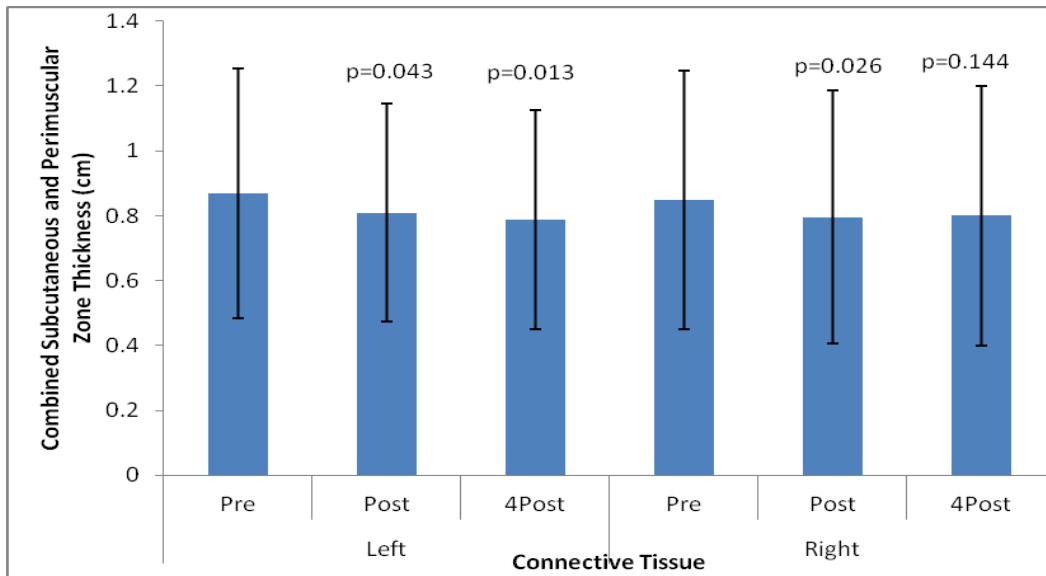


Figure 3.1.3a Combined subcutaneous and perimuscular zone thickness of connective tissue pre-MELT, immediately after MELT and 4 weeks post MELT on left and right sides of L2 paraspinal muscles.

No statistical significant change in combined subcutaneous and perimuscular zone thickness was seen in control subjects who did not MELT (Figure 3.1.3b).

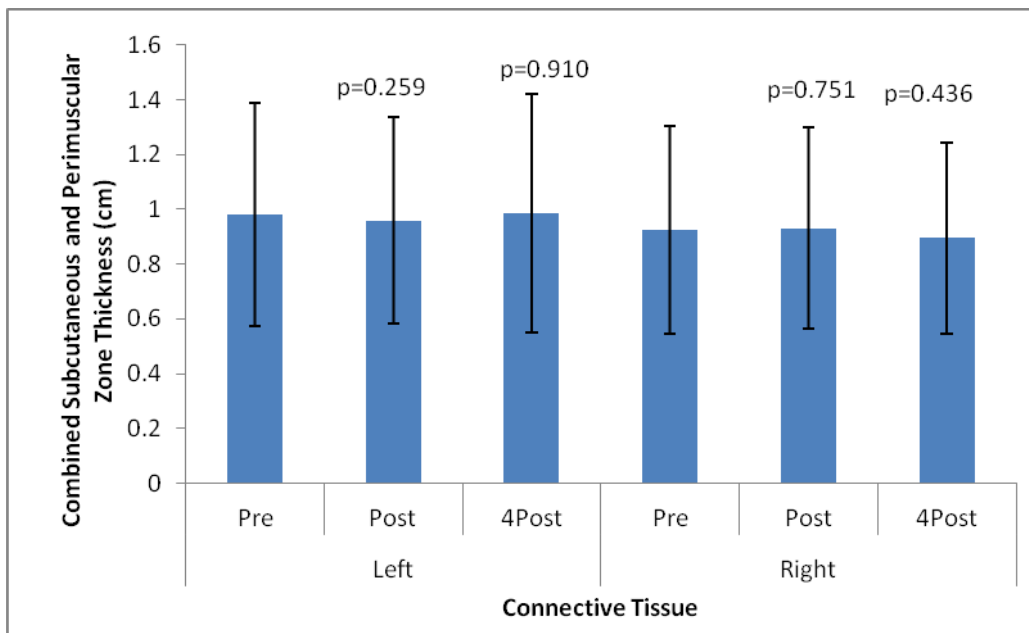


Figure 3.1.3b Combined subcutaneous and perimuscular zone thickness of connective tissue of subjects who did not MELT on left and right sides of L2 paraspinal muscles.

3.2 MyotonPRO

The MyotonPRO measured the biomechanical and viscoelastic properties of myofascia in the thoracolumbar fascia (TLF) region in three regions on both left and right sides – 3 cm lateral to the spinous process of L1 (upper area), 5 cm lateral to the spinous process of L3 (mid area) and the region immediately below the 12th rib (lower area).

3.2.1 Stiffness

An overall summary of average, standard deviation and percent change in stiffness values for MELT and control group participants is provided in Tables 3.3 and 3.4.

Table 3.3 Mean, standard deviation and percent change for stiffness measurements of MELT group participants at left and right sides

MELT Group	LEFT	Stiffness (N/m)	Lower			Mid			Upper		
			PRE	POST	4POST	PRE	POST	4POST	PRE	POST	4POST
		Mean	342.1	339.6	330.9	251	248.4	245.3	307.1	305.6	291.3
		SD	112	79.3	88	72.6	55.3	61.5	70	59.2	75.6
		% Change		-0.73	-3.27		-1.04	-2.27		-0.49	-5.14
	RIGHT	Mean	347.3	341	347	229.7	230	227.9	289.8	290.9	289.7
		SD	102	77	99	56.8	58	63.7	66.3	56.8	59.5
		% Change		-1.81	-0.09		0.13	-0.78		0.38	-0.03

Table 3.4 Mean, standard deviation and percent change for stiffness measurements of control group participants at left and right sides

Control Group	LEFT	Stiffness (N/m)	Lower			Mid			Upper		
			PRE	POST	4POST	PRE	POST	4POST	PRE	POST	4POST
		Mean	310.5	324.9	298.2	242.5	247.6	249.5	304.6	300.9	289.2
		SD	70.1	97	68.1	77.1	86.2	82	71.6	65.3	67.4
		% Change		4.63	-3.96		2.10	2.89		-1.21	-5.06
	RIGHT	Mean	324.8	323.1	316.5	226.1	229.9	233.7	304.8	299	295.6
		SD	91.5	70.1	95.5	61.2	68.5	65.8	73	73.5	85.9
		% Change		-0.52	-2.55		1.68	3.36		-1.90	-3.02

There was no statistical significant change in stiffness measurements before, immediately after and 4 weeks after MELT in the lower area in the region immediately below the 12th rib (Figure 3.2.1a).

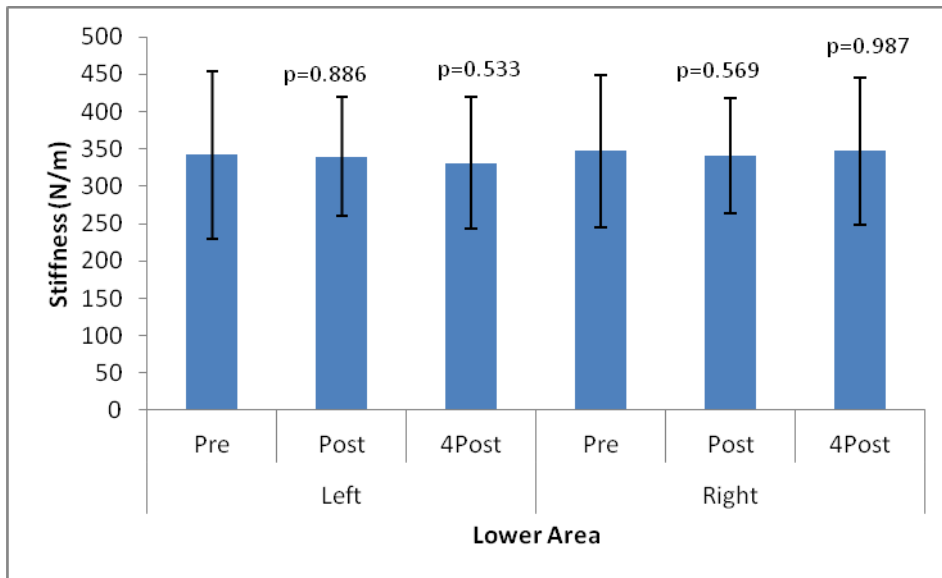


Figure 3.2.1a Stiffness measurement before, immediately after and 4 weeks after MELT in the region below the 12th rib (lower area) on left and right side.

No statistical significant change in stiffness was observed in control participants' measure of stiffness in the lower area (Figure 3.2.1b).

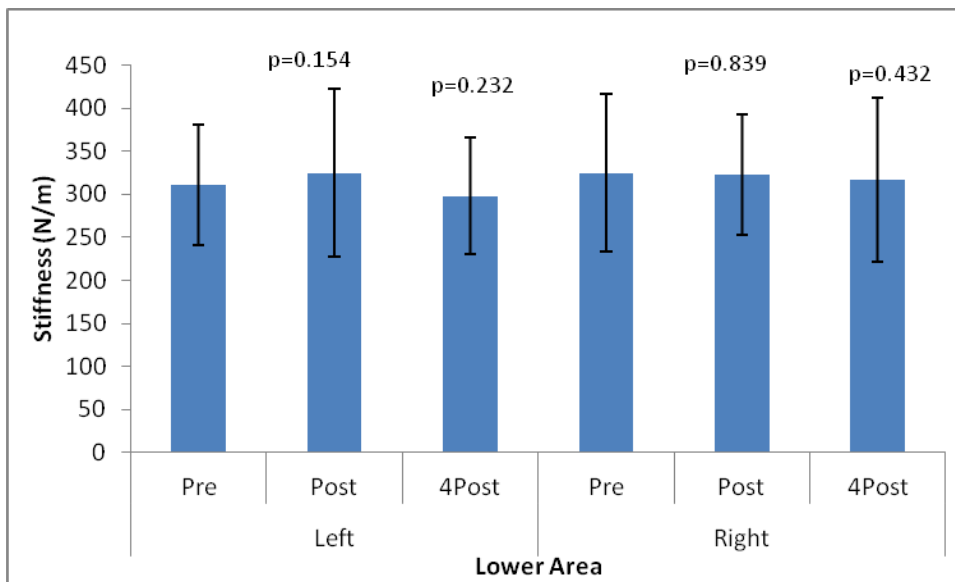


Figure 3.2.1b Stiffness measurement in control participants who did not MELT in the region below the 12th rib (lower area) on left and right side.

No statistical significant change in stiffness was seen in the mid area, 5 cm lateral to the spinous process of L3 before, immediately after and 4 weeks after MELT (Figure 3.2.1c).

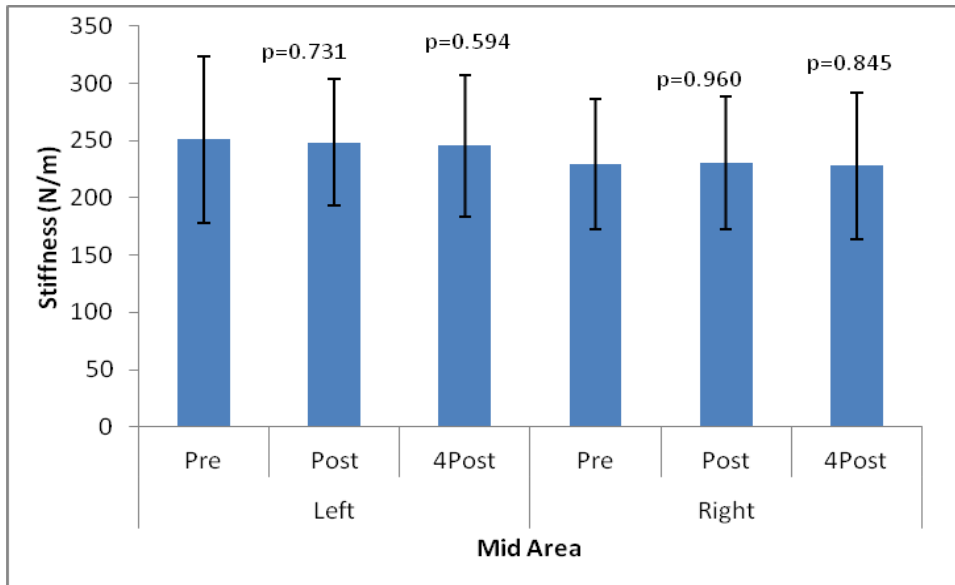


Figure 3.2.1c Stiffness measurement before, immediately after and 4 weeks after MELT in the region 5 cm lateral to the spinous process of L3 (mid area) on left and right sides.

No statistical significant change in stiffness was observed in control participants' measure of stiffness in the mid area (Figure 3.2.1d).

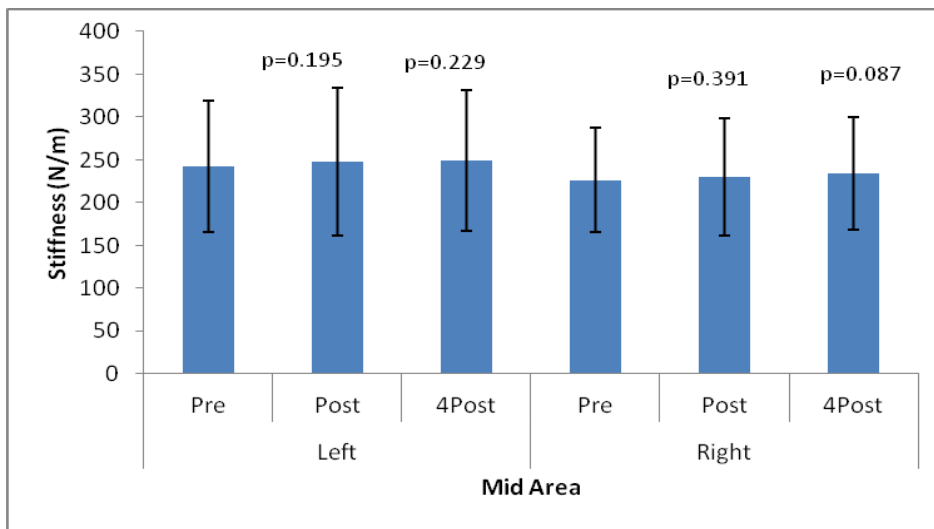


Figure 3.2.1d Stiffness measurement in control participants who did not MELT in the region 5 cm lateral to the spinous process of L3 (mid area) on left and right side.

There was also no statistical significant change in stiffness in the region 3 cm lateral to the spinous process of L1 on either left or right sides before, immediately after and 4 weeks after MELT in the treatment group (Figure 3.2.1e).

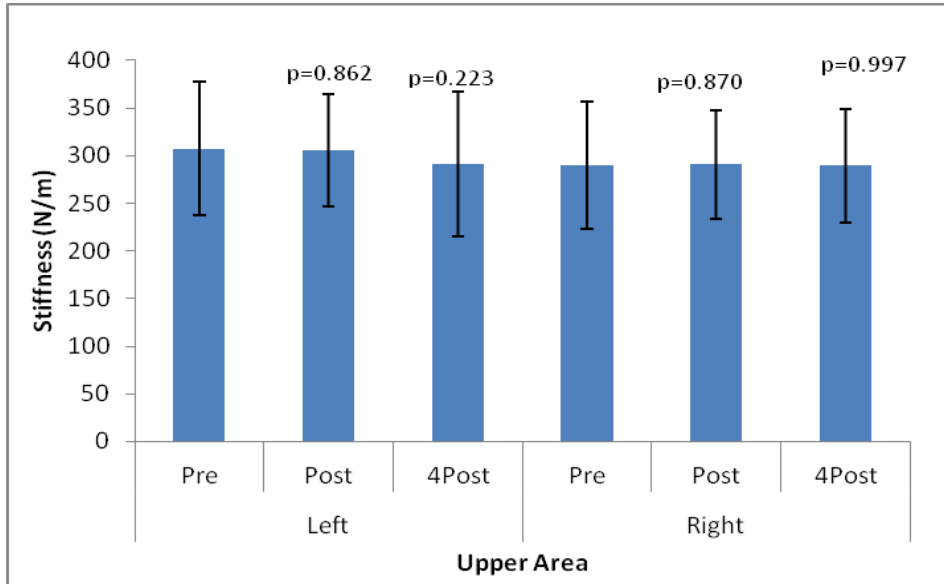


Figure 3.2.1e Stiffness measurement before, immediately after and 4 weeks after MELT in the region 3 cm lateral to the spinous process of L1 (upper area) on left and right sides.

No statistical significant change in stiffness was observed in control participants' measure of stiffness in the upper area (Figure 3.2.1f).

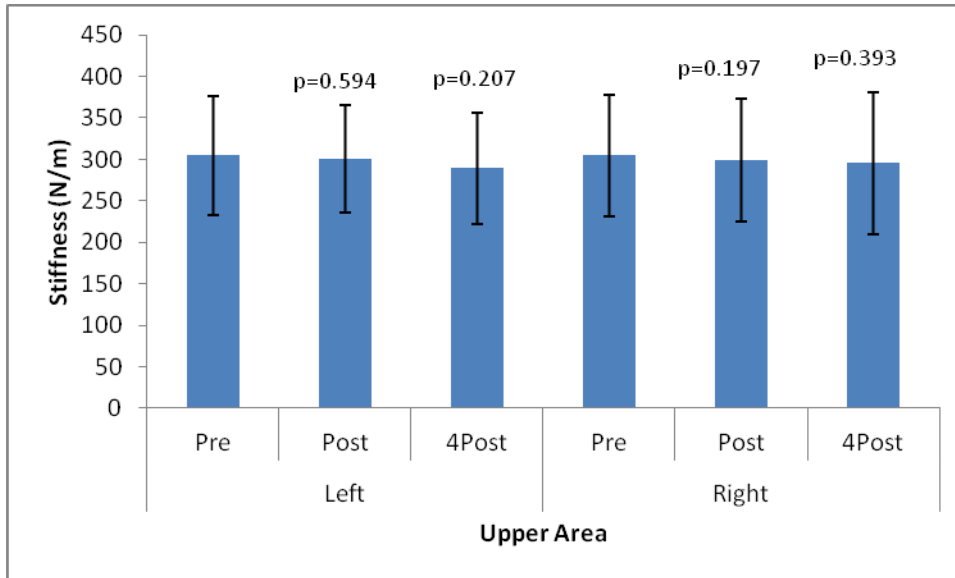


Figure 3.2.1f Stiffness measurement in control participants who did not MELT in the region 3 cm lateral to the spinous process of L1 (upper area) on left and right side.

3.2.2 Mechanical Stress Relaxation Time

An overall summary of average, standard deviation and percent change in mechanical stress relaxation time values for MELT and control group participants is provided in Table 3.5 and Table 3.6.

Table 3.5 Mean, standard deviation and percent change for mechanical stress relaxation time measurements of MELT group participants at left and right sides; values in bold exhibit statistical significance ($p < 0.05$)

MELT Group	LEFT	Stress Relaxation Time (ms)	Lower			Mid			Upper		
			PRE	POST	4POST	PRE	POST	4POST	PRE	POST	4POST
		Mean	17	18.3	18.2	21.9	21.8	22.4	17.6	17.6	18.4
		SD	4.53	3.75	3.17	4.66	3.88	4.38	3.07	2.69	3.46
		% Change		8.04	7.20		-0.54	2.11		0	4.41
	RIGHT	Mean	17	16.7	16.5	23.6	23.9	23	18.5	18.3	18
		SD	4.76	4.04	4.21	4.56	4.90	4.35	3.55	3.44	3.46
		% Change		-1.76	-2.94		1.26	-2.54		-3.10	-2.53

Table 3.6 Mean, standard deviation and percent change for mechanical stress relaxation time measurements of control group participants at left and right sides

Control Group	LEFT	Stress Relaxation Time (ms)	Lower			Mid			Upper		
			PRE	POST	4POST	PRE	POST	4POST	PRE	POST	4POST
		Mean	18	17.7	19.5	22.9	22.5	23.1	18	18.3	19.4
		SD	3.58	3.87	4.86	4.88	5.12	5.36	3.29	3.22	4.15
		% Change		-1.67	8.33		-1.75	0.87		1.67	7.78
	RIGHT	Mean	17.6	17.8	18.9	24.1	24	23.6	18.1	18.4	19.4
		SD	3.86	3.70	5.07	4.52	4.97	5.38	3.86	4.16	4.64
		% Change		1.14	7.38		-0.41	-2.07		1.66	7.18

A statistical significant increase in mechanical stress relaxation time was seen on the left side of the region below the 12th rib in treatment participants immediately after ($p=0.004$)

and 4 weeks after ($p=0.048$) MELT (Figure 3.2.2a). The change in right side in these treatment participants was not statistically significant (Figure 3.2.2a).

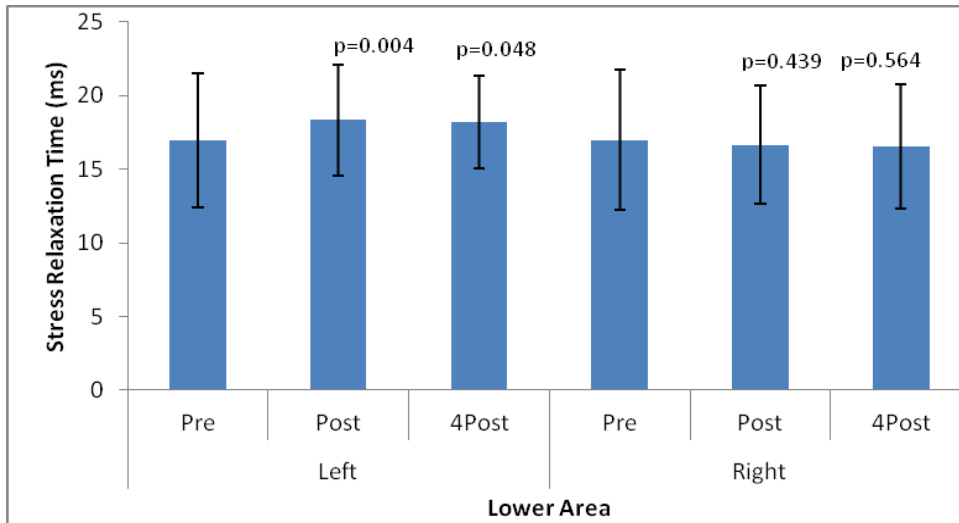


Figure 3.2.2a Mechanical stress relaxation time in before, immediately after and 4 weeks after MELT in the region below the 12th rib (lower area) on left and right side.

No statistical significant change in stress relaxation time in the lower area was observed in control participants who did not MELT (Figure 3.2.2b).

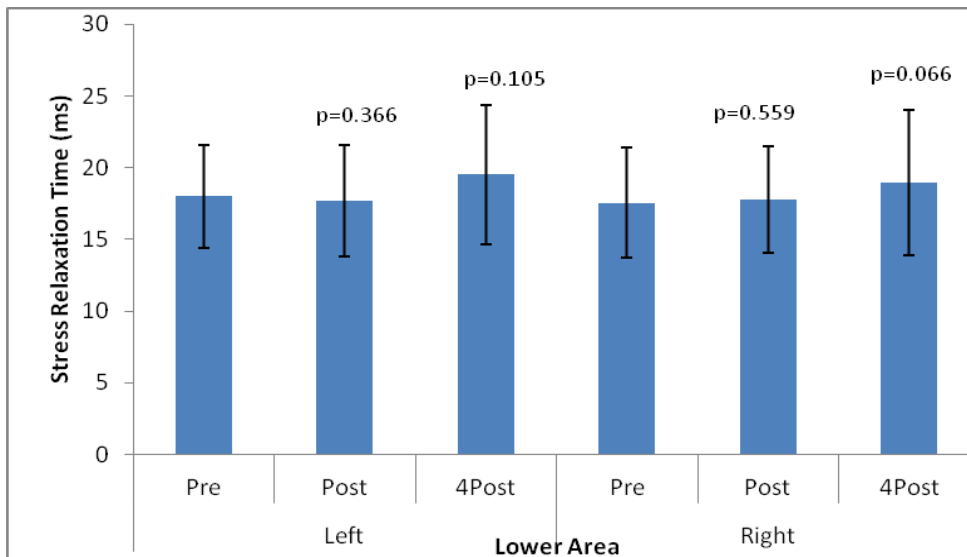


Figure 3.2.2b Mechanical stress relaxation time in control participants who did not MELT in the region below the 12th rib (lower area) on left and right side.

There was no statistical significant change in stress relaxation time in the mid region of 5 cm lateral to the spinous process of L3 of treatment participants before, immediately after and 4 weeks after MELT (Figure 3.2.2c).

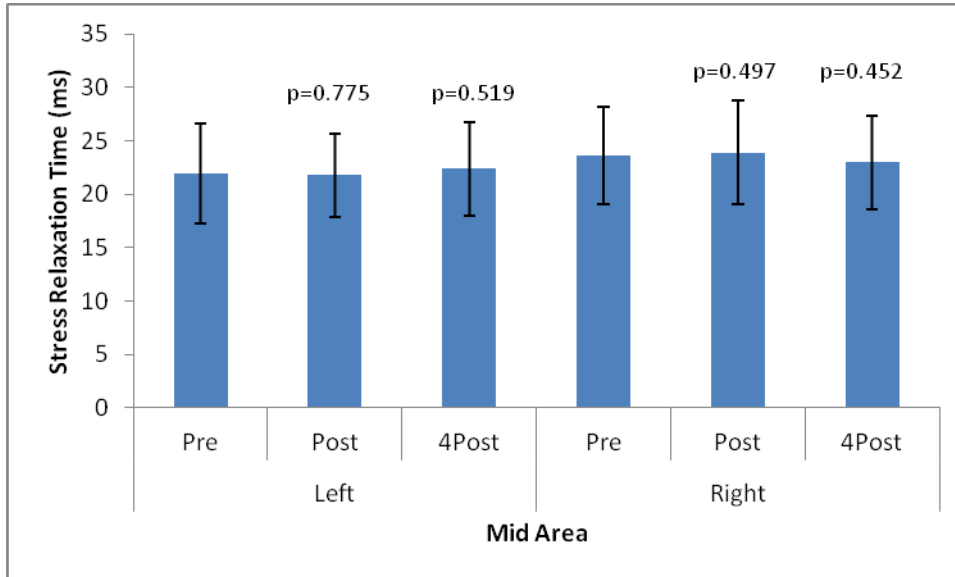


Figure 3.2.2c Mechanical stress relaxation time in before, immediately after and 4 weeks after MELT in the region 5 cm lateral to the spinous process of L3 (mid area) on left and right side.

No statistical significant change in stress relaxation time in the mid area was observed in control participants who did not MELT (Figure 3.2.2d).

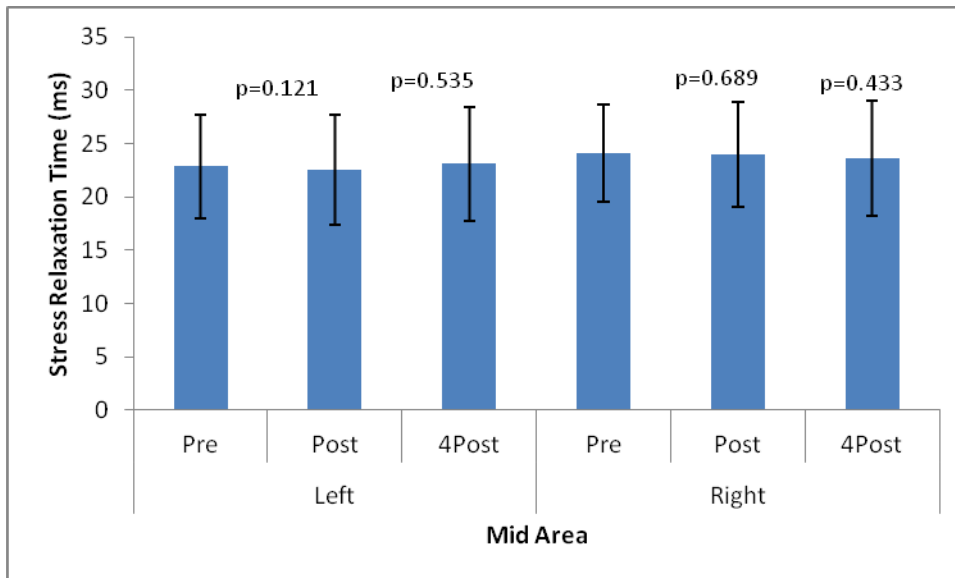


Figure 3.2.2d Mechanical stress relaxation time in control participants who did not MELT in the region 5 cm lateral to the spinous process of L3 (mid area) on left and right side.

No statistical significant change in stress relaxation time in the upper region of 3 cm lateral to the spinous process of L1 of treatment participants before, immediately after and 4 weeks after MELT (Figure 3.2.2e).

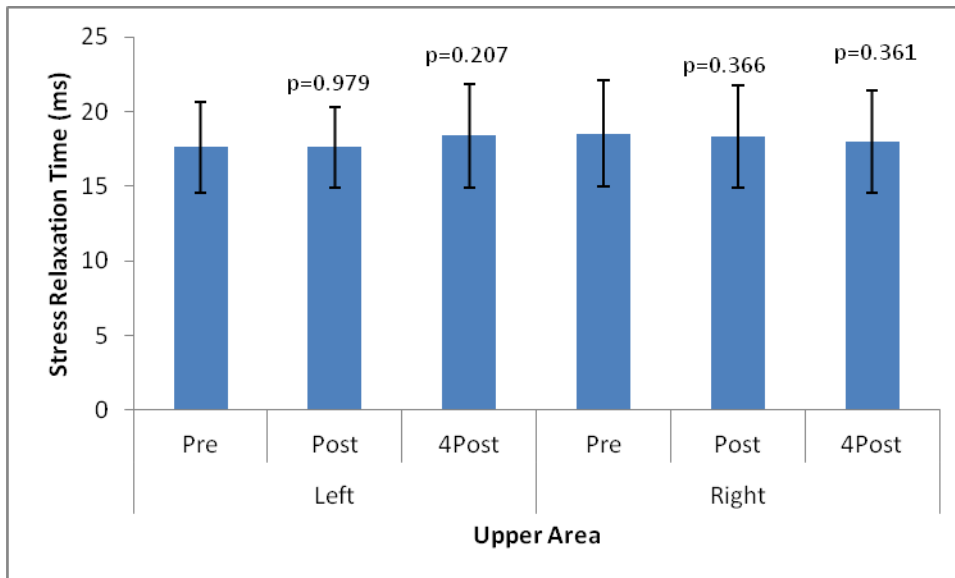


Figure 3.2.2e Mechanical stress relaxation time in before, immediately after and 4 weeks after MELT in the region 3 cm lateral to the spinous process of L1 (upper area) on left and right side.

No statistical significant change in stress relaxation time in the upper area was observed in control participants who did not MELT (Figure 3.2.2f).

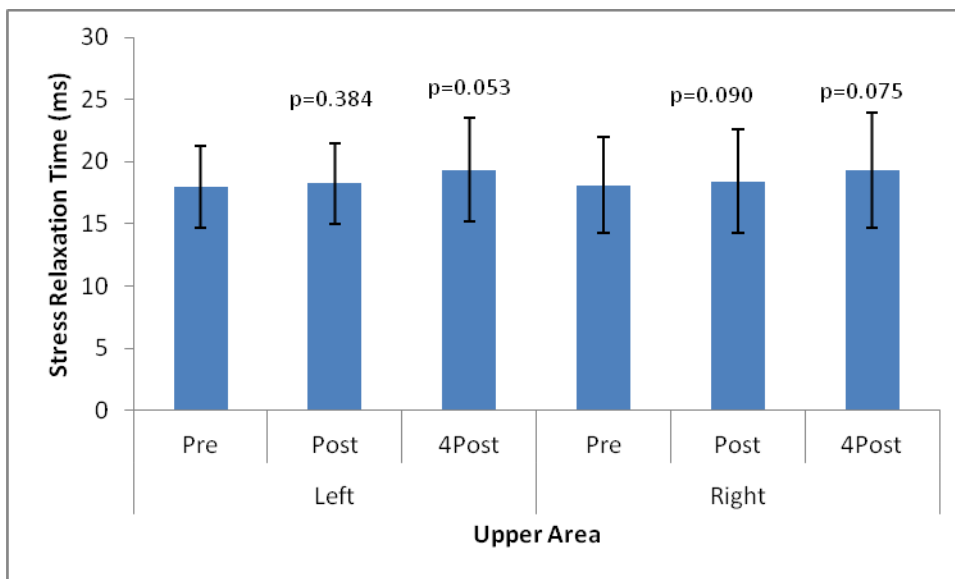


Figure 3.2.2f Mechanical stress relaxation time in control participants who did not MELT in the region 3 cm lateral to the spinous process of L1 (upper area) on left and right side.

The remaining two biomechanical properties, elasticity and tone were also measured for treatment and control participants in the three areas mentioned above. No statistically significant changes were observed in either properties on both groups (not pictured).

3.3 Flexibility Test

Flexibility tests were conducted by participants performing trunk flexion reaching down to the floor. Flexibility increased significantly in treatment group participants from -17.64 ± 7.2 inches to -16.05 ± 6.5 inches ($p=0.012$) immediately after MELT. Flexibility also increased significantly in treatment group participants from -17.64 ± 7.2 inches to -13.41 ± 4.5 inches ($p=0.002$) after 4 weeks of MELT.

Flexibility changed from -16.75 ± 7.4 inches to -17.41 ± 6.4 inches ($p=0.179$) in control participants after initial measurement and from -16.75 ± 6.7 inches to -16.86 ± 7.5 inches ($p=0.825$) after 4 weeks of non-MELT. None of these changes were statistically significant.

3.4 Pain Scale

Pain scale of participants in both treatment and control groups were determined by Oswestry Low Back Pain Scale. There was a significant reduction of pain in treatment participants from 4.4 (range 2-8) to 2.5 (range 1-6) after initial MELT treatment ($p=0.00014$, Wilcoxon Signed Rank Test). There was also a significant reduction of pain in the same group from 4.4 (range 2-8) to 3.0 (range 0-7) after 4 weeks of MELT treatment ($p=0.0124$, Wilcoxon Signed Rank Test). No significant change in pain was observed in control participants.

CHAPTER 4

DISCUSSION

The goal of this study was to demonstrate whether thoracolumbar connective tissue and biomechanical and viscoelastic properties of myofascia in the low back region of chronic LBP subjects change as a result of MELT. Results from ultrasound imaging showed that the perimuscular zone thickness of connective tissue decreased 26.4% on the left side and 27.8% on the right side of L2 paraspinal muscles in MELT group participants (Tables 3.1 and 3.2). This decrease was significant ($p < 0.05$) (Figure 3.1.1a) and can be seen in Figure 4.1. There was barely any significant change in subcutaneous zone connective tissue thickness (Tables 3.1 and 3.2).

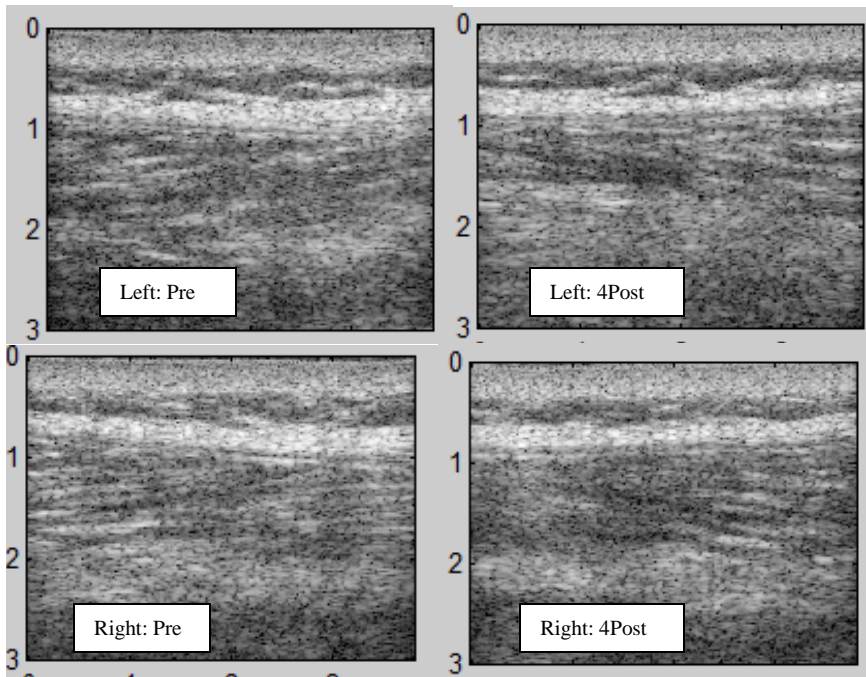


Figure 4.1 Ultrasound images showing connective tissue thickness change. Left images show thickness before application of MELT. Right images show thickness change 4 weeks after MELT.

For combined subcutaneous and perimuscular zone connective tissue thickness, about 6% decrease was seen immediately after MELT on left and right sides and 9.21% and 5.54% decrease was seen 4 weeks after MELT on left and right sides respectively (Tables 3.1 and 3.2). All changes were significant ($p < 0.05$) except for the post 4-week decrease on the right side. Although there were no significant decreases in subcutaneous zone thickness, the perimuscular zone thickness decrease was so profound that its change transmitted into the combined subcutaneous and perimuscular zone thickness bringing it down by a significant level.

After averaging for both left and right sides of perimuscular zone connective tissue thickness, the decrease came about to be 27%. This percentage decrease coincided with Langevin et al.'s study where they showed that chronic LBP patients had a 25% increased perimuscular zone thickness (Langevin et al., 2009). Langevin et al. were not sure whether their observed increase was due to cause or effect of LBP. They hypothesized that inherent abnormal connective tissue could be the cause of LBP. Abrupt movement or relaxation patterns different from the subjects' daily lives could alter connective tissue structure, which was hypothesized as effect (Langevin et al., 2009). In both cases, altered connective tissue was present whether as a cause or effect of LBP. Moreover, Langevin et al.'s study exhibited data on subjects without LBP and how their connective tissue thickness is significantly lower than subjects with LBP. They suggested that increased thickness and disorganization of tissue layers, due to inflammation, fibrosis, adhesions and fatty infiltrations decrease range of motion (Langevin & Sherman, 2007). Trapping of nerve fibers in the collagen matrix can also occur due to fatty

infiltrations, thereby causing pain (Dittrich, 1956). These observations add to the hypothesis that increased connective tissue thickness is associated with pain and limited range of motion.

It cannot be said that in this study, connective tissue was being remodeled as a result of MELT, since remodeling of tissues cannot occur so fast and can take from several weeks to months for tissues affected by chronic pain (Langevin et al, 2009). Rehydration of connective tissue due to increased hyaluronic acid production is also unlikely as this would increase the volume of the tissue and cause swelling, as opposed to the observed reduction of thickness. It was also not in the scope of this study to measure amount of hyaluronic acid produced if any. This could be a future aspect to look into if follow up studies are conducted.

A more plausible reason for the decrease in connective tissue thickness can be attributed to the stretch of tissue in the lateral direction. Manual fascial techniques, as mentioned in the “Background Information” section, contribute to stretching of tissue, as a result of tension, where touch or pressure is applied. This causes a permanent (plastic) deformation of tissue, making it elongated in the lateral direction. Connective tissue mobility is changed by breaking the links between adjacent bundles of collagen fibers in tissue. The term “microfailure”, is used to describe the breakage of individual collagen fibers and fiber bundles when placed under tension (Threlkeld, 1992). If the force is removed, then the broken fibers do not add to tissue recoiling. Some fibers remain intact and do not break, causing tissue recoiling. The resulting connective tissue structure

reaches a new length that exhibits the balance between intact fibers that were elastically recoiled and the breakage of fibers due to elongation.

In this study, only the thickness i.e. the height in transverse direction was studied. Due to limitation in measuring the elongation, lateral strain could not be obtained. Assuming that biological tissue has a Poisson's ratio between the ranges of 0.3-0.4, the reduction in perimuscular zone connective tissue thickness of 27% (ratio of 0.27) would seem reasonable. With a value of 0.3 for Poisson's ratio, for a 30% decrease in strain in the transverse direction, a 70% increase in strain in the lateral direction would be expected. Therefore, for future studies, a mechanism to measure the elongation of connective tissue would be highly desirable. By measuring the elongation as well as the thickness, the cause of decrease in thickness can be understood more clearly.

Connective tissue is a viscoelastic material, i.e. it is composed of solid-fluid components. Viscoelastic materials disperse energy when stretched, which causes fluid to be pushed out of tissues (Özkaya et al., 2012). This flow of fluid out from connective tissue can be another reason to explain why thickness of tissue decreased. The deformation caused by lateral elongation of tissue is time-dependent on the load applied. A viscoelastic material responds to applied load with a steady increase in deformation (Özkaya et al., 2012). The material deforms under high stress initially but undergoes a gradual decrease in stress with time under constant deformation (Özkaya et al., 2012). The decrease in stress with time is called stress relaxation. Hence, structural changes in connective tissue would depend on stress applied as a result of MELT. For this reason, the effect of discontinuing MELT after 4 weeks should be studied to see if the connective

tissue had deformed permanently as a result of stress relaxation or if the deformation was reversed after the stress was removed. Given our understanding of viscoelastic materials, elongation in subjects' connective tissue of this study was more likely caused by the stress applied from curvature of the soft body roller used at the back for Lower Body Length and Low Back Release sequences of MELT.

It must be noted that the terms superficial and deep fascia was not used in this study when describing connective tissue thickness with ultrasound. This was because of the ambiguity in establishing an anatomical demarcation between these two structures as also observed by Langevin et al. (Langevin et al., 2009). Aligned with Langevin et al.'s study (Langevin et al., 2009), ultrasound images of this study showed multiple layers consisting of dense connective tissue along with fat layers without any clear separation between superficial and deep fascia. Other images showed a thick dense connective tissue layer over the paraspinal muscles making it difficult to know whether this was superficial fascia with multiple connective tissue layers or whether this was superficial fascia rich with excessive fat layers.

Three areas of the low back region, where most change due to Fascial Manipulation technique occurs, was demonstrated by Ercole et al. (Ercole et al., 2010), were chosen for the MyotonPRO measurements. The state of tension (frequency) and elasticity (decrement) data showed no sensible values or statistical significance, and therefore were not further examined. Stiffness was a key biomechanical property that was under investigation. On the left side of all three areas, decreasing trend in stiffness was observed. The lower area below the 12th rib showed stiffness reduction of 0.73% and

3.27% after initial and 4-week MELT respectively (Table 3.3). The mid area, 5 cm lateral to the spinous process of L3, showed about 1% and 2.27% stiffness decrease after initial and 4-week MELT respectively (Table 3.3). The upper area, 3 cm lateral to the spinous process of L1, exhibited 0.49% and 5.14% stiffness decrease after initial and 4-week MELT respectively (Table 3.3). These changes as pointed in the results were not large enough to be statistically significant. It is to be noted that the percent reduction of stiffness increased after participants received long-term MELT treatment of 4 weeks. This suggests that participants may have shown a significant reduction in stiffness if they were tested after a longer period of time in which they would have continued MELT. However, this suggestion is under speculation because the Myoton measures stiffness for all layers of soft biological tissue, in addition to connective tissue.

A sample size calculation was performed according to the subjects' mean difference and standard deviation and it was found out that at least 120 subjects in MELT group alone, would be needed to achieve a significant reduction in stiffness with a 95% confidence interval. Therefore, the absence of significant change in stiffness could be due to the fact that the target sample size had not been met.

As mentioned in the "Limitations" section, exact testing location for the Myoton between the initial treatment measurements and after 4 weeks of treatment measurements, could not be determined as the marks made on the first day did not last for 4 weeks. Hence, when the participants came back the second time to get re-measured after 4 weeks, the location of testing that had been selected 4 weeks earlier may not have exactly matched up, providing possible inconsistent results. This observation was also made by

Lam et al. when they found that myometric measurements made on the same day were more reliable than measurements made across two consecutive days (Lam et al., 2015). Mooney et al. made a similar observation where reliability of stiffness, tone and elasticity occurred when measurements were taken in the same session (Mooney et al., 2013). However, the decreasing trend in stiffness values after initial and 4-week MELT treatment does not suggest that the results obtained in this study was inconsistent. Nevertheless, in future studies, it is imperative that a method of determining exact measurement locations of each subject of the study for all measurements be established to minimize errors and inconsistencies.

The Myoton measurements showed a significant increase of 8% and 7.2% immediately after and 4 weeks after MELT respectively, in mechanical stress relaxation time on the left side in the lower area below the 12th rib. The area below the 12th rib consists of the fascia over the latissimus dorsi. It is unclear why the myofascia in this particular region would be in a relaxed state after MELT for a longer time than other areas, suggesting a new aspect of research. Moreover, significance in one site among twelve sites tested can be a result of statistical variation as opposed to the tissue being in a relaxed state for a longer period of time.

Both stiffness and mechanical stress relaxation time values showed a decreasing trend on the left side. This one-sided effect could be a result of subjects' hand dominance. All but two subjects in the MELT group were right-handed. This means that the left side of these subjects was probably stiffer due to less activity and therefore, a greater effect of change in muscles and tissues on the left side was observed. Chung et al. showed that

trunk stabilization exercises performed by subjects with chronic LBP improved their capability of weight bearing, pain and functional disorders, suggesting how balanced use of both limbs are required to maintain a healthy and active daily life (Chung et al., 2013). This article, along with other studies (Hodges and Richardson, 1999; Yang et al., 2015), also helps to explain why right handed subjects showed a significant change on their left sides.

CHAPTER 5

CONCLUSION

Correlation scatter plots showed very little correlation between reduced thickness and pain or flexibility. Therefore, it can be said that these were independent measures. While it could be theorized that rolling the back might narrow the tissues temporarily from applied pressure, this explanation would not hold 4 weeks post measure of MELT. Hence, we are left with the conclusion that MELT group subjects have reduced thickness and pain, and increased flexibility. Examination of the correlations between these 3 variables in individual data does not suggest any mechanism tying them together at the individual level.

This study is the first to report the effect of the MELT method on chronic LBP subjects by seeing changes in thoracolumbar connective tissue thickness and biomechanical and viscoelastic properties of thoracolumbar myofascia. Although, positive changes have been observed in the form of reduced connective tissue thickness, increased flexibility and reduced pain, further research needs to be done in order to validate the claim that MELT reduces chronic pain by rehydrating connective tissue and rebalancing the regulators of the nervous system.

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