A Study Investigating

The Effects of Osteopathic Muscle

Energy Technique on the

Viscoelasticity of Skeletal Muscle

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Declaration

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This Thesis/Dissertation/Research Project is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

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- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
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Abstract

This study was performed to investigate the effects of an osteopathic treatment technique (muscle energy technique) on the viscoelasticity of skeletal muscle (biceps brachii). Fifteen 18-30 year old healthy non obese right handed male volunteers participated.

Data collection was undertaken over four days with each subject attending two sessions separated by an interval of 1 day. On day one, three measurements of muscle viscoelasticity (stiffness, power of resistance) were taken from each individual participant’s left biceps brachii muscle. Measurements were made using a purpose designed force dial viscoelastometer. This device is designed to perform incremental compression of tissue and to calculate stress - strain data for muscle tissue during periods of controlled deformation.

On day two, three measurements were again taken followed by five 10 second cycles of muscle energy technique on the subject’s left biceps brachii muscle; three further measurements were again taken post intervention. Analysis of deflection and resistance of the measuring probe was then plotted as a linear equation ($y = kx + b$). The deformed muscle tissue was conceptually modelled and represented using 3 subsequent springs in series, representing 3 different compartments (layers) of skeletal muscle.

Indices of total compressive stiffness of skeletal muscle and specific power of resistance during tissue compression were calculated using multiple mathematical formulas. A comparative statistical analysis between pre-intervention and post-intervention data was performed with the single tailed paired samples $t$-test from the software program SPSS 12.0.1 for Windows.

There was no significant difference in stiffness (95% CI = -0.06419 to 0.23786 degrees; $t = 1.233; df = 14; P < 0.238$) and power of resistance (95% CI = -0.00804 to
0.01988 degrees; \( t = -0.910; \) df = 14; \( P < 0.378 \) between pre-intervention and post-intervention states. After intervention the stiffness and power of resistance of the biceps brachii muscle did not decrease. The Cohen’s \( d \) post-hoc test showed that the effect size of the intervention was considered to be small, low, minor. No significant individual difference was demonstrated in terms of the stiffness (95% CI = -0.36715 to 0.07369 degrees; \( t = -1.428; \) df = 14; \( P < 0.175 \)) and power of resistance (95% CI = -0.02503 to 0.01245 degrees; \( t = -0.719; \) df = 14; \( P < 0.484 \)) between pre-intervention (baseline) trials for each subject.

This study demonstrates that muscle energy technique did not decrease indices of viscoelasticity (stiffness and power of resistance) of the biceps brachii muscle. These findings encourage further research on the physiological background of MET.
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I

Introduction

Stiffness is a term used in many different branches of science to represent the resistance of a material to any deformation, and can be further defined as the amount of stress over strain (Gutnik et al., 2005; Hamill & Knutzen, 1995). Stiffness is quantified by the modulus of elasticity called Young’s modulus, which represents stress over strain (expressed as N/m² or Pascals) (Lieber, 1992). Stiffness of different substances has been studied in fields such as engineering, biology and biomechanics. In the study of skeletal muscle, stiffness as well as power are considered to be major distinguishing features between average and high muscle performance (Hamill & Knutzen, 1995). Muscle power is the product of force and velocity, and represents the activation of fast twitch fibres (Hamill & Knutzen, 1995).

Human locomotion and physical performance is known to be affected by the degree of stiffness and power of resistance to deformation of the skeletal muscles, and plays a role in the efficiency of motion of a segment. Human whole body movement can be influenced by gradual changes in Young’s modulus of a biological tissue over a period of time. This alteration may increase the amount of work performed and thus play a vital role in locomotion (Voigt et al., 1995).

Muscle stiffness has been regularly studied in the fields of biomechanics, using different methods of measurement and calculations. Some methods used force vectors with mathematical calculation to calculate muscle stiffness and did not measure muscle stiffness directly, these methods are not considered to be accurate in measuring muscle stiffness since stiffness is a mechanical property and not a measurable force.

Muscle energy technique (MET) is a technique used by osteopaths and other manual therapists to treat many different muscular and fascial complaints. Muscle energy technique is described as a manual therapy procedure which involves the voluntary contraction of the patient’s muscle in a precisely controlled direction at varying levels of intensity, against counter resistance applied by the operator (Greenman, 1989).
Muscle energy technique is claimed to be effective for a variety of purposes, including; lengthening of a shortened or contracted muscle, strengthening muscles, as a lymphatic or venous pump to aid the drainage of fluid or blood, and, to increase the range of motion of a joint (Ballantyne et al., 2003).

Many researchers such as Fryer et al. (2004), Leneham et al. (2003) and Ballantyne et al. (2003) have investigated the effects of MET on the range of joint motion, and all reported a significant increase in range. Although this procedure is widely used in the clinical field of many different manual therapy professions, there is a limited amount of research supporting and validating its use (Ballantyne et al., 2003).

A proposed explanation of the physiological mechanism behind the effects of MET is a change in the viscoelastic properties of muscle post-application of MET. Viscoelasticity determines the tissue’s response to load applied, which may represent a property of the elastic and viscous components (Ballantyne et al., 2003).

Researchers such as Ballantyne et al. (2003) and Lenhan et al. (2003) link the increase in muscle length following isometric contraction to the viscoelastic and plastic changes in the myofascial connective tissue elements. Furthermore, resistance of skeletal muscle is provided mainly by myofibrils, and as the muscle stretches the limit to the range of motion is attributed to the viscoelastic elements of the connective tissue.

One of the viscoelastic properties that might be responsible for the change in muscle length is muscle stiffness. Another measurable factor that could be linked to muscle stiffness is the power of mechanical resistance to deformation. Although this explanation may seem logical, there is limited evidence to justify this physiological theory, and a lack of published material in relation to MET and muscle stiffness.

The aim of this study is to investigate the effects of osteopathic muscle energy technique on the resistance of skeletal muscle, using the biceps brachii muscle as a model. An understanding of the viscoelastic response of skeletal muscle to this technique may provide an improved explanation of the physiological mechanisms behind its effect.
Because manual therapy is related to the constant deformation of muscle; this work can help in understanding some biomechanical patterns in a wide range of manipulative procedures.
II
Literature Review

2.1 Skeletal Muscle

The human body is composed of many cells that are contractile in nature. Muscle cells (myocytes) are considered to be the main contractile tissue, alongside small numbers of other cells such as myofibroblasts and myoepithelial cells (Gray, 1995). Myocytes differentiate into one of three subtypes of muscle which include skeletal, cardiac and smooth. Both skeletal and cardiac muscle are referred to as striated muscle, due to the myosin and actin filaments being organised into repeating elements that give the cell a finely cross-striated appearance under the light microscope (Gray, 1995). Skeletal muscle force and gravity are the major producers of movement in the human body, skeletal muscles function to hold a static position, raise or lower a body part, slow down a fast moving segment, and to generate great speed in the body or in an object being propelled into the air (Hamill & Knutzen, 1995). Skeletal muscles achieve large movements of joints through the amplification provided by the lever system of the skeletal system, hence their name (Gray, 1995).

Viewing the structure of skeletal muscle, each individual muscle has a more centralised portion where the muscle is thicker, termed the belly. The outer layer of the muscle is covered by fibrous tissue, the epimysium, which plays a vital role in the transfer of tension to the bony attachment via the tendon (Hamill & Knutzen, 1995). Muscles may contain thousands of muscle fibres all contained within small compartments known as fascicles, which are covered by a dense connective sheath, the perimysium. The perimysium provides protection from external force for the cluster of muscle fibres and creates pathways for nerves and blood vessels (Gray, 1995). The connective tissue in the perimysium and epimysium provides the muscle with much of its ability to stretch and return to a normal resting length (Hamill & Knutzen, 1995). The parallel, aligned fascicles contain the long cylindrical muscle fibres, which can be as large as 50µm wide and 10cm long (Billeter & Hoppeler, 1992). Covering the muscle fibres is a very fine sheath called the endomysium. This sheath carries capillaries and nerves to supply each
muscle fibre and also acts as an insulator for neurological activity within the muscle (Hamill & Knutzen, 1995). Lying directly beneath the endomysium is a thin plasma membrane surface that branches into the muscle fibre, called the *sarcolemma*. Axons of neurons supplying the muscle travel through the sarcolemma ultimately influencing each individual contractile unit through chemical neurotransmission (Gray, 1995). Within the sarcolemma hundreds of smaller *myofibrils* are tightly contained, and then further composed of parallel contractile units called *sarcomeres*, that connect to each other in series (see Fig. 2.1).

![Illustration of the organisation of skeletal muscle tissue](image)

Figure 2.1: Illustration of the organisation of skeletal muscle tissue (Hamill & Knutzen, 1995, P.73)
The sarcomere consists of thick protein filaments of *myosin*, and thin polypeptide bands of *actin*. According to the sliding filament theory, the loose myosin heads attach to the actin filaments to form cross-bridges after the release of calcium in the sarcomere. The myosin heads pull the actin filaments towards the centre line (M zone); then detach and move on to the next site. The result is shortening of the muscle and an increase in tension (Gray, 1995; Hamill & Knutzen, 1995) (see Fig. 2.2).

![Figure 2.2: The sliding filament theory (Hamill & Knutzen, 1995, P.74)]
2.2 Biceps Brachii

The muscle that was the focus of this study is the biceps brachii muscle, located in the flexor compartment of the upper arm. The name is derived from two proximal attachments (also termed heads) at the shoulder girdle. The long head of biceps starts within the capsule of the joint as a long narrow tendon, running from the supraglenoid tubercle at the apex of the glenoid cavity and the glenoid labrum. The long head tendon descends down the humerus in the intertubercular sulcus where it is securely detained by

Figure 2.3: Biceps Brachii (right arm anterior view)  
(Ross et al., 2005, P.290)
the transverse humeral ligament and fibrous expansion of the pectoralis major tendon (Gray, 1995). The short head originates from a thick flattened tendon from the coracoid process together with coracobrachialis (Gray, 1995). The two tendons connect to two elongated muscle bellies that insert into the rough posterior area of the radial tuberosity, where a bursa is present to separate the tendon from the anterior area of the tuberosity (Gray, 1995; Ross et al., 2005) (see Fig. 2.3). The vascular supply of biceps brachii is by the anterior circumflex artery (branch of the brachial artery), and the muscle is innervated by the musculocutaneous nerve that originates from the 5th and 6th cervical nerve roots. The biceps brachii has multiple functions involving the shoulder and elbow joints, including supination and flexion of the elbow joint, shoulder flexion, stabilisation of the humeral head during deltoid contraction, abduction and internal rotation of the humerus (Ross et al., 2005).

The biceps brachii is structurally defined as a fusiform muscle (Hamill & Knutzen, 1995), where the fibres are arranged in parallel and fascicles that run the length of the muscle. The fibres of the biceps run parallel to the direction of its pull, indicating the fibres direction of force is the same direction as the musculature (Hamill & Knutzen, 1995). A muscle such as the biceps brachii, which has a greater ratio of muscle fibre to tendon, has the potential of shortening a greater distance. Baechle (1994) illustrates that when biceps brachii is in stretched or resting state, it lies along the anterior surface of the humerus bone. During contraction, the bulk of the muscle belly shifts superiorly along the humerus bone, shortening up to 30 - 50% of its length (Hamill & Knutzen, 1995).

Trigger points can be found in many different areas in the body, including the biceps brachii muscle. A trigger point is defined as “A small hypersensitive site that, when stimulated, consistently produces a reflex mechanism that gives rise to referred pain and other manifestations in a consistent reference zone which is consistent from person to person” (Ward et al., 2002, p. 1253). Palpation of a hypersensitive bundle or nodule of muscle fibre of harder than normal consistency is the physical finding most often associated with a trigger point (Alvarez et al., 2002). Two trigger points lie centrally in the muscle belly of biceps brachii (Ward et al., 2002). Most of the pain
originating from biceps brachii trigger points projects to the region of the distal tendinous attachments of the muscle (Alvarez et al., 2002) (see Fig. 2.4).

![Figure 2.4: location of biceps trigger points](image)

Muscle tissue is a highly adaptable tissue which rebuilds and adapts under stress. Muscle tissue is highly responsive to load, and will contract under sufficient tension. A large amount of tension will cause stronger contraction which may eventually cause muscle fatigue. Muscle fibres later regenerate into a thicker, stronger form to satisfy the high demands. Logically, muscle tissue is linked with the size and distance between its bony attachments, the longer a bone grows the longer and larger its surrounding musculature will grow. The biceps brachii muscle will develop to its adult size once the individual is in their early twenties when the humerus bone will stop growing. Also, the muscle’s growth will be proportional to the amount of tension it experiences, its blood and nerve supply and the amount of protein intake in the diet (Hamill & Knutzen, 1995). Maximum strength development occurs around nine months to a year after the peak velocity of growth. This pattern suggests that muscle increases first in mass, followed by the ability to express strength (Baechle, 1994).
Structural variations from a multiple of perspectives occur in the biceps brachii. In 10% of the population, a third head of biceps brachii occurs, arising from the superomedia\l part of the brachialis muscle and attaching to the bicipital aponeurosis and medial side of the tendon of insertion (Gray, 1995). The general body muscle mass is much greater in males than females, due to higher amounts of testosterone production (Baechle, 1994); this is clearly observed in the biceps brachii muscle since it is a superficial and easily accessible muscle. Fatigue of muscles can also manifest differently in the two genders, where females show a higher rate of muscle fatigue than males; this phenomenon is linked directly to the amount of muscle bulk present (Albert et al., 2006). The ratio of slow-twitch / fast-twitch fibres also varies in the population; since the biceps brachii is a non-postural muscle, it will contain more fast-twitch than slow-twitch fibres (Hamill & Knutzen, 1995). The ratio also varies depending on the activity level and the type of activity the individual performs. For activities such as long distance running, slow-twitch fibres will dominate due to high endurance capacity and minimal fatigue. Otherwise in activities that require more force production such as throwing, the fast-twitch fibres will dominate the biceps brachii muscle fibre ratio, due to biceps’s high force production requirement (Baechle, 1994).

Variations also occur in relation to body morphology. Typical mesomorphs are muscular, broad shouldered, thick chested, and have a narrow waist with a minimal or moderate amount of adipose tissue (Baechle, 1994). This group of people tend to have larger biceps, hence larger arm circumference. Typical endomorphs are rounder and more pear shaped, while ectomorphs tend to be taller and late maturers (Baechle, 1994). Endomorphs may possibly have less muscle bulk in their arms and hence smaller biceps, while ectomorphs are likely to have elongated bone structure, and therefore longer biceps muscles.

The biceps brachii has been involved in many studies in a number of fields of science and sport due to the ease of its accessibility (Gennisson et al., 2005; Holcomb, 2006; Mattiello-Sverzut et al., 2003). Researchers have investigated this muscle in terms of physiological, biomechanical, anthropometric and rehabilitation properties. A study by
Mattiello-Sverzut et al. (2003) investigated the effects of aging on the biceps brachii muscle fibres via autopsies and biopsies; and revealed that aging changes were present from the sixth decade and consisted of atrophy and/or type-grouping. The size of muscle fibres was also found to gradually decrease due to the aging process (Mattiello-Sverzut et al., 2003). Holcomb (2006) investigated the effects of neuromuscular electrical stimulation (a method used for rehabilitation of immobilized muscles) on biceps brachii in comparison to isometric muscle training in post-fracture patients. The study concluded that isometric muscle training was much superior to neuromuscular electrical stimulation in terms of regaining muscle strength in the biceps brachii. The hardness of the biceps muscle was also investigated. Gennisson et al. (2005) explored a new method called transient elastography to measure the hardness of the biceps brachii muscle during incremental isometric contraction. The study concluded the method to be reliable, non-invasive and useful to investigate deep musculature affected by neuromuscular diseases.

2.3 Muscle Stiffness

The Dorland medical dictionary (2003) defines the term ‘stiffness’ as “a quality of rigidity or inflexibility”. It is a term used in many different branches of science to represent the resistance of a material to any deformation (Gutnik et al., 2005). As a physical term, stiffness of any material is defined as “the amount of stress over strain” (Hamill & Knutzen, 1995). Stiffness is measured by the modulus of elasticity called Young’s Modulus, which represents stress over strain (expressed as Newtons/meter$^3$ or Pascals/meter) (Lieber, 1992). Stress is considered as loading force that causes the deformation (tensile force / cross sectional area) and strain is the range of deformation of neutral strain (change in length / length) (Reese, 2000).

\[
\text{Tensile stress} = \frac{\text{Force (N)}}{\text{Area (m}^2\text{)}} \quad \text{Stiffness} = \frac{\text{Tensile stress}}{\text{Tensile strain}}
\]

\[
\text{Tensile strain} = \frac{\Delta \text{Length}}{\text{Length}}
\]
A higher Young’s modulus value indicates greater stiffness of the substance producing higher tensile strength of the tested substance (Reese, 2000). Stiffness, as measured by Young’s modulus also plays a role in the efficiency of motion of a segment. Human whole body movement can be influenced by gradual changes in stiffness of the tendon tissue, since this determines the amount of tendon work performed and plays a vital role in locomotion (Voigt et al., 1995).

From a physiological perspective, stiffness can be measured in any soft tissue, including skeletal muscle. Stiffness is a mechanical property that determines how effectively external forces delivered to the skeletal system are absorbed or transmitted by the articular soft tissues (Riemann et al., 2001). Tension is a mechanical property that is defined by Dorland’s medical dictionary (2003) as “the degree to which anything is stretched or strained”. Stiffness of skeletal muscle fibres is related to their experienced tension, simply because tension and stiffness are both directly proportional to the number of cross bridges between actin and myosin (Proske & Morgan, 1987).

Stiffness is also linked to muscle tone, which Dorland’s medical dictionary (2003) defines as “the resistance to passive elongation or stretch”. Pisano et al. (1996) describe muscle tone as “the resistance felt to externally imposed movement in a state of voluntary relaxation”. Mullany (2006) mentioned the classical definition of Robert Wartenberg which is commonly cited in biomechanical literature, describing muscle tone as the summation of intrinsic viscoelastic resistance, contractile and relaxation activity, and limb inertia opposing changes in joint orientation. Muscle tone increases during muscle contraction.

It is well known from palpation that muscle becomes more tonic in some physiological conditions such as voluntary contraction (Gennisson et al., 2005), and also in pathological conditions such as spasm, cramps, oedema and delayed onset muscle soreness (Gennisson et al., 2005). However, even when at rest and functionally inactive skeletal muscle is potentially elastic with some level of tone (Guyton & Hall, 2000; Gutnik & Leaver, 2006). Muscle tone is highly dependent on the intrinsic stiffness
determined by the elastic and dampening properties of the contractile elements, also the inherent elasticity of the tendon insertions and connective tissue within the muscle (Pisano et al., 1996). Fatigue may also be related to stiffness. Inaba et al. (2000) found significant differences in stiffness between trained and non-trained muscles, where conditioned muscles were much stiffer. Sustained muscle tone is considered essential for the maintenance of blood flow in resting muscle. Lederman (2005) considers rhythmic arteriolar pulsations a maintaining factor for muscle tissue perfusion during periods of inactivity.

Stiffness in resting human skeletal muscle is provided by contractile and non-contractile tissues such as series and parallel elastic elements (Herzog, 1999; Panjabi & White, 2001). Gosselin et al. (1998) claim that passive viscoelastic properties of skeletal muscle are attributed to different factors concerning collagen, including the amount of collagen, its phenotypic distribution, the extent of collagen cross-linking and the architectural organisation of the collagen fibrils. Other researchers claim that viscoelasticity is also due to factors concerning elastin, titin and extra-cellular fluid pressure (Lieber, 1992; Trotter & Purslow, 1992).

Lederman et al. (2005) also relate the mechanical behaviour of soft tissue to the overall properties of connective tissue, especially viscoelasticity. Viscoelasticity is a property that is described as a function of a composite, biological material that contains a combination of stiff and elastic fibres embedded in a gel medium, giving the tissue its unique behaviour. Viscous properties can be considered as the tissue’s dampening and lubricating element while elasticity is the spring-like element within the tissue. An important feature of elastic tissue is the ability to store elastic energy when stretched and to recoil this energy afterwards as mechanical work (Babic & Lenarcic, 2004). Viscoelasticity can be simply illustrated as a spring for the elastic component and a piston for the viscous component (see Fig. 2.5).
Viscoelasticity has been shown in various studies to have a great influence on human motor performance; determination of the viscoelastic properties is essential for analysis and modelling of human dynamics (Babic & Lenarcic, 2004).

The term skeletal muscle stiffness is directed towards the stiffness properties specifically exhibited by the tendino-muscular tissues; in contrast, the term joint stiffness encompasses contributions from all structures located within and over the joint, which may include muscles, tendon, ligaments, joint capsule and cartilage (Riemann et al., 2001). Another variation is angular stiffness, which can be explained as the rate of change of the movement of the muscle force about a joint centre, with respect to changes in the angle of oscillation (measured in Nm/rad) (Jennings & Seedhom, 1998).

Over the last decade a number of studies investigated skeletal muscle stiffness in relation to viscoelasticity (Babic & Lenarcic, 2004; Gosselin et al., 1998; Halbertsma et al., 1996; Inaba et al., 2000; Nordez et al., 2006; Riemann et al., 2001). Riemann et al. (2001) investigated the relationship between sex, joint angle and the gastrocnemius

Figure 2.5: Simple representation of the viscoelasticity model
(Lederman, 2005, P.49)
muscle on passive ankle joint complex stiffness. The study concluded that the gastrocnemius muscle contributed significantly to passive ankle joint stiffness, providing a scientific basis for clinicians incorporating stretch regimes into rehabilitation programs. Gosselin et al. (1998) investigated the effects of exercise training on passive stiffness in locomotor skeletal muscle of rats as affected by age. Findings indicated that ten weeks of endurance exercise significantly increased the passive viscoelastic properties of the soleus muscle in the older rats with a comparatively small increase in the young adult rats. These findings highlight the suggestion that the condition of collagen plays a role in influencing the passive viscoelastic properties of skeletal muscle.

Babic & Lenarcic (2004) performed an original study investigating in vivo viscoelastic properties of the triceps surae muscle tendon complex using voluntary contraction, and an active pulley system. Results showed that the soleus muscle was consistently more viscous than the gastrocnemius muscle, and different subjects also showed different levels of muscle tissue stiffness. The method used was found to be reliable and results obtained were considered applicable to other studies of human motion dynamics.

Nordez et al. (2006) looked at the acute effects of static stretching on passive stiffness of the inactive hamstring muscles. Although there were positive and negative findings observed among different groups, the main interpretation was that stretching has a significant decreasing effect on muscle stiffness. This conclusion does not comply with the finding of Halbertsma et al. (1996), who also investigated the effects of stretching on short hamstring muscles. Here the conclusion was that one session of static stretching does not influence the course of the passive muscle stiffness curve; the increased range of motion observed was related to an increase to the stretch tolerance rather than alteration to stiffness.

A study by Inaba et al. (2000) investigated using stiffness measures to detect fatigue in the latissimus dorsi muscle prior to cardiomyoplasty. Using a sophisticated tactile sensor, stiffness and tension of a canine latissimus dorsi were monitored. A
statistical difference was present between conditioned and unconditioned muscle in terms of stiffness, where conditioned muscle showed greater stiffness values; once again suggesting a relationship between stiffness and tone of the muscle. The tactile sensor system used was shown to be an efficient method for evaluating fatigue of muscle in situ without measuring muscle tension.

Over the years there has been a large number of different stiffness measuring methods, many of them have similar approaches but used different equipment. Proske & Morgan (1987) claim that there are only two valid methods by which it is possible to measure the stiffness of the whole tendon and the intramuscular portion. The $\alpha$ method used by Morgan (1977) measures the stiffness of the muscle over a certain range of isometric tension during brief constant velocity stretch. Stiffness values are used to calculate the amount of movement in muscle fibres and tendons, and are represented as two measurable springs in series. The second and more recent method is the null-point method of stiffness measurement, using the muscle spindles as monitors of muscle fibre length. This method applies small sinusoidal stretches to the muscle; from the values of the tension variation and the size of the stretches it is then possible to calculate the stiffness (Proske & Morgan, 1987).

The $\alpha$ method of Morgan (1977) was adapted by Cook & McDonagh (1996) in a study of muscle stiffness during a period of rapid isometric muscle contraction. Stress was measured on the muscles crossing the ankle joint through passive dorsiflexion of the foot. The measured force is a torque force provided by the ankle joint, therefore it is often referred to as ‘joint stiffness’ (Riemann et al., 2001) or ‘tendon stiffness’ (Cook & McDonagh, 1996) rather than muscle stiffness. This method is not considered to be specific for muscle stiffness measurement, because the stiffness was not measured in isolation from joint structures.

A study by Haji et al. (1992) used a new method of measuring muscle stiffness of the vocal fold in-vivo. Stiffness was measured by a probe that applied a deformation force at one millimetre increments compressing the vocal fold. The strain was measured
as the amount of deformation the probe imposed on the vocal fold, while stress was measured as the resultant force that the vocal fold imposed in resistance to the probe. The data of this experiment was illustrated as a stress vs. strain curve; a line of best fit was then created to represent stiffness.

This method was later adapted for measurement of skeletal muscle tissue stiffness by Gutnik et al. (2006). This study investigated human muscle stiffness of the first dorsal interosseus muscle in vivo. A probe was used to compress the first dorsal interosseus muscle where the subject’s hand was placed flat on a bench in a relaxed position. This method is considered to be more specific for measuring muscle stiffness due to the probe applying compression directly on a muscle without any resultant joint motion.

A new method used by Gennisson et al. (2005) is also considered to be specific to passive muscle stiffness. The transient elastography technique uses a shear elasticity probe that produces ultrasonic signals, which are recorded and then stored in a digitizer device. The ultrasonic pulses produced penetrate into the muscle as a shear wave, which can later be interpreted to provide Young’s modulus data using multiple mathematical equations.

Another new method which is still under testing is magnetic resonance elastography (MRE) which is a painless method of measuring the stiffness of the deepest muscle fibres by taking snapshots of the tested area with a magnetic resonance imaging (MRI) scanner (Knott, 2003). Magnetic resonance elastography works by measuring the wave-length of the vibrations sent through the muscle fibres by a vibrating metal plate placed on the skin, the magnetic field in the MRI scanner is synchronised with the mechanical vibrations. The MRE scanner then freezes the pattern of waves in the muscles, allowing the wavelength to be measured and assessed; this measure can be used to calculate the stiffness of the muscle (Knott, 2003).

Jennings & Seedhom (1998) investigated the stiffness in angular motion of the anterior cruciate ligaments of the knee, using a different approach. A force transducer
was used to measure different cycles of contractions on the knee joint; the following formula was applied to the data:

\[ F = 4\pi^2 M r^2 (d)^2 \]

Where \( F \) is the angular stiffness of the hamstring muscle, \( M \) is the total mass acting on the foot, \( r \) is the radius of the lever arm from the foot to the knee and \( d \) is the frequency of oscillation.

Another study by Zinder et al. (2005) also investigated joint angular stiffness on the ankle joint. Using a complex linear spring mass oscillator, with an inversion / eversion swaying cradle device, the transient motion oscillation of the ankle joint was measured. The method was demonstrated to be valid and can produce repeatable and consistent results. Unfortunately, such a method does not directly measure stiffness because stiffness is a mechanical property that is not measured as a force of activity.

Ultrasonography is another known method of measuring muscle stiffness in-vivo; Hansen et al. (2005) used this method to investigate the mechanical properties of human patellar tendon, later describing it as a reliable non-invasive method of measuring muscle stiffness. This method uses an ultrasound probe to produce sound waves that cause strong pulses of sound (beyond the range of a human ear) to enter the muscle. The sound waves return to the transducer to be converted to electrical pulses, which are then sent to a scanner where the data is processed and translated into digital images / data (Hansen et al., 2005).

Analysis of the literature suggests that muscle stiffness is a primary viscoelastic property that determines the mechanical behaviour of muscle tissue. Currently there are multiple methods of measuring skeletal muscle stiffness, with each having different validity and reliability standards. Although a sufficient amount of research exists in the field of biomechanics regarding muscle stiffness; only one abstract was published in an International Congress for the study of Biomechanics (Stanley et al., 2001) that measured
the effects of a manual therapy technique on skeletal muscle stiffness. The study claims that the effects of a ten minute effleurage intervention on passive muscle were measured. However, the data measured represented stress, not strain. Because strain was not measured in this study, stiffness could not have been calculated, which means that no claims can be made about the effect of effleurage massage on muscle stiffness. An unpublished thesis by Mullany (2006) adopted the same method used by Gutnik et al. (2006) to measure the viscoelastic response to pettrisage massage. The results showed no statistical difference in muscle stiffness in relation to massage and suggested further research to be conducted in this area. Other than these studies, no research has been published in regards to changes of skeletal muscle stiffness with respect to any manual therapy technique, identifying an opportunity for research in this area.

2.4 Muscle Energy Technique

Muscle energy technique (MET) is a contract-relax technique developed by osteopaths, and used in many other forms of manual therapy. Various authors have agreed that MET can be defined as a form of osteopathic manipulative treatment in which the patient’s muscles are voluntarily activated on request in a precisely controlled direction, at varying levels of intensity, against a distinctly executed counterforce applied by the operator (Greenman, 1989; Ward et al., 2003). Other authors and researchers also had similar descriptions (Chaitow, 1999; Kuchera & Kuchera, 1993; Lenehan et al., 2003). Muscle energy technique procedures have wide application and are classified as active techniques in which the patient contributes to the corrective force and is responsible for the dosage applied (Greenman, 1989). The term MET is synonymous with proprioceptive neuromuscular facilitation (PNF) (Chaitow, 1999), which is claimed to be a practice used within the physiotherapy discipline (Milliken, 2003).

Muscle energy technique can be used in a variety of clinical applications in the fields of manual therapy and sports. Kuchera & Kuchera (1993) explain that MET can be used in the treatment of individual joints, stretching muscles, activations of fluid pumps
and somatic dysfunctions. Muscle energy technique may also assist in treatment of respiratory disorders, spinal segmental somatic dysfunction, it also aids to induce muscular relaxation and in regaining muscle strength (Ward et al., 2003). Other authors claim that MET can be used to lengthen shortened musculature, decrease hypertonicity and improve range of motion of a joint as well as strengthening the muscle and improving lymphatic / venous drainage (Ballantyne et al., 2003; Fryer & Ruszlowski, 2004). Muscle energy technique is commonly used by osteopaths and manual therapists to treat specific conditions such as neck and cervical spine lesions (Fryer & Ruszlowski, 2004; Schenk et al., 1994).

Various theories have been proposed to explain the physiological function behind MET and PNF, yet the mechanism providing the effects of MET is still unclear (Chaitow, 1999). Early investigation of MET-induced muscle lengthening suggested an involvement of the Golgi tendon organ. This encapsulated sensory receptor is located in the tendons of skeletal muscle and detects changes in muscle tension. The Golgi tendon organ provides the nervous system with instantaneous information on the degree of tension in each small segment of each muscle (Guyton & Hall, 2000). This negative feedback mechanism prevents development of high amounts of tension, which protects the muscle and tendon from tearing (Guyton & Hall, 2000).

This information was applied to the observed muscle lengthening post MET; when skeletal muscle is under contraction, the Golgi tendon organ transmits an excitatory signal via type 1b afferent fibres to the dorsal horn of the spinal chord (Chaitow, 1999; Greenman, 1989; Kuchera & Kuchera, 1993; McPartland, 2002; Ward et al., 2003; Williams et al., 2004). The signal then enters an interneuron, which releases neurotransmitters (such as GABA and Glycine) (Guyton & Hall, 2000) that inhibit the activation of the anterior alpha motor neuron (see Fig. 2.6). This activity inhibits the excitatory signal reaching the muscle, ultimately forcing the muscle to relax and elongate.
Although the involvement of the Golgi tendon organ seems pivotal in the elongation of the muscle, this speculation is poorly supported by research (Ballantyne et al., 2003). Taylor et al. (1990) investigated the viscoelastic properties of muscle-tendon units in rabbit legs, and found that denervated muscle responded similarly to innervated muscle for all stretch parameters measured. The study concluded that muscle-tendon units respond viscoelastically to tensile loads such as stretching. Lederman (2005) implies that when the muscle is held in a static stretch, the overall activity of the muscle spindle decreases. Ballantyne et al. (2003) add that various studies have shown that passive stretch does not influence the electrical activity of muscles tested using electromyography (EMG). All these findings do not support the proposal of neurological involvement during MET.

Subsequent research suggests that the muscle lengthening process observed after MET / PNF results from biomechanical adaptations and not by neurological mechanisms, the viscoelastic properties of the muscle allow the stretching or lengthening process to occur (Milliken, 2003). Proprioceptive neuromuscular facilitation was found to provide
short term and long term changes within the viscoelastic components of the muscle-tendon-fascial unit (Schmitt et al., 1999). After measuring the sarcomere length changes in response to stretch, Sugi & Kobayashi (1983) proposed that the viscoelastic multisegmental nature of muscle fibres should be taken into consideration in interpreting the tension responses to quick length changes.

The lengthening process involved may be characterised as the toe region, the elastic region and the plastic region (Lederman, 2005). The toe region can be considered as the initial elongation of the tissue that accounts for 1.5-4% in total length of connective tissue, it does not involve true elastic elongation (Lederman, 2005). Once the stretch is released the structure will return to its normal wavy structure. Connective tissues such as ligaments and tendons have higher stiffness, meaning the toe range may be relatively small. As for muscles which are less stiff, the toe region may possibly be larger (Lederman, 2005).

Next is the elastic region, in which true structural elongation occurs. The overall elasticity of the tissue is highly dependent on the amount of the protein elastin present (Lederman, 2005). The more elastic elements present, the longer the elastic region will be, without failure of the collagen fibres. In connective tissue, the elastic region accounts for 2-5% of the elongated length, while in muscle this region is most likely to be longer due to the muscle being a more elastic structure (Lederman, 2005).

The plastic region is where there is progressive failure of the tissue due to microscopic tearing of the collagen. Here the tissue is stretched beyond its mechanical limit, which causes tissue changes; the tissue will not return to its original length once the stretching load is removed (Lederman, 2005). Further stretching in the plastic range will lead to progressive increase in the number of myofibrils failing, which may lead to a complete rupture of the tissue (at around 6-10% of resting tissue length). If the tissue is taken into the plastic range, its return to normal length, behaviour and tensile strength is through inflammation and tissue repair (Lederman, 2005). Microscopic failure of collagen fibres can be observed in the early stages of the elastic range (beginning at
around 3% of tissues’ resting length). Lederman (2005) suggests that for muscle tissue, the plastic region is well delayed and occurs at maximal stretch, with the majority of tissue damage occurring at the muscle-tendon junction.

An active stretching process such as MET stimulates further elongation in each successive cycle. The active contraction component of MET produces tensile forces within the target muscle resulting in elongation of the connective tissue. Passive stretch of the muscle in the relaxation phase allows the muscle to elongate further, thus both active and passive phases of MET contribute to muscle elongation (Milliken, 2003).

Therapeutic MET is divided into three different subtypes; first isometric MET, where the origin and insertion of the targeted muscle remains constant during contraction. This method is regularly used in clinical practice to treat shortened restricted muscles and also if other treatments are painful for the patient (Kuchera & Kuchera, 1993). Isotonic MET is another type. In this method, the resistance the practitioner applies is less than the patient’s resistance, therefore the muscle gradually becomes shorter. This type of MET is used to build muscle strength and endurance (Kuchera & Kuchera, 1993). Isolytic MET is the third type. In this method the practitioner’s resistance is more than the patient’s resistance, where the treated muscle elongates. This method may be used to break down adhesions and fibrosis within the muscle tissue (Kuchera & Kuchera, 1993).

Immobilised muscles lacking mechanical stimulation such as stretching or contraction have been shown to atrophy, and to have increased accumulation of connective tissue elements (Williams et al., 1988). This theory may suggest that hypertonic muscles may have increased deposition of connective tissue elements that contribute to increase in stiffness of muscle (Milliken, 2003), resulting in decreased joint mobility. Both components of MET (passive stretching and active contraction) can prevent this accumulation of connective tissue and help maintain the normal amount of muscle cells in the muscle (Williams et al., 1988).
Muscle energy technique can be applied to painful hypertonic muscle by manual therapists with an intention to increase the blood supply to the area, decrease the muscle tone, maintain connective tissue arrangement and improve the function of the affected joint. Muscle energy technique is contraindicated in patients presenting with acute injuries or excessive pain (Ward et al., 2003). It is also not recommended to be used on patients who are uncooperative, unconscious, unable or unwilling to assist or follow instructions (Kuchera & Kuchera, 1993).

Although muscle energy technique has been advocated by many clinicians as an effective method to treat many different forms of muscle lesions, the literature reporting the characteristics of MET is still minimal (Wilson et al., 2003). The majority of research conducted has investigated the effects of MET on the range of movement of joints. There is an absence of research that explores the mechanisms behind MET.

Fryer et al. (2003) investigated the effects of MET on the gross trunk range of motion; the study demonstrated significant changes after intervention, and concluded that MET is an effective method to increase the restricted range of trunk rotation and ameliorating rotational asymmetry in asymptomatic subjects. Investigating the effects of MET on cervical range of motion using a randomised controlled trial, Schenk et al. (1994) observed that the MET group demonstrated a greater increase of movement compared to the control group, and suggested that MET is an effective method to increase cervical range of motion. Ballantyne et al. (2003) examined the effects of MET on hamstring extensibility, and found that a single application of MET produced an increase in the amount of passive stretch of the hamstring muscle. The study concluded that a single application of MET produces no mechanical changes to the muscle but creates a change in tolerance to stretch.

Other researchers investigated other properties of MET. Fryer et al. (2004) investigated the influence of contraction duration in muscle energy technique applied to the atlanto-axial joint in the neck. Although various clinicians use different contraction duration, which may range between five and twenty seconds, the results failed to
demonstrate a significant benefit in the use of a longer isometric contraction when treating the upper neck with MET. In fact, using a shorter isometric contraction seemed to be more beneficial, but further investigation was recommended.

Wilson et al. (2003) performed a pilot clinical trial investigating the effects of muscle energy technique in patients with acute lower back pain. The researchers compared MET with other forms of therapy such as neuromuscular re-education and resistance training. Using the Oswestry questionnaire, which is a validated scale of pain measure (Anderson et al., 1999), the results of the study showed that MET used in combination with supervised neuromuscular re-education and resistance training exercises was superior to supervised neuromuscular re-education and resistance training exercises alone for decreasing disability and improving function in patients with lower back pain.

Research has been undertaken to investigate the characteristics of proprioceptive neuromuscular facilitation, which involves comparisons with other therapies and effects on specific properties of skeletal muscle. Burke et al. (2001) compared the effects of PNF with hot / cold water immersion on the length of the hamstring muscle in healthy subjects; the results showed no significant improvement in the hamstring length when using hot/cold water immersion in association with PNF, compared to using PNF alone. This finding suggests the use of PNF alone is effective enough to increase muscle length.

Williams et al. (2004) combined PNF with motor imagery and observed any effects on the range of motion of the hip joint in comparison to regularly prescribed physical training. The results of the study show that motor imagery combined with PNF is much more effective in enhancing and retaining range of motion of the hip joint in comparison with physical training. The researcher suggests this treatment combination can benefit both athletes and those undergoing rehabilitation.

Proprioceptive neuromuscular facilitation was also recommended by Godges et al. (2003) as an effective and immediate treatment for shoulder disorders and movement
restrictions, especially external rotation and overhead reach. Marek et al. (2005) performed a randomised cross sectional study investigating the effects of PNF on muscle strength and power output on the quadriceps femoris muscle. Proprioceptive neuromuscular facilitation caused a small deficit in the strength, power output and muscle activation, attributed to alteration in the length tension relationship and plastic deformation of connective tissue limiting the maximal force producing capabilities of the musculotendinous unit. Marek et al. suggested that practitioners need to consider the risk-to-benefit ratio when incorporating PNF in clinical practice.

The literature indicates that MET can result in elongation and a decrease in tone of the treated tissue. Understanding of the elongation process has evolved in recent years from a neurological model to a biomechanical model, demonstrating that the biomechanical elements of muscle control both the rate and the amount of stretch that occurs. The amount of research and literature regarding MET is still considered to be minimal, signifying the opportunity for further research to be undertaken.

This study aims to investigate short-term effects of MET on the viscoelasticity of skeletal muscle by measuring stress, strain and power of resistance of skeletal muscle pre- and post-intervention.
III
Method

3.1 Selection and Ethical Approach

A total of 15 subjects who were healthy young males between the ages of 18 and 30 years; with a mean age of 23.7 years ± 4.7 years were accepted in the study. Other inclusion criteria were set as right handed, reasonably fit and active but not professional athletes, a non-obese build and no current musculoskeletal pathology or injury.

Ethics approval was granted for this study by the Unitec New Zealand Research Ethics Committee. Subjects were recruited via poster advertising and word of mouth on Unitec’s Mt Albert campus, and then accepted in the study if the inclusion criteria were met.

3.2 Anthropometric Approach

Subjects of one gender were used to reduce the number of variables within the sample (Jenkins et al., 1998). There is a large variations in muscle size between males and females, due to higher amounts of the androgen testosterone in males (Marieb, 2003; Porth, 2002), which increases protein synthesis and therefore increases muscle development. Testosterone has a great effect on increasing musculature during puberty, with boys averaging an approximately 50% increase in muscle mass compared with girls (Porth, 2002).

The lower limit of the age range was set to 18 to exclude individuals without legal ability to give consent (Ward, 1997). Additionally, because puberty in males is complete by the age of 14 to 16 years (Marieb, 2001), setting the lower limit to 18 years guarantees avoiding any muscle tissue that has not matured to its adult size. The upper limit was set
at 30 to exclude any major changes in collagen related to age, which may affect the viscoelastic properties of the tested muscle (Gosselin et al., 1998). This criterion was applied because there is evidence that skeletal muscle tissue loses size, strength performance and peak power from young adult levels, mainly caused by a decrease in the rate of protein synthesis (Baechle, 1994).

The reason for subjects to be moderately fit and active but not professional athletes is to produce a population sample that will have similar stiffness, tone and force values. In addition, this criterion will exclude any individuals with atrophied muscles, and individuals with highly trained muscles, that may produce high stiffness and tone values (Inaba et al., 2000). Moderate-build individuals were required in order to exclude subjects with high amounts of adipose tissue. Excess amount of adipose tissue on the surface of the tested muscle might cause the testing apparatus to produce inaccurate measurement, regardless of its high sensitivity. An acceptable maximal figure was determined to be level six on the endomorphic scale of the Heath Carter somatotype form (Kreighbaum & Barthels, 1996; Ross & Marfell-Jones, 1991).

The aim of this study was to investigate stiffness changes in healthy individuals, therefore subjects with musculoskeletal pathologies were excluded. Furthermore, certain musculoskeletal pathologies such as inflammatory arthritis, seronegative spondyloarthopathies and polymyalgia rheumatica may cause an increase in the tone of the muscle; or other pathologies such as fibromyalgia, chronic fatigue syndrome and osteoarthritis which may cause muscle fatigue (Clark & Kumar, 2005; Farber & Rubin, 1999). If such subjects participated, the stiffness data could be highly individualised and the results of the study may be skewed.

Arguably, 2% to 30% of any human population is left-handed or ambidextrous, with most estimates approximating around 10%, depending upon the criteria used to assess handedness (Holder, 1992). The non dominant arm was tested in this study to avoid testing the trained arm, which may exhibit higher muscle stiffness (Inaba et al., 2000). Avoiding left handed individuals will prevent the presence of ambidextrous
subjects, avoiding the testing of highly trained muscle. Therefore all of the participating subjects were preferred to be right handed. Handedness was established by questionnaire (Oldfield, 1971).

3.3 Biomechanical Approach

In contrast to the methods described by Morgan (1987), where stiffness of the muscle was measured during a short period of muscle contraction; the method of measurement in this study is the same as that of Gutnik & Leaver (2006) (see Fig. 3.1), where the stiffness of a completely relaxed muscle was measured in vivo by means of

![Figure 3.1: Schematic of Viscoelastometer (Gutnik & Leaver, 2006)](image-url)
direct compression, using the biceps brachii muscle. A custom-built force dial viscoelastometer encompassing a flat circular stylus of diameter 3.5mm and a very sensitive monitoring sensor (sensitivity of 0.001 N) (Gutnik & Leaver, 2006) was used. A signal amplifier and computer containing custom-made software specific to the apparatus (Gutnik & Leaver, 2006) was connected to the viscoelastometer. The sensor mechanism is designed to compress the tested soft tissue progressively, the transducer within the sensor measures the levels of depression and resistive forces at distance intervals of 0.05mm ± 0.0005 mm, the rate of deflection is constant at 1.0 mm/s (Gutnik & Leaver, 2006) (see Fig. 3.2). The outline of this method has been previously documented and the apparatus has been validated (Gutnik & Leaver, 2006).

The experiment was performed on two consecutive days. On the first day each subject was supplied with a consent form and an information sheet explaining the experimental protocol. Each subject then completed a handedness questionnaire (Oldfield, 1971) to ensure that they were right handed. Further data were gathered by the
researcher to generate specific measures for each subject. Collected data included date of birth (to ensure subjects were between 18 and 30 years of age); height and weight, using a mechanical flat medical scale (SECA 762 accurate to ± 0.25kg) and a wall-mounted stadiometer to calculate the body mass index as a secondary measure of somatotype (see Fig. 3.3).

Figure 3.3: A: measuring weight
B: measuring height
Skin folds were also measured from the triceps muscle, medial aspect of the calf muscle and medial border of the scapula (to calculate the endomorphy level using the Heath Carter somatotype form) (see Fig. 3.4).

![Figure 3.4: measurement of skin fold using measuring clippers;](image)

- A: medial border of scapula
- B: medial skin fold of calf in passive plantar-flexed position
- C: triceps skin

The circumference of the wrist and the elbow joint were bilaterally measured using a measuring tape to ensure absence of any significant difference in size between the two limbs, thus ruling out any major deformities. Finally, the bicipital skin fold for each subject’s left arm was measured in 3 locations. A straight line was measured from the acromion process of the shoulder to the centre of the cubital fossa to represent the length of the biceps brachii muscle. From this line three points were trisected (25%, 50%, 75% of length of biceps) and skin fold measurements were taken from each point three times to reduce measurement error. The average skin fold of the biceps muscle was later calculated (see Fig. 3.5). Pre-measurement data for all subjects can be viewed in Tables 8 and 9 in the appendices section.
Each subject then lay on a flat cushioned bench in a supine position; added pillows were placed under the subjects’ heads to ensure comfort. The subject’s left (non-dominant) arm was positioned at 90° abduction, and was securely clamped to avoid forearm pronation using a custom made clamp composed of a flat cushioned surface and a thick nylon belt (see Fig. 3.6).

After the experimental protocol was explained clearly to the subject, the sensor was initially lowered to a point at which the subject first experienced the sensation of
touch. The sensor was positioned directly above the 50% point of the biceps (the point previously measured and marked). The viscoelastometer was then activated and the sensor was progressively lowered to compress the tissue at a rate of 1.0 mm/s (see Fig. 3.7). The resistive force of the muscle (stress) was measured by the sensor at each cumulative increment of 0.05mm (strain). Data was recorded using a specially designed software program (Gutnik & Leaver, 2006). The subjects were instructed to press a safety button to indicate when the compression sensation became uncomfortable. This button stopped the procedure immediately, and withdrew the elastometer’s sensor back to the starting position. The subjects did not experience any pain throughout the procedure. This process was repeated three times during the first day. The collected data was stored in the computer and was illustrated as a stress-vs.-strain line graph.

On the second day of data collection, subjects were re-measured at the same time as the previous day. Subjects were again positioned in a supine position with the left arm abducted at 90˚, and three stiffness measurements were taken from the biceps muscle on the 50% measurement point. Each subject was then seated upright on the edge of the bench. At this stage, five cycles of muscle energy technique were applied to the left biceps brachii muscle. The technique was referenced from Chaitow (1999), where the arm was held in extension and internal rotation (see Fig. 3.8), which is regarded as the best position to achieve maximum stretch of the biceps muscle (Dally & Moore, 1999).
The muscle energy technique was performed by a qualified and registered New Zealand osteopath, with over 10 years of teaching and clinical experience. The osteopath clearly instructed each subject on the muscle energy procedure and guided them through the correct direction of resistance. Five contractions of MET were performed using 15 – 20% of the subject’s counter force, where the subject was attempting to relocate the stretched arm back to the neutral position. The osteopath resisting the force was preventing the shortening of the biceps muscle, marking this technique as an isometric muscle energy technique (Kuchera & Kuchera, 1993). Using a stopwatch, each MET contraction was timed to be 10 seconds in duration, followed by a 10 second lag period when the muscle was initially kept at its starting length, then stretched further to its new barrier of restriction (see Fig. 3.9).
After the MET procedure finished, the subject was then positioned back into the supine position with the left arm abducted at 90°, the arm was securely clamped again and measurements were taken from the same point of measurement on the biceps. The time taken between the end of the MET procedure and post-intervention measurement was controlled to be less than three minutes. The same measuring protocol was repeated three times and resultant data was saved in the computer.

The next stage was plotting the data into a stress vs. strain graph; the graphs were then be further analysed using a number of mathematical formulas, and the ANOVA statistical method was used to determine any changes in data after applying MET.
3.4 Primary Data Analysis

Primary data analysis was performed using the software program Microsoft Excel® 2003 edition. The values of $x$ (deflection in mm) and $y$ (resistance in Newtons) were transformed from the storage device of the viscoelastometer into the Microsoft Excel® program. The numbers of values ranged from 180 to 400 in each individual trial. The Excel® software plotted the data of each individual into a force vs. deflection graph, (see Fig. 3.10).

![Figure 3.10: Example of a force vs. deflection graph, plotted data represented in a line graph](image)

The bicipital skin fold for each subject was subtracted from the graph for each trial (see Fig. 3.11).

![Figure 3.11: Area of stress vs. strain graph that represents skinfold (L)](image)
In Fig. 3.11, (L) is the depth of skin on the biceps muscle measured in mm, this amount obtained by halving the average skin fold (measured earlier) of each individual. The results for the muscle were obtained from the plot after removal of section ‘OA’ which represents skinfold. The remaining part of the deflection “A₁, D₁” represents the strain on the skeletal muscle tissue; the section “AD” of the plot was divided into 3 equal segments of strain along the x axis, they were AB, BC and CD (see Fig. 3.12).

A trend line was added to each of the three segments on the initial plot using Microsoft Excel® software. In this case the three plotted segmental portions were transformed into a linear plot, making the segments AB, BC and CD appear as three consequent lines (see Fig. 3.13).
These lines was then used for further analysis, where in each case the coefficient of similarity of the initial segment with its linear view ($R^2$) was calculated. This coefficient shows how similar the data is to a straight line and is necessary for creation of primary regression equations of the plotted data (Thomas et al., 2005). The software program Microsoft Excel® enabled the presentation of the linear equation with the standard formula:

$$y = kx + b$$

Where $k$ is the stiffness coefficient, represented by the inclination of the line and $b$ is a constant representing projection of the segment to the $y$ axis (see Fig. 3.14).

![Figure 3.14: Example of linear plot with added equation and $R^2$ value for each segment](image)

In each case the $R^2$ value was very close to 1, suggesting that the linear regression equation is very similar to the curved portion of the segment of the initial plot. An $R^2$ value greater that 0.950 was determined to effectively represent a straight line. Segments with $R^2$ values less than 0.950 were not included in any further analysis.

In physical terms, the coefficient $k$ in the linear equation ($y = kx + b$) is proportional to Young’s modulus, and is expressed in N/mm$^2$. For example, from Fig.
3.14, the segment AB on the plot has a $k$ value of 0.5225 N/mm$^2$, or 522.5 kN/m$^2$ and the segment BC on the plot has a $k$ value of 872.5 kN/m$^2$.

The deformed muscle tissue that represents the three segments on the plot also represents specific muscle compartments. The three compartments may be conceptualised and biomechanically modelled as three subsequent springs, each spring is therefore a model (analogue) of stiffness of its related muscle compartment (see Fig. 3.15). While this model is biomechanically accepted (Dukkipati, 2005), a possible critique of the overall method is the lack of anatomical specification of the depth and boundaries of each of the three muscle compartments. A specialised tissue imaging technique such as MRI or ultrasound could be used to produce this type data. This data could then be assimilated into the overall model to enhance specification and reduce variables.

![Figure 3.15: Illustration of the three subsequent springs in relation to three different layers of skeletal muscle](image)

The three springs are $k_{AB}$ (superficial), $k_{BC}$ (middle) and $k_{CD}$ (profound). According to Dukkipati (2005), the total stiffness of compressed muscle can be calculated as:
\[ k_{\text{total}} = k_{AB} + k_{BC} + k_{CD} \]

Applying this formula to our previous example:

\[
k_{\text{total}} = 522.5 + 872.5 + 1197
\]
\[ k_{\text{total}} = 2529 \text{ N} / \text{m} \]

The average force (\(F_{\text{average}}\) measured in N) of resistance of muscle fibres against the measuring probe in each trial was calculated using the formula:

\[
F_{\text{average}} = \frac{1}{n} \sum_{i=1}^{n} F_i
\]

The average power of resistance was also calculated, this index is the product of force of resistance of muscle fibres and the velocity of deflection of the probe:

\[
P = F_{\text{average}} \cdot V \cdot 1000
\]

Where \(P\) is the power of force of resistance, expressed in Watts, \(F_{\text{average}}\) is the average force of resistance over the trial, and \(V\) is the velocity of deflection, which was constant at 0.001 m.s\(^{-1}\).

The specific power of resistance (expressed in Watts / m) was then calculated as:

\[
P_{\text{specific}} = \frac{P}{d}
\]

Where \(P\) is the power of force of resistance, and \(d\) is the total deflection of the measuring probe in m.
Thus in this study, two measuring indices were used to analyse the results; $K_{\text{total}}$ (total stiffness of skeletal muscle tissue in compression), and $P_{\text{specific}}$ (specific power of resistance during compression).

3.5 Statistical Analysis

Statistical analysis of the experimental data was performed using the single tailed paired samples $t$-test method from the software program SPSS 12.0.1 for Windows®. The single tailed paired samples $t$-test is an extension of the dependent $t$-test, which assumes that the difference between two means lies in one direction only (Thomas et al., 2005). The $t$-test is usually a repeated measures design; the same subjects are measured before and after an intervention to compare the differences between pairs of scores. The use of this method was considered to be appropriate for the design of this study (Hopkins, 2000; Lang, 1997). Statistical significance was set at the alpha $< 0.05$ level and pre-post effect sizes (Cohen's $d$) were calculated.
IV
Results

After completing the group statistical analysis, a number of outcomes were analysed from the results:

1) There was no significant individual difference in terms of stiffness data for each subject between pre-intervention trials 1, 2, 3, and trials 4, 5, 6 (95% CI = -0.36715 to 0.07369 N/mm²; single tailed paired samples t-test, $t = -1.428$; df = 14; $P = 0.175$).

2) There was no significant difference in terms of stiffness data between pre-intervention and post-intervention (95% CI = -0.06419 to 0.23786 N/mm²; single tailed paired samples t-test, $t = 1.233$; df = 14; $P = 0.238$), stiffness of the biceps muscle did not change after intervention.

3) There was no significant difference in specific power of resistance data between pre-intervention trials 1, 2, 3, and trials 4, 5, 6 (95% CI = -0.02503 to 0.01245 Watts/m; single tailed paired samples t-test, $t = -0.719$; df = 14; $P = 0.484$).

4) There was no significant individual difference between subjects in terms of specific power of resistance data between pre-intervention and post-intervention trials (95% CI = -0.00804 to 0.01988 Watts/m; single tailed paired samples t-test, $t = -0.910$; df = 14; $P = 0.378$). The power of resistance did not change post-intervention.

Total stiffness ($k_{\text{total}}$) showed a small decrease after MET; the mean group total stiffness before intervention was 1.8793 N/mm² and after intervention was 1.7925 N/mm² ($t = 1.233$; $P = 0.238$). The specific power of resistance of the biceps brachii during compression was also marginally reduced after MET; before intervention the mean of specific power of resistance was 0.1919 Watts/m, which decreased after applying the
intervention to 0.1859 wWatts/m ($t = -0.910; P = 0.378$). Although a small decrease was observed in the values of both measurable indices, the statistical analysis showed that no significant changes were observed after the intervention was applied; also the post-hoc test showed that the effect sizes of all the comparisons were considered to be small (Hopkins, 2000). Figures 7.1 – 7.4 in the appendices section shows the steps of the statistical analysis that were used to analyse the data. Figures 7.5 – 7.8 in the appendices section shows steps of the post-hoc test used to find the effect sizes.

The average group changes in stiffness and specific power of resistance values are displayed in Table 1. Individual values for the average figures of each subject for all the 9 trials performed can be viewed in the appendices section (Tables 2 - 7). Tables 2 - 4 show values for stiffness (N/m) and Tables 5 - 7 show values for specific power of resistance (Watts/m).

<table>
<thead>
<tr>
<th>Stiffness</th>
<th>Average stiffness values (N/mm²)</th>
<th>$t$-test value</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>1.8793</td>
<td>1.233</td>
<td>0.238</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>1.7925</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Power of resistance</th>
<th>Average resistance values (Watts/m)</th>
<th>$t$-test value</th>
<th>$P$ value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-intervention</td>
<td>0.1919</td>
<td>-0.91</td>
<td>0.378</td>
</tr>
<tr>
<td>Post-intervention</td>
<td>0.1859</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
V
Discussion

The topic chosen for this study is one that has been previously poorly researched, a minimal number of studies investigated passive muscle stiffness in relation to an intervention, or MET in relation to skeletal muscle viscoelasticity. Difficulty was experienced in locating articles/references that contained information relevant to the aim. This apparent lack of study in the field provided an opportunity to conduct research that incorporated skeletal muscle stiffness with a manual therapy technique. A possible reason for the minimal prior research could be that MET is a clinical procedure and the majority of the studies investigated its clinical effects, and not the physiological/biomechanical effects. Another reason could be that measuring muscle stiffness is a relatively new concept in bioscience; the majority of research investigates the validity of the measuring device used, or stiffness changes using animals’ muscle tissue, but not the effects of any clinical procedure on human subjects.

The results of this study showed no significant changes in the viscoelasticity of the biceps brachii muscle after application of MET. For both of the measured indices (total stiffness and specific power of resistance), no significant statistical differences were observed (P= 0.238 and P= 0.378 respectively) after the intervention was applied. The average stiffness figure decreased from 1.8793 N/mm to 1.7925 N/mm post-intervention, while the specific power of resistance of the biceps brachii during compression also reduced after MET from 0.1919 W.m⁻¹ to 0.1859 W.m⁻¹. Although as a group a decrease in both figures was noticed, the results of the statistical analysis showed the decrease to be insignificant, and the post hoc test also demonstrated that the effect of the intervention on the subjects was minor.

Looking at individual subjects results, nine subjects showed a decrease in muscle stiffness after MET, and ten subjects showed a decrease in specific power of resistance values post MET. The remaining subjects showed an increase in both of the measured indices after the application of MET. Various reasons may explain these observations.
When MET was applied to the biceps brachii, voluntary contraction of the patient’s arm increased blood flow to the activated skeletal muscle. The musculoskeletal system receives approximately 20% of the cardiac output during rest, during a high contraction state such as exercise, almost all the increase in cardiac output flushes into skeletal muscles (Herzog, 2000; Leber, 2002). In fact total blood flow to skeletal muscle can increase from 1200 mL/minute to 12500 mL /minute during exercise (Marieb, 2001).

Lederman (2005) suggests that changes in the rate of blood flow to muscles are an immediate adaptation to the increased metabolic activity of the contracting muscle. In this case the primary pathway of arterial perfusion to the biceps brachii muscle is via distributional branches of the brachial artery and deep brachial artery. The distal third of the muscle however may have variable blood supply, but is usually via penetrating branches of the superior and inferior ulnar collateral arteries (Gray, 1995). Rhythmical muscle contraction such as MET will increase blood and lymph flow rate, and rhythmical muscle contraction is the most potent method of stimulating blood flow to skeletal muscle (Lederman, 2005). Other authors such as Kuchera & Kuchera (1993) and Ward (2003) claim that MET can be a very effective method to pump fluid (blood, lymph) into the affected areas such as hypertonic muscles.

During the contraction phase of MET, the blood vessels within the muscle are partially collapsed as the muscle is deformed by compression encouraging venous flow but partially reducing arterial flow (Lederman, 2005). This contention can be further explained in terms of arterial blood vessels being deep in the muscle and venous blood vessels being more superficial. Contraction will increase the pressure on the deep arterial vessels which may (depending on contraction intensity) become occluded; this increase in pressure may possibly pump out venous blood via the superficial veins. During relaxation, the decompression of blood vessels allows resumption of arterial blood flow and flow may even increase as a result of arterial-venous pressure gradient and dilatation of capillaries within the muscle (See Fig. 5.1).
The outer layer of skeletal muscle is covered by the thick fibrous epimysium, which maintains the organised muscle structure by encapsulating the muscle, and plays a vital role in the transfer of tension to the bony attachment via the tendon (Hamill & Knutzen, 1995). When the muscle’s blood supply increases, the total amount of fluid within the epimysium-covered muscle will increase. Thus the tested muscle will contain more fluid (blood and interstitial fluid), and the increase of interstitial fluid surrounding the muscle fibres can enable the muscle to become more deformable under compression, due to the interstitial fluid moving to other areas of the muscle that are not under compression (see Fig. 5.2). The testing probe of the viscoelastometer was therefore compressing a structure that was easier to deform and provided less resistance to the probe; this reason may explain why stiffness data decreased in some subjects after five consecutive cycles of MET.
Although the intensity of muscle contraction during the intervention was low (around 20% of subject’s maximal force); it can be predicted that slow twitch fibres (type I) were contracting and intermediate fibres (type IIa) were also possibly minimally involved (Saltin, 1981). These tensioned fibres would attract a significant amount of blood to the muscle to enable these changes in perfusion rate (Lederman, 2005). This contention further supports the above explanation regarding the influence of fluid volume on muscle stiffness.

Even when muscles are at rest, a certain amount of muscle tone usually remains. Since skeletal muscle fibres, with the exception of certain pathological conditions, require an action potential to initiate contraction, skeletal muscle tone results entirely from nerve impulses coming from the spinal cord (Guyton & Hall, 2000). These fibres in turn are controlled partly by impulses transmitted from the brain to the appropriate anterior motoneurons and partly by impulses that originate in muscle spindles located in the muscle itself (Guyton & Hall, 1996). An explanation for the changes in muscle length and palpable tone post MET seen in other studies and clinical practice, may be the theory
supported by multiple authors (Chaitow, 1999; Greenman, 1989; Kuchera & Kuchera, 1993; McPartland, 2002; Ward et al., 2003; Williams et al., 2004) regarding the inhibition of the Golgi tendon organ that alters muscle tone. This theory suggests that as a protective mechanism for skeletal muscle against sudden forceful muscle length change (Guyton & Hall, 1996), the Golgi tendon organ transmits an excitatory signal to the dorsal horn of the spinal chord and to an interneuron. This signal then inhibits the activation of the anterior alpha motor neuron, inhibiting the excitatory signal reaching the muscle, ultimately forcing the muscle to relax. Again if muscle is relaxed, the passive tone decreases and the muscle will be more compliant to stretching. The theory linking the Golgi tendon organ with muscle length changes post MET is poorly supported by research (Ballantyne et al., 2003) and further study would be needed to validate its plausibility.

The above explanations can also be linked to the decreased specific power of resistance figures seen in some subjects; Ward (2003) explains that MET can decrease hypertonicity in skeletal muscle. The increase of the blood supply to the muscle and the relaxation due to the Golgi tendon organ inhibition both cause a decrease in passive muscle tone and make the muscle softer to palpate. The power of resistance is the product of force of resistance of muscle fibres and the velocity of deflection of the probe. Since the measuring probe was lowered at a constant rate, force of resistance of the muscle fibres towards the measuring probe decreases, which may explain the drop in the recorded specific power of resistance value. The reduction observed in some subjects matches the findings of Marek et al. (2005). In their study a decrease in muscle power was also noticed after applying PNF, apparently caused by muscle fatigue.

Post-intervention increases in the measurable indices observed in some subjects may be reflective of the individual’s level of hydration. During dehydration the total blood volume within the body will decrease (Marieb, 2001), which may decrease the total amount of blood and interstitial fluid within the muscle after contraction. Physical exercise while dehydrated has been shown to exacerbate skeletal muscle damage, leading to structural, contractile, and enzymatic protein denaturation (Cleary et al., 2005; Cleary
et al., 2006). There is a possibility that some subjects were dehydrated. If this was the case, the measuring probe would have been compressing muscle tissue that was less deformable to compression.

Another explanation for the increase in viscoelasticity is neural excitation by the sympathetic nervous system. Xanathines such as caffeine cause an excitation of the sympathetic nervous system, which increases muscle tone (Guyton & Hall, 2000; Page et al., 2002). Since this aspect was not controlled, the results of some subjects may have been influenced by prior consumption of foods or beverages containing xanathines. Caffeine is known to have a diuretic effect (Page et al., 2002), which may also decrease the total amount of body fluid and further reduce muscle deformation by compression.

There is also a possibility that the measuring stylus was in contact with a trigger point on the biceps brachii muscle. If this was the case compression from the stylus could have caused a small degree of discomfort which elicited a protective muscle contraction, increasing the measured stiffness values. Mullany (2006) related post intervention stiffness increases to discomfort from the measuring probe that increased with subsequent measurements. The point of application of the compressive stylus on subjects arms were reddened and tender during and following experimentation. It is possible that soft tissue inflammation resulted in transient acute inflammatory exudate local to the area of compression, and the increase in extra-cellular pressure provided by inflammation may have contributed to increased compressive stiffness (Mullany, 2006). This idea however is speculative, and the author suggested future studies using a compressive stylus that reduce soft tissue irritation.

Being a clinical procedure, it is important to investigate MET in order to understand the magnitude of its effect, safety and utility. This knowledge can enable a practitioner to decide whether MET is a useful technique to be applied on a patient. Previous studies that investigated MET focused on its effects on joint range of movement (Lenehan et al., 2003; Ballantyne et al., 2003). The results of such studies found an increase in joint range of motion and recognised MET to be an effective technique in the
treatment of shortened muscles. Burke et al. (2001) compared the effects of PNF to hot/cold water immersion on muscle length, and found no difference between the two treatments.

Other studies such as Fryer & Ruszkowski (2004) investigated the influence of the contraction duration of MET on the gained increase in range of motion, and failed to demonstrate any significant benefit in using longer contraction durations. Marek et al. (2005) investigated the effects of PNF on muscle strength and power output, the results however indicated a decrease in strength and power output after MET was applied, suggesting the need for the practitioner to consider the risk-benefit ratio when incorporating PNF into a treatment.

Although no previous published study could be sourced that used a similar aim and method to be compared with the results of this study; the statistical insignificance in changes to stiffness and specific power of resistance values post MET indicates that the tested muscle did not experience changes in its viscoelastic properties. These results are similar to that of Mullany (2006), where no statistical difference in stiffness data was achieved after five minutes of pettrisage massage. The results of this present study support the view of multiple authors (Ballantyne et al. (2003); Chaitow, 1999; Greenman, 1989; Kuchera & Kuchera, 1993; Lenehan et al. (2003); McPartland, 2002; Ward et al., 2003; Williams et al., 2004) that the changes of muscle tone and length post MET are linked to the inhibition of the Golgi tendon organ, which explains the increase in joint range of motion observed in previous studies and clinical practice.

The results of this study showed a tendency to support the theories mentioned in the literature review, which suggest that the treating therapist can use MET on different types of symptomatic patients with stiff muscles to decrease tone. Since more subjects showed a decrease in viscoelasticity figures after MET was applied, it can be argued that there is a tendency for a decrease in the indices of viscoelasticity post-intervention, and demonstration of this effect may be possible by using a larger sample size.
As a physical therapist, it is important to know the characteristics of the affected tissue in order to assign appropriate treatment. Mechanical properties of muscle tissue such as stiffness can have a significant effect on homeostasis and function. The majority of previous literature investigates the validity of the measuring device and not the changes of muscle stiffness to an applied intervention. Other previous studies investigated changes in muscle stiffness due to physiological or pathological tissue changes. No previously published studies could be sourced that investigated stiffness changes with respect to a manual therapy intervention.

Haji et al. (1992) found different levels of stiffness in the vocal cords altering their vibration and movement during phonation and therefore determining different voice tones. Morgan & Proske (1987) found a significant role of tendon stiffness in movement control, where the tendon becomes much stiffer during muscle tension. Gennisson et al. (2005) found increases of stiffness during muscle contraction using a non-invasive method. The study by Inaba et al. (2000) measured muscle stiffness in order to evaluate the fatigue resistance of skeletal muscle, and found a strong correlation between fatigue resistance and stiffness. The present study recorded no statistical changes in the viscoelasticity of the biceps brachii muscle after application of MET. This finding may aid the treating physician in justifying their clinical thinking towards neural inhibition of the muscle and tissue changes post treatment. Because only one previous study using a similar aim and method could be sourced with which to compare the results of this study, the opportunity remains to conduct further study regarding changes in muscle stiffness and manual therapy.

The initial reason for choosing the biceps brachii muscle was because it is an easily accessible muscle, and the ease of experimental measurement proved the utility of this criterion. Similar experimentation could be repeated on other major muscles or muscle groups, using different positions to apply MET to further explore the applicability of this technique.
Variation in sample data showed that there is a large individual difference in terms of stiffness and specific power of resistance data between participants in both pre-intervention and post-intervention trials. Although initial exclusion criteria were set to minimise variations in subjects; this finding shows the difficulty of selecting a sample size with similar qualities such as anthropometry, body morphology and activity level, all of which may affect the data of the measured indexes. There was no significant difference between the pre-intervention trials (1, 2, 3, 4, 5 and 6) of each subject, which suggests that there was no difference in the resting muscle state or the experimental setup between the two days of experimentation. These results also suggest the testing apparatus was reliable in measuring the resting stiffness and specific power of resistance values.

Although some subjects showed large changes between the two pre-intervention trials, the majority of subjects showed little change in both stiffness and power of resistance values. It is possible that the measuring probe may cause a small degree of irritation to the local muscle fibres, causing minor local inflammation near the measuring point. This occurrence may vary depending on the condition of the muscle and on the discomfort threshold of different subjects. Large changes in data between the two pre-intervention trials may be related to the lack of time for the muscle fibres to recover from compression. A suggestion for future research is to increase the number of pre-intervention trials for a more adequate baseline comparison, and to increase the time period in between successive trials to reduce the effect of micro-irritation in the muscle due to the descending probe. The data recorded in this study was adequate to allow statistical exploration. Since each of the 15 subjects was measured three times on each of three separate occasions, a large amount of data was available for analysis.

After the completion of this study, it was obvious that further research would be required to investigate other variations regarding the effects of manual therapy on the viscoelasticity of skeletal muscle. Athletes trained in different sports could also be investigated, which would give the opportunity for a comparison of results between different targeted populations. In this example the level of caffeine and other neural stimulators can be controlled to avoid any alterations to viscoelasticity. More research
can also be conducted to investigate the difference in results between stiffness levels of males vs. females, and whether MET can exert similar or different effects on different genders.

During this study, only the short term effects of MET on skeletal muscle were observed, the approach was to investigate the immediate effects of MET on skeletal muscle viscoelasticity, this aspect was well controlled since all subjects post-intervention measurements were taken less than three minutes after the end of the intervention. A suggestion would be to investigate the long term effects of MET on the stiffness of skeletal muscle. Another opportunity for study would be to investigate the effects of MET on skeletal muscle viscoelasticity in elderly subjects, to provide comparative information to that of the younger subjects used in this study. The method of this study can also be used to investigate the effects of other myofascial and manual therapy techniques on skeletal muscle viscoelasticity.

This study also investigated the effects of MET only on the biceps brachii muscle, further research may look at different muscles/muscle groups and compare results with this study. Another approach can be taken to measure the resting stiffness of skeletal muscle; a longitudinal study can be performed to measure stiffness changes over a prolonged period of time in relations to rest, normal daily physical activity and exercise. Changes in muscle stiffness can also be investigated in relation to the individual’s level of hydration, and whether the results will differ in comparison with the results of this study.

Finally, an interesting approach could be the investigation of the effects of MET on symptomatic patients. Patients who may exhibit an increase in the tone of the skeletal muscle as typified by those with musculoskeletal conditions such as inflammatory arthropathies, seronegative spondyloarthropathies and polymyalgia rheumatica could be investigated. Pathologies that may cause muscle fatigue such as fibromyalgia, chronic fatigue syndrome and osteoarthritis can also be investigated in relation to MET.
VI
Conclusion

Within the scope of this study, it can be concluded that muscle energy technique was not shown to be effective to decrease indices of viscoelasticity (stiffness and specific power of resistance) of the biceps brachii muscle. It is possible that these findings may also be applied to other skeletal muscles or muscle groups. The apparatus used in this study is considered to be reliable and to produce consistent results for measuring viscoelasticity data of skeletal muscle, and can be used in further research on different muscles or muscle groups with the recommendation of increasing the time period in between each consecutive trial.

The small relative decrease in the measured indices observed in the majority of subjects may be linked to the increased amount of perfusion towards the muscle post MET, or possibly the inhibition of the Golgi tendon organ of the tested muscle. Future research could validate these theories and further investigate the physiological mechanisms behind MET.
References


Wartenberg lecture, Neurology, 30: 1303-1313

guidelines for authors, editors and reviewers, Philadelphia, United States,
American Collage of Physicians.

Basis of Rehabilitation, Philadelphia, United States, Lippincott Williams and
Wilkins:

Churchill Livingstone.

facilitation on balance and mobility performance of individuals with chronic

technique on gross trunk range of motion. Journal of Osteopathic Medicine,

Philadelphia: Lippincott Williams & Wilkins.

Marek, S. M., Cramer, J. T., Fincher, A. L., Massey, L. L., Dangelmaier, S. M.,
and proprioceptive neuromuscular facilitation stretching on muscle strength and


Appendices
The Effect of Muscle Energy Technique on the Viscoelasticity of Skeletal Muscle

Consent Form

This research is being undertaken by Ghassan Yagot Al-Araji from Unitec New Zealand, and will be supervised by Associate Professor Boris Gutnik and Dr Andrew Stewart.

Name of Participant: ...........................................................................................................

I have seen the Information Sheet dated 01/02/2006 for people taking part in the research project that is investigating viscoelasticity response of Biceps Brachii muscle to Muscle Energy Technique. I have had the opportunity to read the contents of the information sheet and to discuss the project with the project team and I am satisfied with the explanations I have been given. I agree that raw data from this Research project can be held indefinitely for the purposes of future analysis and research. I understand that taking part in this project is voluntary (my choice) and that I may withdraw from the project if necessary.

I understand that I can withdraw from the project at any time, for any reason, within two weeks of the final data collection.

I understand that my participation in this project is confidential and that no material that could identify me will be used in any reports on this project.

I have had enough time to consider whether I want to take part.

I know whom to contact if I have any questions or concerns about the project
The principal researcher and first contact for this project is:

Ghassan Yagot Al-Araji
Master of Osteopathy student
27 Cascades Road, Pakuranga, Auckland
09 576 9419
021 161 5500
The Effects of Muscle Energy Technique On The Viscoelasticity of Skeletal Muscle

Information Sheet

You are invited to take part in a research project being undertaken as a part of the Masters of Osteopathy Degree. This research involves investigating the effect of muscle energy technique on the viscoelasticity of skeletal muscle. This information sheet is designed to inform you as to the nature of the research, and what will happen should you decide to take part. We currently need right handed participants aged between 18 and 30 years of age who are reasonably fit, have a moderate build and have no current musculoskeletal pathology or injuries. The intervention and outcome measurements will be performed on the Biceps Brachii muscle which is located on the outside frontal part of the arm.

The Researchers
The researcher is Ghassan Y. Al-Araji. The research project is being supervised by Associate Professor Boris Gutnik.

What will participation involve?

- Attending a brief initial screening appointment to ensure that the inclusion and exclusion criteria are met and that you are eligible for the project. At this appointment you will be weighed, your height will be measured, and two brief questionnaires will be filled out pertaining to your hand dominance / preference.
- Discussing the procedures, and being informed of what happens in the research. After you've had time to consider participating you will be invited to sign the consent form
- Being available for 2 sessions of approximately 30 minutes. Both sessions will need to be on two consecutive days. Measurements of muscle viscoelasticity will be performed on both days and five applications of Muscle Energy Technique will be performed on the final day.

What is the nature of the outcome measurement and intervention?
The intervention that will be performed on the final day is five procedures of Muscle Energy Technique (each are 10 seconds in duration) that will be performed by a fully qualified and registered osteopath. Muscle energy technique (MET) is a technique used by osteopaths and many other manual therapists to treat many different muscular and facial complaints. Muscle energy technique is described as a manual therapy procedure which involves the voluntary contraction of the patient’s muscle in a precisely controlled direction at varying levels of intensity, against counter resistance applied by the operator.

The primary outcome measure will be muscle stiffness. This will be measured at three sessions; once on the first day and twice on the second day. Each measurement will involve mechanically lowering a stylus against the belly of the Biceps Brachii muscle. The stylus will be lowered at the rate between 0.05mm to 0.2mm per second providing external pressure to the belly of the Biceps Brachii muscle. At the point that the pressure becomes discomfiting the subject will press a button that stops the procedure. This process will be repeated three times for each measurement.

Potential Risks to Research Participants

There are no known published data indicating any risks associated with this research. However, the researcher accepts that it is possible there may be some undetermined risks involved in the research process. In the case that any potential risk of harm should arise for any research participant, it will be treated on an individual basis. In any such case the research process will be halted immediately.

Confidentiality

Confidentiality and your anonymity will be protected in the following ways:

- All consent forms and completed questionnaires will be seen only by the researchers.
- All hard copies will be stored in a locked file in a secured room. Only the researchers will have access to this file.
- Only anonymous data will be presented in reports related to this research.
- Electronic files will be protected with an electronic password.

You have the right not to participate, or to withdraw from this research project within two weeks of the final data collection. This can be done by contacting Ghassan Al-Araji or Associate Professor Boris Gutnik by telephone or email, or by verbally informing them when they contact you that you no longer wish to participate.

A final report containing the information from this study will be available at the Unitec Main Library on completion.

Information and Concerns

For further information or concerns please contact the researchers by phone, email, or fax.

Ghassan Al-Araji
School of Health and Community Studies
Unitec New Zealand
Thank you for your valuable time and contribution to this research.

This study has been approved by the Unitec Research Ethics Committee from 3rd of May to 31st of December 2006. If any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the Secretary (ph: 09 815-4321 ext 8041). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.

Hand Preference Questionnaire
Adjusted to Oldfield (1971)

Please indicate the preference in the use of hands in the following activities by putting + in the appropriate column. Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, put ++. If in any case you are really indefinite, put + in both columns. Some of the activities require both hands. In this case indicate which hand you use as the upper or lower hand respectively (as indicated in the brackets). Please try to answer all the questions, and only leave a blank if you have no experience at all of the object or task.

<table>
<thead>
<tr>
<th>Subject’s name: …………………………………………</th>
<th>R</th>
<th>L</th>
</tr>
</thead>
<tbody>
<tr>
<td>1) Which hand do you use when writing?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2) Which hand do you use when drawing?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3) Which hand do you use when throwing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4) With which hand do you use a pair of scissors?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5) With which hand do you use a comb?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6) With which hand do you use a toothbrush?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7) With which hand do you use knife (without fork)?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8) With which hand do you use a spoon?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9) With which hand do you use a hammer?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10) With which hand do you use a screwdriver?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11) With which hand do you use a tennis racket?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12) With which hand do you use a knife (with fork)?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
13) With which hand do you use a cricket bat (lower hand)?

14) With which hand do you use a golf club (lower hand)?

15) With which hand do you use a broom (upper hand)?

16) With which hand do you use a rake (upper hand)?

17) Which hand do you use when striking a match (matches)?

18) Which hand do you use when opening box (lid)?

19) Which hand do you use when dealing cards (cards being dealt)?

20) Which hand do you use when threading a needle?

---

**Individual Subject Data Sheet**

Date: .................................................................

Subject’s Name: ..........................................................

Date of birth: ..........................................................

Subject’s height (cm): ..............................................

Subject’s weight (kg): ..............................................

Calculated Body Mass Index (mass/hight\(^2\)): ..................

Scapula skin fold (mm):.............................................

Calf skin fold (mm):...............................................  

Triceps skin fold (mm):..............................

Calculated Endomorphy Somatotype level:
Sum of skin folds x 170.18 / Height (cm) = ..................

Biceps landmarks distance from AC joint (cm):
  Centre of brachial fossa: ..............................  
  Centre point (50%): .......................................

---
Point of probe one (25%): …………………………..
Point of probe two (75%): …………………………

Biceps skin fold:

<table>
<thead>
<tr>
<th>Measurement</th>
<th>1st measurement</th>
<th>2nd measurement</th>
<th>3rd measurement</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>- 25% distance</td>
<td>……………</td>
<td>……………</td>
<td>……………</td>
<td>……………</td>
</tr>
<tr>
<td>- 50% distance</td>
<td>……………</td>
<td>……………</td>
<td>……………</td>
<td>……………</td>
</tr>
<tr>
<td>- 75% distance</td>
<td>……………</td>
<td>……………</td>
<td>……………</td>
<td>……………</td>
</tr>
<tr>
<td>Final average (mm)</td>
<td>……………</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Elbow joints circumference (cm): Left……………….     Right……………….

Wrist joint circumference (cm): Left……………….     Right……………….

Maximum isometric contractile force (N): ………………………….

Subject’s Signature: ………………………………………………………

**Experimental Time Table**

**Thursday 20th April:**

9:00am ……………………………………………………………………………………..

9:30am ……………………………………………………………………………………..

10:00am ……………………………………………………………………………………

10:30am ……………………………………………………………………………………

11:00am ……………………………………………………………………………………

11:30am ……………………………………………………………………………………

12:00noon …………………………………………………………………………………

12:30pm ……………………………………………………………………………………

1:00pm ……………………………………………………………………………………..

1:30pm ……………………………………………………………………………………..
Friday 21st April:

9:00am .................................................................
9:30am .................................................................
10:00am .................................................................
10:30am .................................................................
11:00am .................................................................
11:30am .................................................................
12:00noon ..............................................................
12:30pm .................................................................
1:00pm .................................................................
1:30pm .................................................................

Thursday 27th April:

9:00am .................................................................
9:30am .................................................................
10:00am .................................................................
10:30am .................................................................
11:00am .................................................................
11:30am .................................................................
12:00noon ..............................................................
12:30pm .................................................................
1:00pm .................................................................
1:30pm .................................................................
Friday 28th April:

9:00am ...........................................................................................................

9:30am ...........................................................................................................

10:00am ........................................................................................................

10:30am .........................................................................................................

11:00am ........................................................................................................

11:30am .........................................................................................................

12:00noon ..................................................................................................

12:30pm ........................................................................................................

1:00pm ...........................................................................................................

1:30pm ............................................................................................................

Table 2: Average stiffness figures for subjects 1-5

<table>
<thead>
<tr>
<th>Day</th>
<th>Trial</th>
<th>N/mm Person 1</th>
<th>N/mm Person 2</th>
<th>N/mm Person 3</th>
<th>N/mm Person 4</th>
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<th>N/mm Person 8</th>
<th>N/mm Person 9</th>
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<td>1.451</td>
<td>1.337</td>
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<td>1.782</td>
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<td>0.854</td>
<td>1.154</td>
<td>2.298</td>
<td>1.414</td>
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<td>1.168</td>
<td>1.960</td>
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<td>Trial 4</td>
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### Table 4: Average stiffness figures for subjects 11-15

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<th>N/mm Person 11</th>
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<th>N/mm Person 13</th>
<th>N/mm Person 14</th>
<th>N/mm Person 15</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>Trial 1</td>
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<td>1.758</td>
<td>1.641</td>
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<td>1.978</td>
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<td>2.033</td>
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### Table 5: Average specific power of resistance figures for subjects 1-5

<table>
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<th>Day</th>
<th>Trial</th>
<th>Person 1</th>
<th>Person 2</th>
<th>Person 3</th>
<th>Person 4</th>
<th>Person 5</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day 1</td>
<td>Trial 1</td>
<td>0.2770</td>
<td>0.2070</td>
<td>0.2460</td>
<td>0.1500</td>
<td>0.1980</td>
</tr>
<tr>
<td>Day 1</td>
<td>Trial 2</td>
<td>0.2330</td>
<td>0.1310</td>
<td>0.2390</td>
<td>0.1160</td>
<td>0.1630</td>
</tr>
<tr>
<td>Day 1</td>
<td>Trial 3</td>
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<td>0.1380</td>
<td>0.2030</td>
<td>0.1290</td>
<td>0.1510</td>
</tr>
<tr>
<td></td>
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<td>0.1707</td>
</tr>
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<td>Trial 4</td>
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<td>0.2040</td>
<td>0.2400</td>
<td>0.1460</td>
<td>0.1670</td>
</tr>
<tr>
<td>Day 2</td>
<td>Trial 5</td>
<td>0.2350</td>
<td>0.1630</td>
<td>0.2460</td>
<td>0.1310</td>
<td>0.1770</td>
</tr>
<tr>
<td>Day 2</td>
<td>Trial 6</td>
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<td>0.1630</td>
<td>0.2110</td>
<td>0.1400</td>
<td>0.1730</td>
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<tr>
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<td>Average</td>
<td>0.2290</td>
<td>0.1767</td>
<td>0.2323</td>
<td>0.1390</td>
<td>0.1723</td>
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<td>0.2308</td>
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<td>Trial 7</td>
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<td>Day 2</td>
<td>Trial 8</td>
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<td>0.1750</td>
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<td>Trial 9</td>
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### Table 6: Average specific power of resistance figures for subjects 6-10

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<th>Day</th>
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<th>Person 8</th>
<th>Person 9</th>
<th>Person 10</th>
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<tr>
<td>Day 1</td>
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<tr>
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<td>Trial 2</td>
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<td>0.1610</td>
<td>0.1510</td>
<td>0.2030</td>
<td>0.1600</td>
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<tr>
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<td>Trial 3</td>
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<td>0.1280</td>
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<td>0.2310</td>
<td>0.1530</td>
</tr>
<tr>
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<td>Average</td>
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<td>0.1490</td>
<td>0.1283</td>
<td>0.2033</td>
<td>0.2040</td>
</tr>
<tr>
<td>Day</td>
<td>Trial</td>
<td>Person 11 (watts/m)</td>
<td>Person 12 (watts/m)</td>
<td>Person 13 (watts/m)</td>
<td>Person 14 (watts/m)</td>
<td>Person 15 (watts/m)</td>
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<td>Subject's Endomorphic Level</td>
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Table 9: Subject individual pre-measurement data

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<th>Wrist Joint (cm)</th>
<th>Time of measurement (seconds after MET)</th>
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<td>Right</td>
<td>Left</td>
</tr>
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<td>26</td>
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<td>15.5</td>
</tr>
<tr>
<td>7</td>
<td>25</td>
<td>25</td>
<td>16.5</td>
</tr>
<tr>
<td>8</td>
<td>27.5</td>
<td>27.5</td>
<td>17</td>
</tr>
<tr>
<td>9</td>
<td>27</td>
<td>26.8</td>
<td>16</td>
</tr>
<tr>
<td>10</td>
<td>27</td>
<td>27</td>
<td>16.8</td>
</tr>
<tr>
<td>11</td>
<td>32</td>
<td>31.5</td>
<td>19</td>
</tr>
<tr>
<td>12</td>
<td>27.5</td>
<td>27.5</td>
<td>18</td>
</tr>
<tr>
<td>13</td>
<td>25.5</td>
<td>26</td>
<td>16.5</td>
</tr>
</tbody>
</table>
T-Test for Stiffness: pre-intervention day 1 vs. pre-intervention day 2

<table>
<thead>
<tr>
<th>Pair</th>
<th>Mean stiffness N/mm</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>day1</td>
<td>1.8060</td>
<td>15</td>
<td>.52350</td>
<td>.13517</td>
</tr>
<tr>
<td>day2</td>
<td>1.9527</td>
<td>15</td>
<td>.60702</td>
<td>.15673</td>
</tr>
</tbody>
</table>

Paired Samples t Test

<table>
<thead>
<tr>
<th>Pair</th>
<th>Mean stiffness N/mm</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>95% Confidence Interval of the Difference</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td>day1</td>
<td>-1.4673</td>
<td>.39803</td>
<td>.10277</td>
<td>-.36715</td>
<td>-1.428</td>
<td>14</td>
<td>.175</td>
</tr>
<tr>
<td>day2</td>
<td></td>
<td></td>
<td></td>
<td>.07369</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
stiffness data remained constant over these two occasions. This result suggests the device used provided reliable data.
T-Test for Specific Power of Resistance: pre-intervention day 1 vs. pre-intervention day 2

Paired Samples Statistics

<table>
<thead>
<tr>
<th>Pair 1</th>
<th>Mean power of resistance W/m</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>day1</td>
<td>.1887</td>
<td>15</td>
<td>.05008</td>
<td>.01293</td>
</tr>
<tr>
<td>day2</td>
<td>.1950</td>
<td>15</td>
<td>.03632</td>
<td>.00938</td>
</tr>
</tbody>
</table>

Paired Samples t Test

<table>
<thead>
<tr>
<th>Pair 1 day1 - day2</th>
<th>Mean power of resistance W/m</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>95% Confidence Interval of the Difference</th>
<th>t</th>
<th>df</th>
<th>Sig. (2-tailed)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-.00629</td>
<td>.03384</td>
<td>.00874</td>
<td>-.02503</td>
<td>-.719</td>
<td>14</td>
<td>.484</td>
</tr>
</tbody>
</table>

Figure 7.2: Statistical analysis from SPSS 12.0.1 for Windows®: showing comparison between pre-intervention day 1 data and pre-intervention day 2 data for power of resistance. The analysis shows that the power of resistance data remained constant during pre-intervention.
T-Test for Stiffness: pre-intervention average vs. post-intervention

Paired Samples Statistics

<table>
<thead>
<tr>
<th></th>
<th>Mean stiffness N/mm</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preint</td>
<td>1.8793</td>
<td>15</td>
<td>.53071</td>
<td>.13703</td>
</tr>
<tr>
<td>Postint</td>
<td>1.7925</td>
<td>15</td>
<td>.45802</td>
<td>.11826</td>
</tr>
</tbody>
</table>

Paired Samples t Test

<table>
<thead>
<tr>
<th></th>
<th>Paired Differences</th>
<th>95% Confidence Interval of the Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean stiffness N/mm</td>
<td>Std. Error Mean</td>
</tr>
<tr>
<td>Preint -</td>
<td>-.08683</td>
<td>.07041</td>
</tr>
<tr>
<td>Postint</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 7.3: Statistical analysis from SPSS 12.0.1 for Windows®: showing comparison between average pre-intervention data and post-intervention data for stiffness. There is no significant difference between these sets of data.
T-Test for Specific Power of Resistance: pre-intervention average vs. post-intervention

Paired Samples Statistics

<table>
<thead>
<tr>
<th></th>
<th>Mean power of resistance W/m</th>
<th>N</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Pair 1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>preint</td>
<td>.1919</td>
<td>15</td>
<td>.04035</td>
<td>.01042</td>
<td></td>
</tr>
<tr>
<td>postint</td>
<td>.1859</td>
<td>15</td>
<td>.03714</td>
<td>.00959</td>
<td></td>
</tr>
</tbody>
</table>

Paired Samples t Test

<table>
<thead>
<tr>
<th></th>
<th>Mean power of resistance W/m</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>95% Confidence Interval of the Difference</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Paired</td>
<td></td>
<td>Std. Deviation</td>
<td>Std. Error Mean</td>
<td>Lower</td>
<td>Upper</td>
<td>t</td>
</tr>
<tr>
<td>Pair 1</td>
<td></td>
<td>.00592</td>
<td>.02521</td>
<td>.00651</td>
<td>-.00804</td>
<td>.01988</td>
</tr>
</tbody>
</table>

Figure 7.4: Statistical analysis from SPSS 12.0.1 for Windows®: showing comparison between average pre-intervention data and post-intervention data for power of resistance. The analysis shows no significant individual difference attributable to the intervention.
Post-hoc Test (Cohen test) for post-intervention data (stiffness)

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>2.3235</td>
<td>1.809</td>
</tr>
<tr>
<td>1.6785</td>
<td>1.482</td>
</tr>
<tr>
<td>2.333</td>
<td>2.384333</td>
</tr>
<tr>
<td>0.899</td>
<td>1.270667</td>
</tr>
<tr>
<td>1.323</td>
<td>1.376667</td>
</tr>
<tr>
<td>2.152333</td>
<td>2.386</td>
</tr>
<tr>
<td>1.355167</td>
<td>1.067667</td>
</tr>
<tr>
<td>1.490333</td>
<td>1.640333</td>
</tr>
<tr>
<td>2.3065</td>
<td>2.084667</td>
</tr>
<tr>
<td>1.6635</td>
<td>1.297333</td>
</tr>
<tr>
<td>1.930167</td>
<td>1.785</td>
</tr>
<tr>
<td>2.848167</td>
<td>2.474667</td>
</tr>
<tr>
<td>1.938167</td>
<td>1.666</td>
</tr>
<tr>
<td>2.492333</td>
<td>2.368667</td>
</tr>
<tr>
<td>1.456167</td>
<td>1.794333</td>
</tr>
</tbody>
</table>

**MEAN**

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.879322</td>
<td>1.792489</td>
</tr>
</tbody>
</table>

**SD**

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.530712</td>
<td>0.458015</td>
</tr>
</tbody>
</table>

**SD^2**

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.281655</td>
<td>0.209778</td>
</tr>
</tbody>
</table>

**Numerator**

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.086833</td>
<td></td>
</tr>
</tbody>
</table>

**Denominator**

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.494363</td>
<td></td>
</tr>
</tbody>
</table>

*Effect Stat (d)*

<table>
<thead>
<tr>
<th>Pre-interventions</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>0.175647</strong></td>
<td>small, low, minor</td>
</tr>
</tbody>
</table>

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Figure 7.5: Post-hoc test for pre-intervention vs. post-intervention data (stiffness), showing that the effect size between the two sets of data is small, low, minor (Hopkins, 2000)

**Post-hoc Test (Cohen test) for post-intervention data**
**(specific power of resistance)**

<table>
<thead>
<tr>
<th>Pre-intervention</th>
<th>Post-intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.230667</td>
<td>0.1883</td>
</tr>
<tr>
<td>0.167667</td>
<td>0.1767</td>
</tr>
<tr>
<td>0.230833</td>
<td>0.2350</td>
</tr>
<tr>
<td>0.135333</td>
<td>0.1630</td>
</tr>
<tr>
<td>0.1715</td>
<td>0.1647</td>
</tr>
<tr>
<td>0.1565</td>
<td>0.2097</td>
</tr>
<tr>
<td>0.157</td>
<td>0.1413</td>
</tr>
<tr>
<td>0.1515</td>
<td>0.1493</td>
</tr>
<tr>
<td>0.227833</td>
<td>0.2153</td>
</tr>
<tr>
<td>0.179</td>
<td>0.1423</td>
</tr>
<tr>
<td>0.2190</td>
<td>0.1877</td>
</tr>
<tr>
<td>0.2817</td>
<td>0.2737</td>
</tr>
<tr>
<td>0.1743</td>
<td>0.1507</td>
</tr>
<tr>
<td>0.2180</td>
<td>0.2000</td>
</tr>
<tr>
<td>0.1770</td>
<td>0.1913</td>
</tr>
</tbody>
</table>

**MEAN**

<table>
<thead>
<tr>
<th>Pre-intervention</th>
<th>0.191856</th>
<th>Post-intervention</th>
<th>0.1859</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>SD</strong></td>
<td>0.040344</td>
<td>0.037135</td>
<td></td>
</tr>
<tr>
<td><strong>SD^2</strong></td>
<td>0.001628</td>
<td>0.001379</td>
<td></td>
</tr>
</tbody>
</table>

**Numerator** 0.0059  
**Denominator** 0.038739
Effect Stat \((d)\) \quad 0.152873 \quad \text{small, low, minor}

Figure 7.6: Post-hoc test for pre-intervention vs. post-intervention data (specific power of resistance), showing that the effect size between the two sets of data is small, low, minor (Hopkins, 2000)