

**Immediate effects of High-Velocity thrust to
the cervical spine on Pressure Pain Threshold
and Pain-Free Grip Strength in subjects with
Lateral Epicondylalgia**

Alastair Treacher

A research project submitted in partial fulfillment for the requirements for the degree of

Master of Osteopathy at Unitec 2011

Declaration

Name of candidate: Alastair Graeme Treacher

This Research Project entitled **“Immediate effects of high velocity thrust of the cervical spine on pressure pain threshold and pain free grip strength in subjects with lateral epicondylalgia”** is submitted in partial fulfillment for the requirements for the Unitec degree of Master of Osteopathy.

CANDIDATE'S DECLARATION

I confirm that:

- This Research Project represents my own work;
- The contribution of supervisors and others to this work was consistent with the Unitec Regulations and Policies.
- Research for this work has been conducted in accordance with the Unitec Research Ethics Committee Policy and Procedures, and has fulfilled any requirements set for this project by the Unitec Research Ethics Committee.

Research Ethics Committee Approval Number: 2009-1056

Candidate Signature: _____ Date: _____

Alastair Graeme Treacher

Student number: 1217643

Abstract

Background and objective: To measure the immediate effect of a High-Velocity Low-Amplitude (HVLA) manipulation targeting the C_{5/6} vertebral segment on pain intensity in subjects with Lateral Epicondylalgia (LE).

Design: Randomised assessor blinded controlled experiment.

Methods: Ten subjects (7 male, 3 female; mean age= 37.7, SD=10.8) with unilateral elbow pain participated in this study. Prior to enrolment subjects were screened to establish the presence of LE. Pain-Free Grip Strength (PFGS) and Pressure Pain Threshold (PPT) at the lateral epicondyle were measured for both arms prior to and immediately following the application of either High-Velocity Low-Amplitude thrust targeting the C_{5/6} vertebral segment or the control condition.

Results: The intervention group demonstrated an increase in mean Pain-Free Grip strength ($37.9 \pm 19.2\text{N}$) following a High-Velocity Low-Amplitude manipulation ($p=0.03$, $d=0.32$) compared to a decrease in Pain-Free Grip Strength ($25.6 \pm 24.2\text{N}$, $p= 0.13$) observed in the control group. The increase in Pain-Free Grip Strength observed in the intervention group exceeded the *a priori* Smallest Detectable Difference (14N). Neither the control or intervention group demonstrated substantial within group or between group change in mean Pressure Pain Threshold (control: $p=0.59$, 1.4 N/cm^2 ; intervention: $p=0.3$, 3.3 N/cm^2 ; between group: $p=0.08$, 8.8 N/cm^2) following the intervention.

Conclusion: High-Velocity Low-Amplitude thrust targeting the C_{5/6} vertebral segment can lead to a moderate increase in pain free grip strength in subjects with Lateral Epicondylitis. The change observed in PFGS following HVLA also indicates that further work exploring the proposed cervical component of LE is justified.

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List of Abbreviations

LE	Lateral Epicondylalgia
HVLA	High-Velocity Low-Amplitude
PPT	Pressure Pain Threshold
PFGS	Pain Free Grip Strength
VAS	Visual Analog Scale
SMT	Spinal Manual Therapy
ECRB	Extensor Carpi Radialis Brevis
ECRL	Extensor Carpi Radialis Longus
CEO	Common Extensor Origin
PAG	Periaqueductal Gray area
SEM	Standard Error of Measurement
ICC	Intra-class Correlation Coefficient
SDD	Smallest Detectable Difference
SD	Standard Deviation
MWM	Mobilisation With Movement
MCID	Minimal Clinically Important Difference

Section I: Review of literature

Introduction

The rationale for conducting this review was twofold; to assess current body of evidence relating to the pathophysiology and treatment of Lateral epicondylalgia (LE), and investigate the claim there may be a cervical component that contributes to the condition of LE. The preceding review of literature will begin by outlining the condition of LE including aetiology, relevant anatomy and the current understanding of the associated pathological mechanisms. The following section will focus on pain, the processes involved in pain generation, the proposed effects of manual therapy on pain and validity of the methodological procedures used in this study to measure pain. Finally the review will consider the role of manual therapy in the management of LE and assess the studies that have explored the proposed link the cervical spine and LE and their implications for treatment.

Aetiology and epidemiology

Lateral epicondylalgia is considered a relatively common musculoskeletal disorder and is the most common diagnosis made for patients presenting with elbow pain (Hong, Durand, & Loisel, 2004). Few studies have investigated the prevalence of LE, one Swedish study estimated the prevalence of LE is to be 1-3% of the general population (Allander, 1974) and another study from the United Kingdom reported LE accounts for approximately four out of every 1000 visits to general practitioners per year (Hamilton, 1986). The prevalence in New Zealand remains unclear. Prevalence of LE appears to be highest between 35-54 years of age (mean age= 45) affecting men and women equally (Hamilton, 1986).

Lateral epicondylalgia may occur as a result of an acute or chronic strain of one or more of the wrist extensor tendons (Goguin & Rush, 2003). Acute injuries such as a muscle strain or laceration usually occur as a result of trauma (Kraushaar & Nirschl, 1999). Chronic strain is generally associated with disruption of the internal structure of the tendon that occurs as a result of repeated microtrauma (Kraushaar & Nirschl, 1999). One of the principal risk factors for the development of

chronic LE appears to be repetitive forceful movements of the upper limb that are associated with occupations such as factory line workers (Chiang et al., 1993; Melchior et al., 2006).

Lateral epicondylalgia is often attributed to work related repetitive strain injuries but is also a common sports injury (Gellman, 1992) with up to 50% of tennis players in the USA being affected at some time (Labelle et al., 1992). Some evidence is available that suggests that certain professions may be up to five times more likely to be afflicted by LE. Job classifications that involve repetitive manually intensive tasks with high force demands such as construction workers, auto mechanics and butchers have been reported as being between 2-5 times more likely to suffer from LE than the general population (Fedorczyk, 2006). A Scandinavian study that reported the incidence of shoulder and upper limb disorders in a fish processing plant, found that 31 of 207 employees (15%) were afflicted with LE (Chiang et al., 1993). It is not entirely clear if these findings offer an accurate reflection of the true prevalence of LE. A review exploring occupational disorders of the upper limb concluded that LE has no clear association with manually intensive professions but may be more troublesome for those involved in manual work (Barton, Hooper, Noble, & Steel, 1992). It is plausible that the explanation for this is due to an inability to avoid aggravating factors in work related LE.

Anatomy and Pathophysiology

The elbow is not one joint, but consists of three articulating surfaces; the humero-radial joint, the humero-ulnar joint and the proximal radio-ulnar joint all contained within a single joint capsule (Platzer, 2004). The primary function of the elbow is to orientate the hand in space (Palastanga, Field, & Soames, 2006), which is achieved by the combined articulation of the humero-ulnar joint, which allows flexion and extension; and the radio-ulnar joint which facilitates pronation and supination (Platzer, 2004). The elbow is primarily stabilised medially and laterally by passive ligamentous restraints (Palastanga et al., 2006). A degree of stabilisation is also provided by muscles and their tendons which insert around the elbow (Palastanga et al., 2006).

An anatomical area that is considered to be particularly relevant to the condition of LE is the common extensor origin (CEO). This is a blending of tendons of several muscles including; extensor carpi radialis longus and brevis (ECRL, ECRB), extensor digitorum and extensor carpi ulnaris (ECU) that insert onto the lateral epicondyle (Boyer & Hastings, 1999). Many of the individual tendons that comprise the CEO have been found to be indistinguishable via gross or microscopic anatomical studies (Goguin & Rush, 2003).

Normal tendons are comprised of three elements; collagen, tenocytes (tendon cells) and ground substance (Fedorczyk, 2006). The collagen fibres provide tensile strength with structural support supplied via the ground substance and proteoglycans. Tenocytes are dispersed amongst the collagen fibres and are responsible for synthesizing protein building blocks and ground substance (Ashe, McCauley, & Khan, 2004). Generally the vascularisation of tendons is poor, and in some regions may be absent, it is suspected that this is a factor in poor healing potential of some tendon injuries (Fedorczyk, 2006).

Nerve supply of the joint is provided anteriorly by branches of the median, radial and musculocutaneous nerves and posteriorly the radial and ulnar nerves all of which are supplied by root levels C₅₋₈ (Palastanga et al., 2006). The sensory nerve supply to the elbow region arising from the C₅ dermatome and the lateral cutaneous nerve with motor supply originating from C_{5,6} (flexion) and C_{7,8} (extension) (Petty & Moore, 2001).

Since LE was first described, several pathophysiological mechanisms have been proposed. Cyriax (1936) compiled a list of 26 separate lesions that may be responsible for the condition of LE. It is possible to broadly classify the lesions proposed by Cyriax into three groups involving a neuroirritative process, tendon damage or referred pain (Hong et al., 2004). It is not stated in this article the extent that these processes may coexist or overlap but some authors consider that LE may be a multi- factorial process in many cases (Vicenzino & Wright, 1996; Yaxley & Jull, 1993).

There is still no consensus regarding which anatomical structures are associated with LE. Damage to the tendons that extend the wrist and attach to the lateral epicondyle are often implicated i.e. the common extensor origin (Hong et al., 2004) but, it is difficult to isolate any one specific tendon that is responsible for causing symptoms (Goguin & Rush, 2003). Histological studies do show consistent pathological changes at extensor radialis brevis tendon in symptomatic populations (Kraushaar & Nirschl, 1999). It is now thought that the pain associated with LE is due to degenerative changes in the tendon rather than an inflammatory process (Stasinopoulos & Johnson, 2006) and occurs as a result of an accumulation of microtears leading to a disruption of collagen fibres and infiltration of atypical granulation tissue (Kraushaar & Nirschl, 1999; Scher & Wolf, 2009). The term lateral epicondylitis is still commonly used to describe lateral elbow pain. The term epicondylitis implies an inflammatory process is responsible for the pain associated with LE yet histological studies of tendons in those suffering from LE frequently demonstrate a lack of acute inflammatory markers such as increased infiltration of macrophages and neutrophils into the tissue area (Kraushaar & Nirschl, 1999). Biopsies of the extensor carpal radialis brevis show evidence of necrosis and muscle fibre regeneration over the whole muscle (Goguin & Rush, 2003). It has been postulated that the degenerative process may be a result of hypoxia of the tissue (Regan, World, Coonrad, & Morrey, 1992). Hypoxia is thought to be as a result of mechanical overload and stress. Poor vascularisation then leads to an incomplete healing response which causes degenerative weakening and microtears of the ECRB tendon (Goguin & Rush, 2003).

Other diagnostic possibilities must be considered when presented with elbow pain. The next most common condition of the elbow that may mimic or co-exist with LE is radial tunnel syndrome. This condition usually produces a vague ache experienced distally to the lateral epicondyle and may be difficult to clinically distinguish from LE (Boyer & Hastings, 1999). Radial nerve entrapment has been reported to occur in 5% of cases of LE (Yaxley & Jull, 1993). Yaxley and Jull (1993) used a neurological tension test developed by Butler (1991) to explore adverse tension in the neural system

in subjects who were receiving physiotherapy for unilateral LE. This test was positive in 11 of the 20 subjects which suggests that involvement of the radial nerve may be more prevalent than previously thought.

Another hypothesis within the field of manual therapy proposes LE can arise as a result of cervical spine dysfunction. A study conducted in Canada on 50 patients, who had failed to respond to four weeks of conservative treatment, found that 86% achieved good or satisfactory relief of symptoms within 5 weeks of undergoing a treatment protocol directed at the cervical spine (Gunn & Milbrandt, 1976). The treatments employed in this study included one or more of the following, mobilisation, traction, isometric exercise and heat or ultrasound directed at the cervical spine. In a more recent study by Cleland, Whitman and Fritz, (2004) 112 patients with LE were randomly assigned into two groups, one receiving local management (LM) and the other receiving local management plus cervical manual therapy (LM+C). Both groups in this study achieved similarly positive outcomes when followed up approximately 74 weeks post discharge. Cleland *et al.* (2004) established that the LM+C group received on average 4.1 fewer total treatments than the LM group but did not specify the overall duration of treatment for either group. The findings of both of these studies suggest that inclusion of the cervical spine in manual treatment of LE may improve outcomes, but both studies had limitations. For example; neither study was able to account for the self limiting nature of LE through the use of an untreated control group, both relied on self reported outcome measures and neither study had a control group.

Pain and the role of manual therapy

The international association of the study of pain defines pain as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage or described in terms of such damage” (Merskey & Bogduk, 1994b). This definition illustrates the complex process of pain, which consists of physiological as well as psychological components (Strong, Unruh, Wright, &

Baxter, 2002). However, it is beyond the scope of this review to consider the psychological aspects of pain, so it will focus primarily on the physiology. Pain response arising from a peripheral insult can be classified as either hyperalgesia or allodynia (Sluka, 1996). Hyperalgesia is the term used to describe an increased response to a previously noxious stimulus. Allodynia is the term used to describe a painful response to a previously innocuous stimuli (Merskey & Bogduk, 1994a). Hyperalgesia can be further subdivided into primary and secondary hyperalgesia. Primary hyperalgesia is defined as an increase in pain response to a noxious stimuli at the site of injury whereas secondary hyperalgesia is an increased pain response in areas not directly related to the site of injury (Sluka, 1996). It is believed that secondary hyperalgesia may be as a result of changes in the central nervous system (Sluka, 1996). This phenomenon, known as central sensitisation, will be discussed in the following section.

Neurobiology of pain

Much of the current understanding of pain mechanisms comes from the work of Melzak and Wall (1967). Their hypothesis, known as gate control theory, helped to illuminate the role of the nervous system in the pain process. The nervous system is usually divided into two parts; the central nervous system and peripheral nervous system (Guyton & Hall, 2000). This feedback from afferent neurons allows for the appropriate efferent response to maintain homeostasis and limit any potential tissue damage (Holdcroft & Jaggard, 2005). Nociception is the term used to describe the neurological mechanism responsible for detecting potential tissue harming stimuli (Holdcroft & Jaggard, 2005). Experimental data has been able to establish that nociception is signalled by two sets of nerve fibres; A δ fibres are small thinly myelinated fibres responsible for sharp well localised pain, C fibres are small unmyelinated fibres responsible for dull diffuse pain (Strong et al., 2002). The process of nociception may be subject to modification at various stages leading to changes in intensity as well as the location of pain (Siddall & Cousins, 1997). The two key modifying factors that will be discussed in this section are peripheral sensitisation and central sensitisation.

Pain modifying factors

The extent to which a person reacts to pain varies immensely. This is partly a result of the varying ways that pain can be processed by the brain, central nervous system and peripheral nervous system. The brain is capable of suppressing pain input to the nervous system via a pain control system known as the analgesia system (Guyton & Hall, 2000). The analgesia system consists of three components; 1) the periaqueductal gray and periventricular areas of the upper pons 2) raphe magnus nucleus located in the lower pons and upper medulla 3) a pain inhibitory complex in the dorsal horn of the spinal cord (Guyton & Hall, 2000, p. 602).

Central sensitisation is a result of changes in the excitability of nociceptive pathways in the dorsal horn of the spinal cord and the dorsal root ganglion due to prolonged stimulus of nociceptors (Siddall & Cousins, 1997; Ward & Hruby, 2002). These changes lead to increased firing of neurons in the spinothalamic tract as well as greater responsiveness of neurons to noxious and innocuous stimuli (Sluka, 1996). Furthermore central sensitisation initiates an increase in receptor field size, an increase in the duration as well as a heightened sensitivity to stimulus (Siddall & Cousins, 1997).

Peripheral sensitisation results from tissue damage, the development of a local inflammatory response and the release of inflammatory mediators which can alter the properties of high threshold nociceptors (Ward & Hruby, 2002). Under normal circumstances the activation threshold of mechanical, thermal and chemical nociceptors is high (Butler, 2000; Siddall & Cousins, 1997). When tissue damage occurs, inflammatory mediators such as substance P and histamine are released which can lead to direct activation of nociceptors as well as lowering the activation threshold (Holdcroft & Jaggar, 2005). The ultimate consequence of this process is that low intensity stimulus that would not normally generate pain will be perceived as pain (Butler, 2000). The presence of

ongoing stimulus of the nociceptor will ultimately result in increased pain intensity (Siddall & Cousins, 1997).

Spinal Manual Therapy and Pain

Evidence indicates that spinal manual therapy (SMT) is able to influence nervous system function and produce both local and distal analgesic effects. A systematic review identified 15 studies conducted between 1996-2007 that explored the effect of passive cervical mobilisation on a number of measures including; pressure pain threshold, pain free grip strength, thermal pain threshold and skin conductance (Schmid, Brunner, Wright, & Bachmann, 2008). Schmid *et al.* found sufficient evidence to suggest that cervical mobilisation is capable of altering pain control mechanisms and modulation of the sympathetic nervous system as well as evidence for improvement in Pain free grip strength (PFGS) in the three studies that used this as an outcome measure.

One of the structures implicated as being involved in manually induced hypoalgesia is the periaqueductal grey area (PAG). A seminal study by Reynolds (1969) was able to demonstrate electrical stimulation of the PAG produced analgesia in rats. Similar effects have been demonstrated in human subjects when the PAG is stimulated by electrodes (Behbehani, 1995). Although SMT is unable to provide direct stimulation of the PAG it is hypothesised that it may be able to stimulate the descending pain control system projecting from the PAG to the spinal cord (Wright, 1995). Maigne and Vautravers (2003) propose that activation of the PAG may occur due to rapid stretching of ligaments, muscles and the joint capsule that results from manipulation.

A number of studies exploring gate theory have shown that a mechanical stimulus transmitted along myelinated A fibre neurons can inhibit nociceptive stimuli from C fibres thus modifying the effects of central sensitisation by removing subthreshold stimuli (Pickar, 2002). It is currently not clear if the pain inhibiting mechanisms of SMT share these properties with the gate control theory

(Pickar, 2002) but it does offer another possible explanation of the apparent neurophysiological effects of SMT.

Activation of the neuroendocrine system may provide another explanation of the pain modifying factors that appear to arise as a result of HVLA (Pickar, 2002). Several studies have attempted to explore the effect of high-velocity low-amplitude thrusts (HVLA) on the circulating levels of β -endorphin with mixed results. For example, Vernon *et al.* (1986) reported a statistically significant increase (8%) in circulating β -endorphin levels where as Sanders *et al.* (1990) were unable to detect any increase in β -endorphin levels despite a reduction on the visual analogue scale in the group receiving SMT.

High-Velocity Low-Amplitude thrust techniques

According to Greenman, the goal of manipulation is to “restore maximal pain free movement of the musculoskeletal system in postural balance” (1996, p. 5). There are a number of techniques that are claimed by osteopaths to achieve this goal. HVLA, a subgroup of SMT, are perhaps the best known of all manipulative techniques (DiGiovanna, Schiorowitz, & Dowling, 2005) and when performed is often accompanied by an audible “popping” or “cracking” sound (Gibbons & Tehan, 2001) although some believe that the sound may not be necessary for this technique to be effective (Flynn, Fritz, Wainner, & Whitman, 2003). Bakker and Miller (2004) speculate the audible “pop” that often accompanies HVLA may increase the therapeutic benefits of this technique, but this perceived improvement is likely to be more psychological than physiological. It is also possible to distinguish manipulation from other forms of mobilisation in that it is delivered near the end of physiological range of motion (Pickar, 2002).

There are a number of reasons why a therapist may choose to utilise this type of intervention including; increasing range of motion of dysfunctional joints, reduce hypertonicity of muscles, and reset aberrant neurological pathways (Bruckner & Khan, 1994; Greenman, 1996; Maigne &

Vautravers, 2003). Evidence is emerging that HVLA is beneficial in the treatment of low back pain in both the chronic and acute phase and may be of use in cases of chronic neck pain (Bronfort, Haas, Evans, & Bouter, 2004). This review included 31 studies of LBP and 23 studies involving neck pain. Although there is increasing evidence relating to the benefits of HVLA the mechanism of its effects remains poorly understood (Pickar, 2002).

Outcome measures used in this study

Pressure algometry

Pressure algometry is a widely used and accepted way of assessing pressure pain threshold and the onset of pain sensation both within research literature (Itoh, Okada, & Kawakita, 2004) as well as within the clinical setting (Maquet, Croisier, Demoulin, & Crielaard, 2004). Pressure algometry is used to quantify a patient's pain by determining the pressure pain threshold, or the point at which a subject perceives pain upon the application of force (Vaughan, McLaughlin, & Gosling, 2007). Algometry can be used both as a diagnostic tool (Gracely, Grant, & Giesecke, 2003) as well as a means to assess the effectiveness of treatments that attempt to alleviate soft tissue pain (Potter, McCarthy, & Oldham, 2006).

PPT is measured by applying force to a particular site and increasing the amount of pressure and recording the point where the sensation of pressure changes to a sensation of pain. PPT changes are based on subjective information and the subject's response may be affected by a number of factors, such as gender, age and cultural beliefs. Chesterton et al. (2003) measured PPT at the first dorsal interosseous muscle of 240 healthy volunteers and found that females had a significantly lower PPT than males. Five out of six studies looked at in reviews conducted by Gibson and Helme (2001) reported an increase in pain thresholds in subjects of advanced age. This review failed to specify the

age of the subjects. It is proposed that the changes in PPT associated with age may be related to a decreased density of nociceptors (Turk & Melzack, 2001)

A number of methodological issues with algometry have been identified within the research. These issues include the failure to indicate rate of pressure application (Kosek, Ekholm, & Nordemar, 1993), use of a verbal command to indicate onset of pain which relies on the reaction time of the examiner (Vatine, Shapira, Magora, Adler, & Magora, 1993) and sensitisation or habituation due to pressure previously applied during repeated measures (Hogeweg, Langereis, Bernards, Faber, & Helder, 1992).

Grip dynamometry

Grip strength testing has been utilized in a range of areas to for a number of purposes including; assessment of upper limb impairment, evaluation of people suffering disabilities such as rheumatoid arthritis and determining efficacy of treatments of various disabilities (Innes, 1999). A review of randomised clinical trials exploring the effectiveness of physiotherapy for LE found that most trials (19/23) used grip strength as an outcome measure (Smidt, 2001, as cited in Smidt et al., 2002). Grip strength has been found to be a reliable and valid form of clinical assessment (A. Hamilton, Balnave, & Adams, 1994) and has demonstrated high generalisability of coefficients when used to assess patients with LE (P. W. Stratford, Norman, & McIntosh, 1989).

Manual therapy and LE

There is a limited understanding of the mechanisms involved in LE, and a lack of evidence to support any specific plan of care for the sufferers of this condition (Fedorczyk, 2006). More than 40 different treatments alone or in combination have been reported. These include steroids, anti-inflammatory drugs, numerous physical therapy interventions and surgery (Labelle et al., 1992). Currently, it is uncertain if any of the interventions favourably influence the long term outcome of LE

(Labelle et al., 1992). The lack of understanding of the pathological mechanism, methodological shortcomings of available research and the possibility that LE is a self limiting condition are factors that complicate the development of a successful treatment strategy (Hong et al., 2004). Boyer and Hastings (1999) critiqued a number of non-operative interventions in a review article including steroid injections, acupuncture, stretching, exercise and ultrasonography made the observation that none of the treatments evaluated was able to modify the natural history of LE. A study published in the Lancet suggests that a 'wait and see' approach involving patient education may provide adequate long term outcomes for sufferers of LE when compared to corticosteroid injections or physiotherapy (Smidt, van der Windt, Assendelft, Devillé et al., 2002). However this study did note that there were potential benefits regarding long-term recovery when physiotherapy is utilised.

The aim of LE treatment should be reduction of pain and restoration of muscle function (B. Vicenzino, 2003). One study concluded that the most effective way to restore muscle function is by undertaking a progressive resistance programme focusing primarily on the extensors of the wrist and hand (Pienimäki, Tarvainen, Siira, & Vanharanta, 1996). Some supporting evidence is available via animal studies that show muscle training can also improve the tensile strength of tendons (Ashe et al., 2004) which may assist in recovery and decrease the chance of further injury.

Techniques performed at the elbow such as Mulligan's manipulation with movement (MWM) have been demonstrated to provide a short term reduction of pain in those suffering from LE (Abbott, Patla, & Jensen, 2001; B. Vicenzino, Paungmali, Buratowski, & Wright, 2001). These studies conducted in New Zealand and Australia, included 23 and 24 subjects respectively. Both these studies utilised pain free grip strength as an outcome measure. Manipulation with movement involves lateral gliding of the ulnar with the humerus stabilized while the patient performs active pain free wrist movement (Abbott et al., 2001). Interestingly, it has been found that MWM does not elicit an effect in experimentally induced LE (saline injection and delayed onset muscle soreness)(Slater, Arendt-Nielsen, Wright, & Graven-Nielsen, 2006). The authors of this study suggest

that the explanation for this is that differing neural mechanisms, including central sensitisation, may be involved in the modulation of pain in acute and chronic forms of LE.

Wright *et al.* (1994) proposed that central sensitisation arising from the lower cervical spine may account for some of the clinical signs of LE. A study by Berglund (2008) examining the correlation between neck pain and LE found that 70% of those suffering from LE also indicated cervical spine pain. Although further study is required, these findings do suggest a degree of cervical involvement in the presentation of LE. Data is beginning to emerge that specific techniques directed to the cervical spine can provide short term relief of pain at the elbow (Slater *et al.*, 2006; B. Vicenzino, Collins, & Wright, 1996). However, there is a lack of data on the duration of the hypoalgesic effects of these interventions but, as Vicenzino (2003) points out, even a short term reduction in pain may be useful in the acceleration or optimisation of any rehabilitation programme.

Vicenzino *et al.* (1996) examined a grade III mobilisation directed to the C_{5/6} motion segment while utilising the upper limb tension test 2b (ULTT2b) and its effects on patients suffering from LE. A grade III mobilisation is described as a “large amplitude movement that moves into stiffness or muscle spasm” (Maitland, 1986, p. 95). The ULTT2b is intended to influence the neurodynamics of the radial nerve by combining shoulder girdle depression with internal rotation of the arm (Butler, 1991). This study was conducted as a cross over study on fifteen patients diagnosed with LE who each underwent a treatment protocol, a placebo and control. The outcome measures were; ULTT2b, PFGS, PPT and pain level via visual analogue scale immediately after and twenty four hours post intervention. This study was able to demonstrate manipulation induced hypoalgesia following the intervention as evident by increased PPT; increased grip strength, improved neurodynamics and reduced pain over a 24 hour period.

A study exploring the effects of HVLA technique directed to the C_{5/6} intervertebral joint found an increase in PPT at the lateral epicondyle of asymptomatic patients (Fernandez-de-las-Penas, Perez-

de-Heredia, Brea-Rivero, & Miangolarra-Page, 2007). This study recruited fifteen volunteers between the ages of 19-25 years and was conducted as a repeated measure, crossover single blind trial. The intervention conditions included a control which consisted of manual contact, a sham HVLA without any tissue tension or thrust and an HVLA directed to the C_{5/6} vertebral level. The outcome measure of this study was PPT measured at the lateral epicondyle. This study was able to demonstrate a 35.5% increase in PPT immediately after post HVLA when compared to the control group. Although this study was able to demonstrate a relatively large effect size it only utilised one outcome measure and was conducted on an asymptomatic population so the conclusions reached may have limited applications in a clinical setting.

Conclusion

Currently there is some evidence to suggest that there may be a cervical component to LE. A search of the literature was only able to find two randomised controlled trials (RCT) that explored the effects that SMT of the cervical spine has on LE (B. Vicenzino, Collins, Benson, & Wright, 1998; B. Vicenzino et al., 1996) and one RCT that explored the effects of HVLA on PPT at the elbow in an asymptomatic population (Fernandez-de-las-Penas et al., 2007). This study aimed to recruit a pathological population in order to determine the effects of HVLA on LE. The findings of this study may add to the understanding of the condition of LE and may also help inform practitioners about the appropriateness of including HVLA in the management of LE.

References

- Abbott, J. H., Patla, C. E., & Jensen, R. H. (2001). The initial effects of an elbow mobilization with movement technique on grip strength in subjects with lateral epicondylalgia. *Manual Therapy, 6*(3), 163-169.
- Allander, E. (1974). Prevalence, incidence, and remission rates of some common rheumatic diseases or syndromes. *Scandinavian journal of rheumatology, 3*(3), 145-153.
- Ashe, M. C., McCauley, T., & Khan, K. M. (2004). Tendinopathies in the upper extremity:: A paradigm shift. *Journal of Hand Therapy, 17*(3), 329-334.
- Bakker, M., & Miller, J. (2004). Does an audible release improve the outcome of a chiropractic adjustment? *JCCA. Journal Of The Canadian Chiropractic Association. Journal De L'association Chiropratique Canadienne, 48*(3), 237-239.
- Barton, N. J., Hooper, G., Noble, J., & Steel, W. M. (1992). Occupational causes of disorders in the upper limb. *British Medical Journal, 304*(6822), 309.
- Behbehani, M. M. (1995). Functional characteristics of the midbrain periaqueductal gray. *Progress in Neurobiology, 46*(6), 575-605.
- Berglund, K. M., Persson, B. H., & Denison, E. (2008). Prevalence of pain and dysfunction in the cervical and thoracic spine in persons with and without lateral elbow pain. *Manual Therapy, 13*(4), 295-299.
- Boyer, M. I., & Hastings, H. (1999). Lateral tennis elbow: "Is there any science out there?". *Journal of Shoulder and Elbow Surgery, 8*(5), 481-491.
- Bronfort, G., Haas, M., Evans, R. L., & Bouter, L. M. (2004). Efficacy of spinal manipulation and mobilization for low back pain and neck pain: a systematic review and best evidence synthesis* 1. *The Spine Journal, 4*(3), 335-356.
- Bruckner, P., & Khan, K. (1994). *Clinical sports medicine* Sydney: McGraw-Hill.
- Butler, D. (1991). *Mobilisation of the nervous system*. Melbourne: Churchill Livingstone.
- Butler, D. (2000). *The sensitive nervous system*. Adelaide: NOI Group.
- Butler, D. S. (1991). *Mobilisation of the nervous system*. Melbourne: Churchill Livingstone.
- Chesterton, L. S., Barlas, P., Foster, N. E., Baxter, G. D., & Wright, C. C. (2003). Gender differences in pressure pain threshold in healthy humans. *Pain, 101*(3), 259-266.
- Chiang, H. C., Ko, Y. C., Chen, S. S., Yu, H. S., Wu, T. N., & Chang, P. Y. (1993). Prevalence of shoulder and upper-limb disorders among workers in the fish-processing industry. *Scandinavian journal of work, environment & health, 19*(2), 126-131.
- Cleland, J. A., Whitman, J. M., & Fritz, J. M. (2004). Effectiveness of Manual Physical Therapy to the Cervical Spine in the Management of Lateral Epicondylagia: A Retrospective Analysis. *Journal of Orthopaedic and Sports Physical Therapy, 34*, 713-721.
- Cyriax, J. H. (1936). The pathology and treatment of tennis elbow. *The Journal of Bone and Joint Surgery, 18*(4), 921.
- DiGiovanna, E. L., Schiorowitz, S., & Dowling, D. J. (2005). *An osteopathic approach to diagnosis and treatment* (3rd ed.). Philadelphia: Lipincott, Williams & Wilkins.
- Fedorczyk, J. M. (2006). Tennis elbow: blending basic science with clinical practice. *Journal of Hand Therapy, 19*(2), 146-153.
- Fernandez-de-las-Penas, C., Perez-de-Heredia, M., Brea-Rivero, M., & Miangolarra-Page, J. C. (2007). Immediate effects on pressure pain threshold following a single cervical spine manipulation in healthy subjects. *The Journal Of Orthopaedic And Sports Physical Therapy, 37*(6), 325-329.
- Flynn, T. W., Fritz, J. M., Wainner, R. S., & Whitman, J. M. (2003). The audible pop is not necessary for successful spinal high-velocity thrust manipulation in individuals with low back pain. *Archives Of Physical Medicine And Rehabilitation, 84*(7), 1057-1060.
- Gellman, H. (1992). Tennis elbow (lateral epicondylitis). *The Orthopedic clinics of North America, 23*(1), 75.

- Gibbons, P., & Tehan, P. (2001). Spinal manipulation: Indications, risks and benefits. *Journal of Bodywork and Movement Therapies*, 5(2), 110-119.
- Gibson, S. J., & Helme, R. D. (2001). Age-related differences in pain perception and report. *Clinics in Geriatric Medicine*, 17(3), 433-456.
- Goguin, J. P., & Rush, F. (2003). Lateral epicondylitis. What is it really? *Current Orthopaedics*, 17(5), 386-389.
- Gracely, R. H., Grant, M. A. B., & Giesecke, T. (2003). Evoked pain measures in fibromyalgia. *Best Practice & Research Clinical Rheumatology*, 17(4), 593-609. Science Direct database.
- Greenman, P. (1996). *Principles of manual medicine*. Philadelphia: Williams & Wilkins.
- Gunn, C. C., & Milbrandt, W. E. (1976). Tennis elbow and the cervical spine. *Canadian Medical Association Journal*, 114(9), 803.
- Guyton, A. C., & Hall, J. E. (2000). *Textbook of Medical Physiology* (10th ed.). Philadelphia: WB Saunders Company.
- Hamilton, A., Balnave, R., & Adams, R. (1994). Grip strength testing reliability. *Journal of hand therapy: official journal of the American Society of Hand Therapists*, 7(3), 163.
- Hamilton, P. (1986). The prevalence of humeral epicondylitis: a survey in general practice. *The Journal of the Royal College of General Practitioners*, 36(291), 464.
- Hogeweg, J. A., Langereis, M. J., Bernards, A. T., Faber, J. A., & Helders, P. J. (1992). Algometry. Measuring pain threshold, method and characteristics in healthy subjects. *Scandinavian journal of rehabilitation medicine*, 24(2), 99.
- Holdcroft, A., & Jaggar, S. (2005). *Core topics in pain*. Cambridge: Cambridge university press.
- Hong, Q. N., Durand, M.-J., & Loisel, P. (2004). Treatment of lateral epicondylitis: where is the evidence? *Joint Bone Spine*, 71(5), 369-373.
- Innes, E. (1999). Handgrip strength testing: a review of the literature. *Australian Occupational Therapy Journal*, 46(3), 120-140.
- Itoh, K., Okada, K., & Kawakita, K. (2004). A proposed experimental model of myofascial trigger points in human muscle after slow eccentric exercise. *Acupuncture in Medicine*, 22(1), 2.
- Kosek, E., Ekholm, J., & Nordemar, R. (1993). A comparison of pressure pain thresholds in different tissues and body regions. Long-term reliability of pressure algometry in healthy volunteers. *Scandinavian journal of rehabilitation medicine*, 25(3), 117.
- Kraushaar, B. S., & Nirschl, R. P. (1999). Current concepts review-tendinosis of the elbow (tennis elbow). clinical features and findings of histological, immunohistochemical, and electron microscopy studies. *The Journal of Bone and Joint Surgery*, 81(2), 259.
- Labelle, H., Guibert, R., Joncas, J., Newman, N., Fallaha, M., & Rivard, C. H. (1992). Lack of scientific evidence for the treatment of lateral epicondylitis of the elbow. An attempted meta-analysis. *Journal of Bone and Joint Surgery-British Volume*, 74(5), 646.
- Maigne, J.-Y., & Vautravers, P. (2003). Mechanism of action of spinal manipulative therapy. *Joint Bone Spine*, 70(5), 336-341.
- Maitland, G. D. (1986). *Vertebral manipulation* (5th ed.). London: Butterworths.
- Maquet, D., Croisier, J.-L., Demoulin, C., & Crielaard, J.-M. (2004). Pressure pain thresholds of tender point sites in patients with fibromyalgia and in healthy controls. *European Journal of Pain*, 8(2), 111-117. Science Direct database.
- Melchior, M., Roquelaure, Y., Evanoff, B., Chastang, J. F., Ha, C., Imbernon, E., et al. (2006). Why are manual workers at high risk of upper limb disorders? The role of physical work factors in a random sample of workers in France (the Pays de la Loire study). *British Medical Journal*, 63(11), 754.
- Melzack, R., & Wall, P. D. (1967). Pain mechanisms: a new theory. *Survey of Anesthesiology*, 11(2), 89.
- Merskey, H., & Bogduk, N. (1994a). *Classification of chronic pain*: IASP press Seattle.
- Merskey, H., & Bogduk, N. (1994b). International Association for the Study of Pain. Task for on Taxonomy. Classification of chronic pain: Seattle: IASP Press.
- Palastanga, N., Field, D., & Soames, R. (2006). *Anatomy and human movement : structure and function* (5th ed.). New York: Butterworth Heinmann/Elsevier.

- Petty, N. J., & Moore, A. P. (2001). *Neuromusculoskeletal examination and assessment: a handbook for therapists* (2nd ed.). Edinburgh: Churchill Livingstone.
- Pickar, J. G. (2002). Neurophysiological effects of spinal manipulation. *The Spine Journal*, 2(5), 357-371.
- Pienimäki, T. T., Tarvainen, T. K., Siira, P. T., & Vanharanta, H. (1996). Progressive strengthening and stretching exercises and ultrasound for chronic lateral epicondylitis. *Physiotherapy*, 82(9), 522-530.
- Platzer, W. (2004). *Color atlas of human anatomy, Vol. 1: Locomotor system* (5th ed.). New York: Thieme.
- Potter, L., McCarthy, C., & Oldham, J. (2006). Algometer reliability in measuring pain pressure threshold over normal spinal muscles to allow quantification of anti-nociceptive treatment effects. *International Journal of Osteopathic Medicine*, 9(4), 113-119.
- Regan, W., World, L. E., Coonrad, R., & Morrey, B. W. (1992). Microscopic histopathology of chronic refractory lateral epicondylitis. *American journal of sports medicine*, 20(6), 746-749.
- Reynolds, D. V. (1969). Surgery in the rat during electrical analgesia induced by focal brain stimulation. *Science*, 164(3878), 444.
- Sanders, G. E., Reinert, O., Tepe, R., & Maloney, P. (1990). Chiropractic adjustive manipulation on subjects with acute low back pain: visual analog pain scores and plasma beta-endorphin levels. *Journal of Manipulative and Physiological Therapeutics*, 13(7), 391.
- Scher, D. L., & Wolf, M. (2009). Lateral epicondylitis. *Orthopedics*, 32(4), 276-282.
- Schmid, A., Brunner, F., Wright, A., & Bachmann, L. M. (2008). Paradigm shift in manual therapy? Evidence for a central nervous system component in the response to passive cervical joint mobilisation. *Manual Therapy*, 13(5), 387-396.
- Siddall, P. J., & Cousins, M. J. (1997). Spinal pain mechanisms. *Spine*, 22(1), 98.
- Slater, H., Arendt-Nielsen, L., Wright, A., & Graven-Nielsen, T. (2006). Effects of a manual therapy technique in experimental lateral epicondylalgia. *Manual Therapy*, 11(2), 107-117.
- Sluka, K. A. (1996). Pain mechanisms involved in musculoskeletal disorders. *The Journal of orthopaedic and sports physical therapy*, 24(4), 240.
- Smidt, N. (2001). Conservative Treatments for Tennis Elbow in Primary Care [dissertation]. *Amsterdam, the Netherlands: Vrije Universiteit*.
- Smidt, N., van der Windt, D. A. W. M., Assendelft, W. J. J., Devillé, W. L. J. M., Korthals-de Bos, I. B. C., & Bouter, L. M. (2002). Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. *The Lancet*, 359(9307), 657-662.
- Stasinopoulos, D., & Johnson, M. I. (2006). 'Lateral elbow tendinopathy' is the most appropriate diagnostic term for the condition commonly referred-to as lateral epicondylitis. *Medical Hypotheses*, 67(6), 1399-1401.
- Stratford, P. W., Norman, G. R., & McIntosh, J. M. (1989). Generalizability of grip strength measurements in patients with tennis elbow. *Physical therapy*, 69(4), 276.
- Strong, J., Unruh, A. M., Wright, A., & Baxter, G. D. (2002). *Pain: a textbook for therapists*. Edinburgh: Churchill Livingstone.
- Turk, D. C., & Melzack, R. (2001). *Handbook of pain assessment*: The Guilford Press.
- Vatine, J. J., Shapira, S. C., Magora, F., Adler, D., & Magora, A. (1993). Electronic pressure algometry of deep pain in healthy volunteers. *Archives Of Physical Medicine And Rehabilitation*, 74(5), 526-530.
- Vaughan, B., McLaughlin, P., & Gosling, C. (2007). Validity of an electronic pressure algometer. *International Journal of Osteopathic Medicine*, 10(1), 24-28. Science Direct database.
- Vernon, H. T., Dhimi, M. S., Howley, T. P., & Annett, R. (1986). Spinal manipulation and beta-endorphin: a controlled study of the effect of a spinal manipulation on plasma beta-endorphin levels in normal males. *Journal of Manipulative and Physiological Therapeutics*, 9(2), 115.

- Vicenzino, B. (2003). Lateral epicondylalgia: a musculoskeletal physiotherapy perspective. *Manual Therapy, 8*(2), 66-79.
- Vicenzino, B., Collins, D., Benson, H., & Wright, A. (1998). An investigation of the interrelationship between manipulative therapy-induced hypoalgesia and sympathoexcitation. *Journal Of Manipulative And Physiological Therapeutics, 21*(7), 448-453.
- Vicenzino, B., Collins, D., & Wright, A. (1996). The initial effects of a cervical spine manipulative physiotherapy treatment on the pain and dysfunction of lateral epicondylalgia. *Pain, 68*(1), 69-74.
- Vicenzino, B., Paungmali, A., Buratowski, S., & Wright, A. (2001). Specific manipulative therapy treatment for chronic lateral epicondylalgia produces uniquely characteristic hypoalgesia. *Manual Therapy, 6*(4), 205-212.
- Vicenzino, B., & Wright, A. (1996). Lateral epicondylalgia I: epidemiology, pathophysiology, aetiology and natural history. *Physical Therapy Reviews, 1*(1), 23-34.
- Ward, R. C., & Hruby, R. J. (2002). *Foundations for osteopathic medicine*: Lippincott Williams & Wilkins.
- Wright, A. (1995). Hypoalgesia post-manipulative therapy: a review of a potential neurophysiological mechanism. *Manual Therapy, 1*(1), 11-16.
- Wright, A., Thurnwald, P., O'Callaghan, J., Smith, J., & Vicenzino, B. (1994). Hyperalgesia in tennis elbow patients. *Journal of Musculoskeletal Pain, 2*(4), 83-97.
- Yaxley, G., & Jull, G. (1993). Adverse tension in the neural system: a preliminary study of tennis elbow. *Australian Journal of Physiotherapy, 39*, 15-15.

Section II: Manuscript

Introduction

Lateral epicondylalgia (LE) is generally considered to be a self limiting condition that often resolves spontaneously within 12 months (Scher & Wolf, 2009). However in many cases LE leads to significant ongoing pain and functional impairment. Since first described in 1878 numerous interventions have been advocated, but there remains a lack of sound empirical evidence to support any particular course of treatment (Bisset, Paungmali, Vicenzino, & Beller, 2005). Although the clinical presentation of LE is relatively uncomplicated there is a lack of clarity regarding the underlying pathological mechanisms (Vicenzino, 2003). Lateral epicondylalgia is commonly referred to as 'lateral epicondylitis', but this term may be misleading as it implies an inflammatory process is responsible for the symptoms associated with LE. Histological studies have often failed to find any evidence of active inflammation, particularly in the chronic stages of the condition (Kraushaar & Nirschl, 1999). A theory from within the field of manual therapy that suggests LE may not be an isolated local condition, but could in fact be associated with cervical spine dysfunction (Noteboom, Cruver, Keller, Kellogg, & Nitz, 1994).

Four studies have been conducted examining the effects of Spinal Manual Therapy (SMT) on subjects with LE. Two randomised controlled trials reported improvements in outcome measures including Pain-Free Grip Strength (PFGS), Pressure Pain Threshold (PPT) and neural tension tests in subjects with LE immediately following a specific SMT intervention directed to the cervical spine (Vicenzino, 1998; Vicenzino, 1996). The remaining two studies provided evidence that suggests the inclusion of cervical SMT to the management of LE can improve long term treatment outcomes (Gunn & Milbrandt, 1976) and accelerate rates of recovery (Cleland et al., 2004).

The previous studies have investigated the effects of passive mobilisation techniques such as lateral glide mobilisations of the cervical spine on LE. The author is not aware of any studies

investigating the effects of High-Velocity Low-Amplitude manipulation in subjects with LE. The aim of this study was to observe the immediate effects on Pressure Pain Threshold at the lateral elbow and Pain-Free Grip Strength in subjects with Lateral Epicondylalgia following a High-Velocity Low-Amplitude manipulation directed at the C_{5/6} spinal level.

Methods

Design

A randomised controlled experimental design was used to assess the immediate effect of high-velocity low-amplitude thrust manipulation of the cervical spine on pain intensity associated with lateral epicondylalgia (see figure 1.).

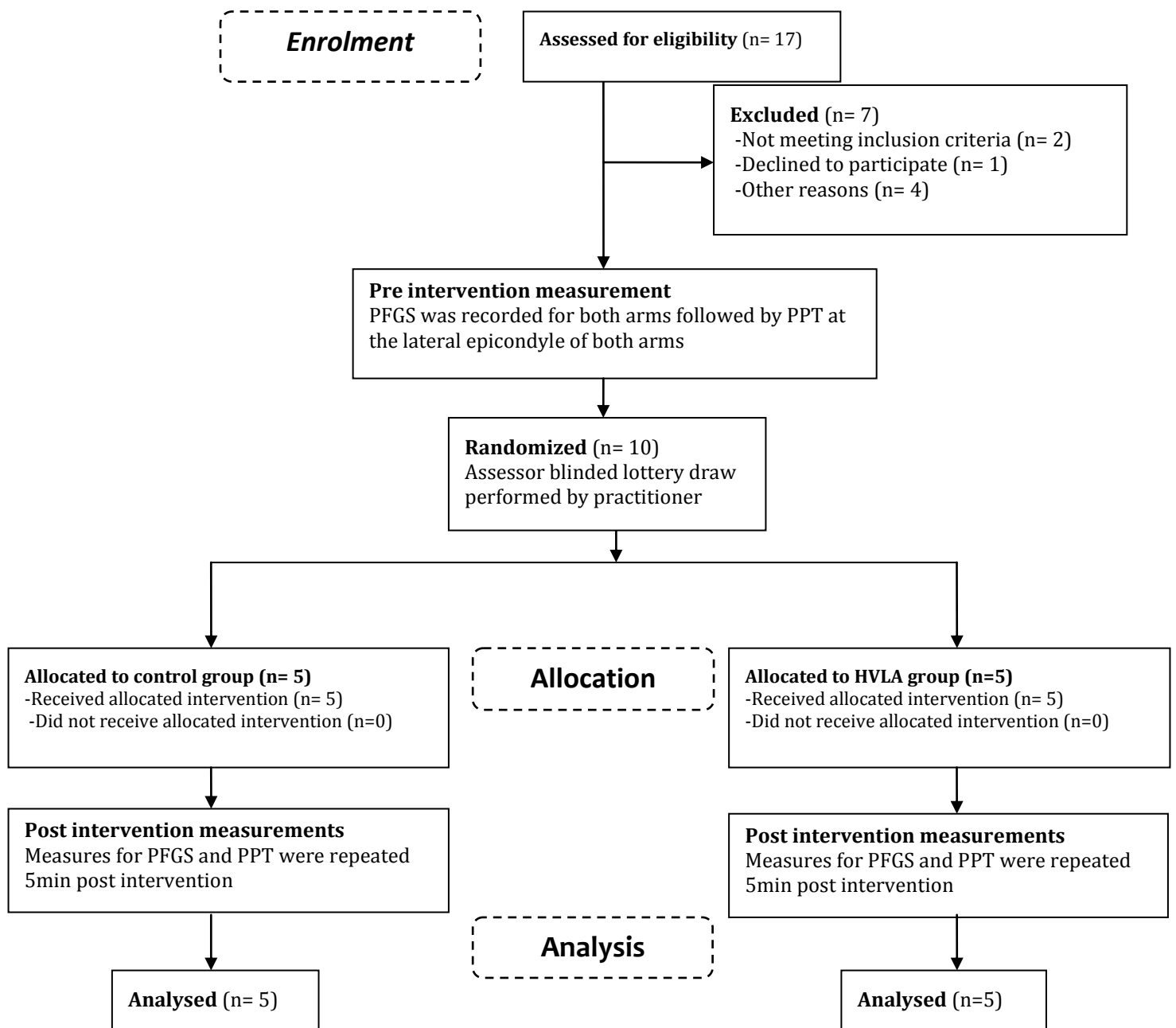


Figure 1 flowchart of study design

Abbreviations PFGS= Pain-free grip strength; PPT= Pressure pain threshold; HVLA= High-velocity low-amplitude

Subjects

Subjects were recruited by distributing notices around the Unitec campus and local racket sports clubs [See Appendix D] as well as targeted internet advertising and listing on a participant recruitment website.

Subjects were evaluated for inclusion in this study using the following criteria:

Inclusion

1. Elbow pain perceived as arising around the lateral epicondyle of the humerus
2. Pain that can be reproduced or increased in response to firm manual digital pressure applied to the lateral epicondyle of the humerus
3. Elbow pain that is reproduced with active resisted extension of the wrist

Exclusion

1. Presence of bilateral elbow pain
2. Previous orthopaedic or other surgery involving the soft tissues of the elbow
3. Presence of contraindications for HVLA as described by Gibbons and Tehan (2000)

Prior to participation all subjects were provided with an information sheet describing the procedure and were given the opportunity to ask questions before signing a consent form. The study was approved by the Unitec Research Ethics Committee.

Outcome measures

The immediate effects of HVLA were measured using two outcome measures: 1) pain free grip strength (PFGS); and 2) pressure pain threshold (PPT).

Pain free grip strength

Grip strength was measured using a strain-gauge isometric dynamometer (Model: MLT003/D AD Instruments, VIC, Australia) and the output was recorded on a notebook computer using Chart for Windows v5.0.1 (AD Instruments). A standardised procedure was used to obtain readings. Subjects were instructed to remain standing and hold their upper limb in a standardized position of elbow extension and forearm supination as described by Vicenzino, Collins and Wright (1996). Subjects were then instructed to squeeze the dynamometer handle with increasing intensity until the initial perception of pain at which point the subjects relaxed their grip. Prior to commencement of measurement each subject was given standardised instructions as per Radpasand and Owens (2009) [see Appendix C]. Three measurements were obtained with a 30-second interval between each attempt.

Pressure pain threshold

Pressure pain threshold has been demonstrated to be a valid and reliable tool for use in the clinical setting (Fischer, 1987). PPT was measured using a digital pressure algometer (JTECH Commander, Salt Lake City) using a 1cm diameter ceramic tip. Force was displayed digitally in increments of 0.1N and the peak force reached with repetition was recorded. Measurement of PPT was carried out in a standardised position with the subject seated, the shoulder placed in 90 degrees of abduction, and the elbow at 90 degrees flexion with the entire arm supported on a plinth. Prior to the initial measurement subjects received standardised instructions about the procedure. Subjects were instructed that when the sensation of pressure changed to one of pain they should indicate this by saying "pain"; at that moment the operator ceased applying pressure and the force was recorded. Three consecutive measures were taken at 30-second intervals at the lateral epicondyle

of both symptomatic and asymptomatic elbows. The mean of three measurements was carried forward for all subsequent analysis. Considerable care and attention was made to apply the force at a consistent rate of 4Ns^{-1} . Subjects were blinded to all readings obtained.

Experimental condition

After the initial measurements subjects were instructed to lie on a treatment plinth in a supine position while the researcher left the room. The subject was then assigned to either the treatment condition or control condition by lottery. The lottery consisted of selecting a coloured piece of paper from an opaque bag; black indicating intervention protocol and white indicating control. The draw was performed by the practitioner performing the intervention and the researcher was blinded to the allocation until data collection was completed.

Both of the conditions utilised in the study were delivered by an experienced, registered osteopath who routinely used HVLA in clinical practice.

The experimental conditions comprised one of the following:

1. Treatment condition: A high-velocity low-amplitude thrust (HVLA) applied to the ipsilateral $C_{5/6}$ segment as the subjects symptomatic elbow. The manipulation was conducted as described by Gibbons and Tehan (2000) using a 'cradle hold'. The thrust was directed towards the contralateral eye of the subject. Post intervention measures were taken 5 minutes after the intervention.
2. Control condition: The subject was instructed to lay supine in a relaxed position and the practitioner placed the index finger of the relevant hand along the articular pillar of the C_5 vertebra while holding the head and neck in a static neutral position for approximately the same duration it takes to deliver an HVLA (10 to 15 seconds).

The subject was instructed to remain in a supine position on the treatment plinth by the practitioner until the researcher returned. After a period of five minutes the researcher re-entered the room and repeated the measures for PFGS and PPT as previously described.

Reliability

A separate asymptomatic sample ($n= 4$ males; $n=7$ females) was recruited to determine the reliability of PPT measurements as used during the experiment. Intraclass correlation coefficients (ICC) were calculated and 95% confidence intervals constructed for the estimate. The ICC was used to calculate the standard error of measurement (SEM) and smallest detectable difference (SDD). The SEM represents an estimate of the extent that the measurement is likely to vary within a subject with repeated measurements (Domholdt, 2000). The SDD represents the proportion of a measure that can be attributed to error that occurs within the measurement process (Lassere et al., 2001). The SDD of PFGS used for this study was based on the findings of Smidt *et al.*(2002) who calculated an SDD of 14N for a sample ($n= 50$) with unilateral LE. The PPT measures conducted by the researcher at the lateral epicondyle were highly reliable (ICC= 0.97; 95% CI = 0.91 to 0.99). The SEM= 3.76 N/cm^2 , SDD= 10.42 N/cm^2 .

Data analysis

Each measurement was repeated three times and the mean was used for subsequent data analysis. Paired *t*-tests were used to interpret within group differences and 95% confidence intervals calculated for mean differences. The magnitudes of effect were expressed using Cohen's effect size (*d*) and interpreted according to the descriptors described by Cohen (1988) for effect size, and, for ICCs Hopkins (2002).

ICCs and 95% confidence intervals were calculated using a two-way mixed model, average measures, (ICC,3,3) for absolute agreement. The SEM was calculated using the formula ($SEM = SD \times \sqrt{(1 - ICC)}$). The SDD was calculated using the formula ($SDD = 1.96 \times \sqrt{2} \times SEM$). All statistical tests were conducted using the SPSS analysis package (SPSS v17.0, SPSS Inc, Chicago, IL).

Results

Pain free grip strength

Ten subjects with unilateral lateral elbow pain, (3 female and 7 male) with a mean age of 37.7yrs (SD=10.7) completed the study. The mean pain intensity scores for the symptomatic arm before intervention were 33mm (± 12). A comparison of within group changes in the experimental group ($n=5$) revealed a small but statistically significant ($P=0.03$, $d=0.32$) increase in the mean PFGS ($37.9 \pm 19.2N$) for the symptomatic arm immediately following HVLA (see fig. 2). Four out of five subjects in the intervention group showed an increase in PFGS exceeding the *a priori* SDD of 14N. The within group changes in PFGS of the control group ($n=5$) were not statistically significant ($p=0.13$) with an overall decrease in mean PFGS ($25.6 \pm 24.2N$). One subject in the control group registered an increase in PFGS (18N) which exceeded the SDD.

There was a large difference in the mean PFGS (148.9N) observed at the initial measurements between the control group (369.8N) and the intervention group (220.9N). The control group demonstrated a 6.9% decrease in mean grip strength post intervention while the HVLA group showed a 15.2% increase in mean PFGS. Neither group demonstrated a substantial change in maximal grip strength in their asymptomatic arm post intervention, and the control group demonstrating an increase of 0.7N ($p=0.97$) in mean grip strength and the HVLA group increasing by 0.4N ($p=0.97$).

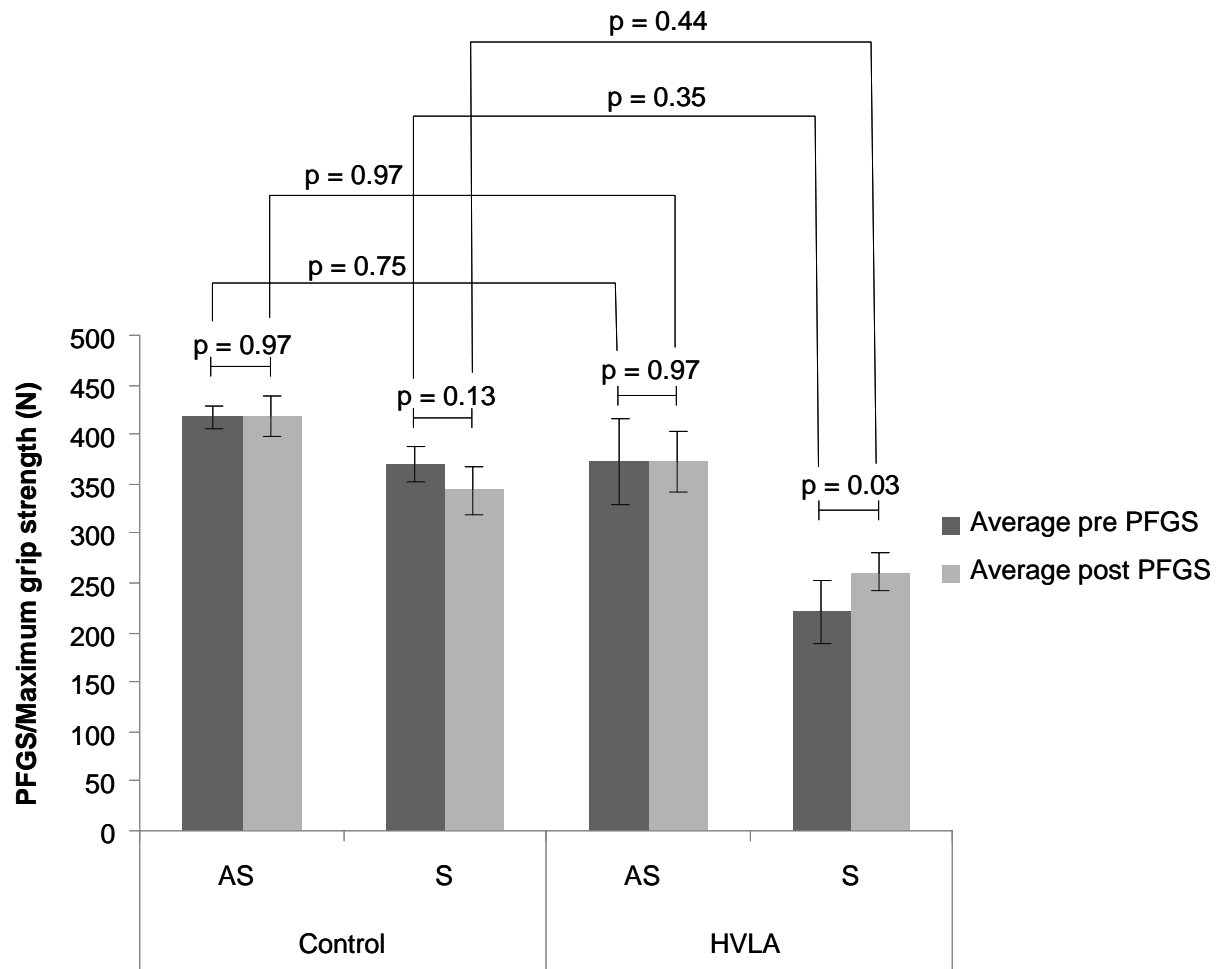


Figure 2 Mean group grip strength (n=10)

Key AS = asymptomatic arm and represents maximal grip strength. S = symptomatic arm and represents pain free grip strength. PFGS= Pain free grip strength, HVLA= High velocity low amplitude; P= p values (shown above graphs). Error bars represent ± 1SD.

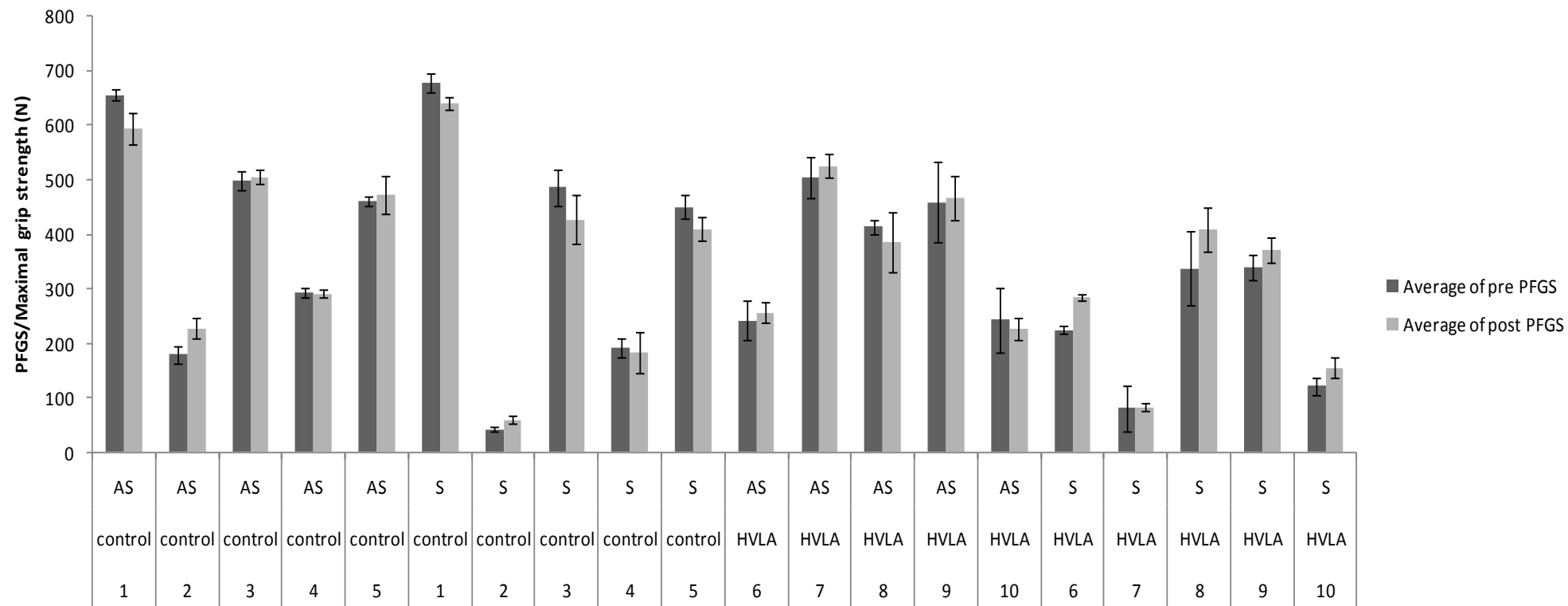


Figure 3 Mean individual grip strength.

Key AS = asymptomatic arm and represents maximal grip strength. S = symptomatic arm and represents pain free grip strength. PFGS= Pain free grip strength; HVLA= High velocity low amplitude. Error bars represent \pm 1SD. Subjects 2, 4 & 10= female subjects.

Pressure pain threshold

A comparison of changes of the mean PPT did not reveal any statistically significant change between pre and post intervention for either the control group ($p=0.59$), the intervention group ($p=0.3$) or between group ($p=0.08$). There was a mean increase in PPT of 7.8% (1.4 N/cm²) observed in the control groups' symptomatic elbow after application of the control protocol. We observed an increase of 12.3% (3.3 N/cm²) in the mean PPT of the symptomatic arm following HVLA in the intervention group. None of the changes observed in mean PPT exceeded the *a priori* SDD (10.4 N/cm²) although one individual in the control group had an increase in PPT (10.6 N/cm²) that marginally exceeded the SDD and one subject in the intervention group demonstrated an increase in PPT (14.1 N/cm²) for their asymptomatic arm.

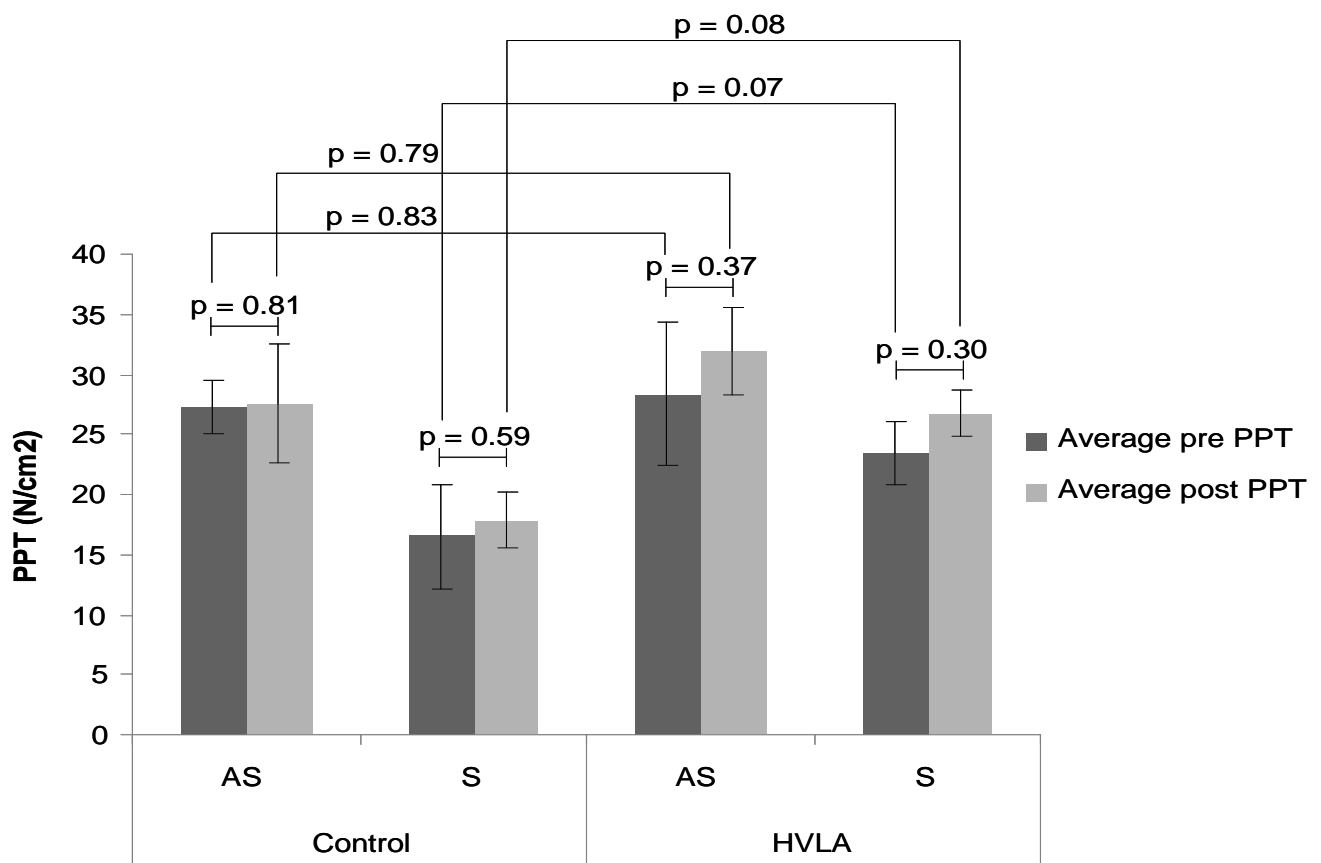


Figure 4 Mean group pressure pain thresholds

Key AS = asymptomatic arm, S = symptomatic arm, PPT= Pressure pain threshold, HVLA= High velocity low amplitude, P= p values (shown above graphs). Error bars represent $\pm 1SD$

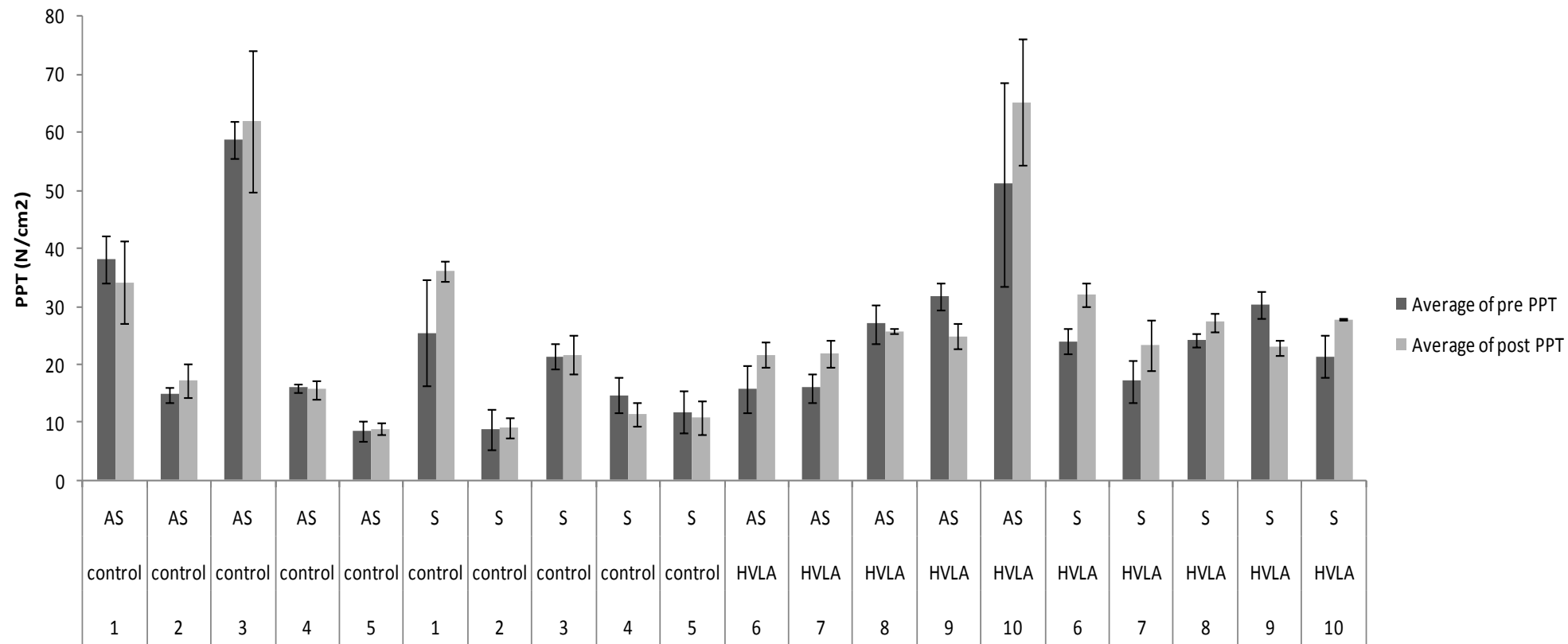


Figure 5 Mean individual pressure pain thresholds.

Key AS = asymptomatic arm. S = symptomatic arm, PPT= Pressure pain threshold, HVLA= High velocity low amplitude. Error bars represent $\pm 1SD$. Subjects 2, 4 & 10= female subjects

Discussion

Overview

Although Lateral Epicondylalgia (LE) is a common condition the mechanisms responsible for causing symptoms remain controversial. A theory exists that extrinsic factors such as referred pain from the cervical spine (Noteboom et al., 1994) or central sensitisation (Wright et al., 1994) may contribute to the symptoms experienced by patients with LE. Two studies have shown that manual therapy of the cervical spine can be beneficial for patients with lateral elbow pain (Cleland et al., 2004; Gunn & Milbrandt, 1976). This present study aimed to further investigate claims that Spinal Manual Therapy (SMT) may be useful to include in the management of LE.

The current study recruited 10 subjects with unilateral LE and used Pain-Free Grip Strength (PFGS) and Pressure Pain Threshold (PPT) as a measure of pain intensity. Five subjects were allocated to the intervention group and received High-Velocity Low- Amplitude (HVLA) thrust targeting the C_{5/6} vertebral level; the other five subjects were assigned to the control group. Pressure Pain Threshold and PFGS were recorded prior to and immediately following the application of the intervention or control protocol.

Findings

Following the application of HVLA a small (15%) but statistically significant increase in mean PFGS was reported in the intervention group (n=5) compared to a decrease (-7%) reported in the control group (n=5). The effect size in PFGS for the intervention group was small and exceeded the *a priori* Smallest Detectable Difference (SDD, 14N), which suggests that the observed change was a result of the intervention rather than measurement error.

These findings are consistent with previous studies that explored the effects of SMT on LE. Vicenzino *et al.* (1996) recruited 15 subjects with LE and found that SMT (grade III lateral glide to C_{5/6}) resulted in a statistically significant increase in mean PFGS (32N). A subsequent study using similar methods observed a 12% increase in mean PFGS for 24 subjects with LE (Vicenzino, Collins, Benson, & Wright, 1998). Increases in PFGS have also been reported when examining the effect of manual mobilisation of the elbow (Mobilisation With Movement; WMW) in subjects with LE. Abbott *et al.* (2001) and Vicenzino *et al.* (2001) both report increases in mean PFGS in subjects with LE following the application of MWM (17% and 46% respectively).

Neither the intervention nor the control group in this study showed any meaningful change post-intervention in mean Pressure Pain Threshold (PPT) as measured at the lateral epicondyle following the application of either the control (1.4N/cm²) or HVLA (3.3N/cm²). This is in contrast to findings from similar studies investigating the effects of SMT on pain in subjects with LE. One study found that HVLA to C_{5/6} leads to an immediate increase in the mean PPT of 35.5% (7.8N/cm²) as measured at the lateral epicondyle in 15 asymptomatic subjects (Fernandez-de-las-Penas *et al.*, 2007). Studies conducted by Vicenzino *et al.* (1998; 1996) that reported increases in PFGS, also reported increases in mean PPT at the lateral epicondyle (2.6N/cm² and 4.5N/cm² respectively) following SMT (grade III lateral glide to C_{5/6}). Although the findings of these studies were reported as significant, the SDDs were not calculated making interpretation of the true effect of this intervention difficult. The current study calculated the SDD for PPT measurement to be 10.4 N/cm² in asymptomatic subjects, which is similar in magnitude to the SDD (15N/cm²) calculated by Smidt *et al.* (2002) in subjects with LE. If we assume a similar SDD is appropriate to interpret the findings of Vicenzino *et al.* and Fernandez-de-las-Penas *et al.* it is difficult to conclude a meaningful change in PPT occurred following the application of the interventions they were investigating.

Pain-Free Grip Strength has been shown to be amongst the most reliable, valid and clinically relevant outcome measures to assess change in subjects with LE (P. Stratford, Lavy, & Gowland,

1993). Low pain-free grip strength is associated with LE and is a main functional impairment of the disorder. While PPT is considered a useful clinical measure to evaluate change in LE it is considered less reliable than PFGS (Trudel et al., 2004). Pain-Free Grip Strength and PPT have been used in combination to assess change in subjects with LE in numerous studies (Paungmali, O'Leary, Souvlis, & Vicenzino, 2003; Vicenzino et al., 1996; Vicenzino et al., 2001) and although many of these studies report concurrent improvements in both PPT and PFGS the real relationship between these two measures remains unclear. In this study, it is difficult to determine the extent of any correlation between PFGS and PPT due mainly to the small sample size. Simple observation of the data suggests that the general changes in PFGS are similarly reflected in changes in PPT. However when considering individual data, 5 out of 20 measurements showed instances where PFGS and PPT changed in opposite directions (see fig. 3 & fig. 5: subjects 1, 3, 9 & 10). Interestingly, two subjects (1 & 10) in this study recorded a change in PPT that was greater than the SDD. Both of these subjects showed a decrease in PFGS. Neither subject 1 or 10 demonstrated a change that was expected, subject 1 was in the control group and subject 10 demonstrated change in the asymptomatic arm, which suggests that the changes are due to variations in the measurement process. Although both PFGS and PPT claim to assess pain it is important to consider that changes in PFGS may not be attributable to pain alone (Tuomo T. Pienimäki, Kauranen, & Vanharanta, 1997). A variety of sensory motor changes have been associated with LE including changes in muscle recruitment and activation as measure by electromyography (Kelley, Lombardo, Pink, Perry, & Giangarra, 1994) and changes in reaction time (Bisset, Russell, Bradley, Ha, & Vicenzino, 2006). Although there may be a relationship between PFGS and PPT when assessing change in LE it is important to be aware change can occur independently in either measure particularly as pain is not the only determinant factor that can be used to explain changes in PFGS.

Limitations of study

The small sample size in this study limits the usefulness and generalisability of the findings. Initial calculations indicated that 20 subjects would be required in order to adequately power the study. Although recruitment was conducted over a period of 10 months, the rate of recruitment was very slow and only 10 subjects completed data collection. Due to time constraints the decision was made to cease recruitment and analyse the available data. In retrospect, it would have been useful to modify the design of the study in order to maximise the data that could be obtained with a small sample. For example, a crossover study would have effectively doubled the data provided by each subject and may have provided an effective means to improve the power of this study. Due to the fact that the average rate of recruitment for this study was one subject per month it is recommended that any future studies conducted on a population with LE allow sufficient time for recruitment. Based on this the rate of recruitment for this study it is suggested that 20 months would be an appropriate time frame to provide a sufficient sample size.

Placebo effects may have had a greater contribution to the changes noted with the intervention group than with the control group. The 'popping' sound that often accompanies HVLA techniques is often considered to be an indication of effectiveness by both practitioner and/or subject (Bakker & Miller, 2004; Maigne & Vautravers, 2003). Unfortunately, it is difficult to control for this factor in a manipulation study. It is not clear if the control protocol used in the current study provided an adequate sham for treatment. It is proposed that a control protocol should present a credible treatment alternative. If the control is not perceived as credible it may alter subject expectations, which can then lead to unpredictable effects on the outcome of the study (Licciardone & Russo, 2006). The credibility of the sham can be assessed by asking subjects if they were aware of which condition they were allocated to. It is recommended that any future studies should incorporate a post intervention follow up question to determine the suitability of any sham used. In addition, the information sheet provided to subjects describes the intervention being investigated in this study

[see Appendix D] therefore may be possible that some subjects guessed which group to which were assigned. Informing patients of the conditions of the experiment may establish a degree of expectation within subjects particularly if they already are familiar with the intervention that is being investigated. A possible solution to this issue, if further studies are conducted, is to assign subjects to groups prior to supplying information and then provide information that is specific to each group.

The non-homogeneous nature of the sample obtained for this study provides another limitation due to the additional degree of variability that occurs as a result. There are documented differences for both PPT (Chesterton et al., 2003) and grip strength (Werle et al., 2009) associated with gender as well as age. The current study grouped male and female subjects together for the purpose of data analysis. Standard deviations of both outcome measures were relatively small, which suggests an acceptable degree of variability for the subjects in the current study. It would be preferable if future studies recruited a more homogenous sample consisting of only one gender and a more specific age range in order to limit the number of variables associated with the outcome measures.

There was a marked difference in base line measurements of both PFGS and PPT between the control and intervention groups meaning it is difficult to make any comparison of between group changes that may have occurred. This disparity is not surprising given the small sample size of this study. It is likely that a larger sample size would provide a more comparable baseline measurement for PPT and PFGS.

Calculations for Interclass Correlation Coefficients (ICC), Standard Error of Measurement (SEM) and Smallest Detectable Difference (SDD) for PPT were performed using an asymptomatic population. For any future studies it may be useful to calculate ICC, SEM and SDD by adding an extra set of baseline measurements. This approach would provide data that is specific not only to the sample population being studied but also to the researcher conducting the measurements. This would offer a very specific means of interpreting the magnitude of effect beyond SDD.

Currently there is no documented Minimal Clinically Important Difference (MCID) for PFGS and PPT that is specific to LE. The absence of an MCID for PFGS and PPT relating to LE makes it difficult to interpret if the changes observed in PFGS provide any indication of possible therapeutic benefit. Until an MCID for PFGS and PPT is established for LE it may be necessary to interpret potential therapeutic relevance of interventions based on past clinical observations. A common clinical paradigm asserts that for a pain relieving technique to be considered therapeutically effective it should reduce pain by 50% during provocative tasks (Vicenzino, Smith, Cleland, & Bisset, 2009). For example, advocates of patellar taping for management of patellofemoral pain syndrome state that taping needs to decrease the patient's pain by 50% immediately following the application of tape to be effective (Crossley, Cowan, Bennell, & McConnell, 2000). If this paradigm is indicative of a MCID for PFGS and PPT specific to LE then the changes in pain intensity arising from SMT appear to fall well short of what is required to achieve meaningful therapeutic benefits.

Generalisability of study

Due to a lack of power it is difficult to draw any meaningful conclusions regarding the effects HVLA has on the outcome measures. The change observed in PFGS following HVLA does suggest that further investigation is warranted, particularly in light of the findings of previous similar studies.

The current study did not consider the long term effects that this intervention may have and therefore any implications for clinical practices are limited. Thus far only two studies have explored the effect cervical SMT has on the long term outcomes of LE (Cleland et al., 2004; Gunn & Milbrandt, 1976). Although the available studies suggest that SMT is a useful method of managing LE it does not appear that immediate changes resulting from an intervention can be used to infer improvements in long term outcomes. Vicenzino *et al.* (2009) evaluated a number of factors including PFGS during and immediately following treatment (MWM) as possible predictors of long term outcomes in a sample ($n=64$) with LE. Their findings suggest that an increase in PFGS does not offer a reliable indication of favourable long term treatment outcomes relating to LE. It is worth

considering however that changes in PFGS resulting from SMT and manipulation with movement may be due to different mechanisms and therefore may offer differing degrees of predictive values.

Because this study investigated only one specific technique rather than overall management of LE it is not directly applicable to clinical practice. Although it is useful to investigate the contribution individual techniques have, in clinical practice techniques are not used in isolation and therefore need to be considered in the context of a wider management plan. There are a wide variety of techniques available for therapists to incorporate in the management of LE. Ideally therapists should use evidence to inform them which interventions are the most appropriate to achieve the best outcomes for their patients. The outcomes that should be considered for LE include reduction of pain, improved function, improved tendon integrity and resumption of regular activities (Trudel et al., 2004). This current study suggests that HVLA may prove useful in reducing pain but more research is required to understand how useful it is in the context of an overall management plan for LE.

Future studies

In the absence of a MCID for PFGS and PPT specific to LE it is difficult to determine whether the changes observed in PFGS in this study are clinically relevant. One possible way of inferring clinical relevance is to consider the extent to which this intervention is able to return sufferers of LE towards 'normal function'. This could be approached a number of ways. For example, if a large enough sample is obtained the mean grip strength of the asymptomatic arm may provide an indication of normal grip strength, in individuals the asymptomatic arm could be used as a benchmark for normal function for that individual. A number of factors would need to be taken into consideration in order to apply these measures such as variation in grip strength due to age and gender as well as differences between the dominant and non-dominant arm.

Another possible solution is to use normative data to assess the degree of change. For instance, applying these methods using subject 8 as an example suggests that HVLA is capable of restoring normal grip strength in the short term. Subject 8's average grip strength in the asymptomatic arm was 399N compared to the interventions group average grip strength of 372N. Following HVLA subject 8's grip strength increased from 337N to 409N. A comparison to age and gender specific normative grip strength data for a Swiss population ($n=978$) shows that prior to intervention subject 8 was below the minimum normative grip strength of 373N, immediately following HVLA the subjects grip strength moved towards normal (536N) (Werle et al., 2009). Unfortunately it is difficult to apply this example to the mean changes observed in PPT and PFGS in this study due to the non-homogenous nature of the sample obtained.

A number of different interventions have been reported to influence PFGS. It may be useful for future studies to compare the effects that different manual techniques such as MWM and HVLA have on PFGS in subjects with LE.

No assessment of the cervical spine was conducted on subjects in this study. Berglund, Persson, & Denison (2008) reported an association with the presence of LE and findings of increased pain on palpation and decreased active range of motion in the cervical and thoracic spine. Future studies could incorporate an assessment of the cervical spine in order to establish if any meaningful relationship between LE and cervical dysfunction exists and the implications that the presence of findings may have on the outcome of any interventions directed to the cervical spine.

Conclusion

Based on the findings of this study, it appears that High-Velocity Low-Amplitude thrust targeting the C_{5/6} vertebral segment can lead to a moderate increase in Pain-Free Grip Strength in subjects with LE, (i.e. a real improvement in function). However, this study does not determine if there are

any beneficial effects beyond this immediate change. Although further study is needed, these findings suggest that cervical HVLA may be a useful technique to incorporate in the management of symptoms of LE such as reduced grip strength. In addition, it is not clear whether HVLA is treating the cause of LE or simply managing symptoms. If the former is true, it might be more likely to expect that longer term benefits may result after treatment. Although PFGS is intended to measure pain, other factors contribute to change in PFGS. It is likely that PFGS offers a better indication of a patient's functional improvement, while PPT is more applicable for clinical measures than it is for measuring functional change. The change observed in PFGS following HVLA in this study justifies further work exploring the proposed cervical component of LE. This study while limited in power and application may be a useful addition to future meta analysis.

References

- Abbott, J. H., Patla, C. E., & Jensen, R. H. (2001). The initial effects of an elbow mobilization with movement technique on grip strength in subjects with lateral epicondylalgia. *Manual Therapy, 6*(3), 163-169.
- Bakker, M., & Miller, J. (2004). Does an audible release improve the outcome of a chiropractic adjustment? *JCCA. Journal Of The Canadian Chiropractic Association. Journal De L'association Chiropratique Canadienne, 48*(3), 237-239.
- Berglund, K. M., Persson, B. H., & Denison, E. (2008). Prevalence of pain and dysfunction in the cervical and thoracic spine In persons with and without lateral elbow pain. *Manual Therapy, 13*(4), 295-299.
- Bisset, L., Paungmali, A., Vicenzino, B., & Beller, E. (2005). A systematic review and meta-analysis of clinical trials on physical interventions for lateral epicondylalgia. *British journal of sports medicine, 39*(7), 411.
- Bisset, L. M., Russell, T., Bradley, S., Ha, B., & Vicenzino, B. T. (2006). Bilateral sensorimotor abnormalities in unilateral lateral epicondylalgia. *Archives Of Physical Medicine And Rehabilitation, 87*(4), 490-495.
- Chesterton, L. S., Barlas, P., Foster, N. E., Baxter, G. D., & Wright, C. C. (2003). Gender differences in pressure pain threshold in healthy humans. *Pain, 101*(3), 259-266.
- Cleland, J. A., Whitman, J. M., & Fritz, J. M. (2004). Effectiveness of Manual Physical Therapy to the Cervical Spine in the Management of Lateral Epicondylalgia: A Retrospective Analysis. *Journal of Orthopaedic and Sports Physical Therapy, 34*, 713-721.
- Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*: Lawrence Erlbaum.
- Crossley, K., Cowan, S. M., Bennell, K. L., & McConnell, J. (2000). Patellar taping: is clinical success supported by scientific evidence? *Manual Therapy, 5*(3), 142-150.
- Domholdt, E. (2000). *Physical therapy research: principles and applications*: Saunders Philadelphia.
- Fernandez-de-las-Penas, C., Perez-de-Heredia, M., Brea-Rivero, M., & Miangolarra-Page, J. C. (2007). Immediate effects on pressure pain threshold following a single cervical spine manipulation in healthy subjects. *The Journal Of Orthopaedic And Sports Physical Therapy, 37*(6), 325-329.
- Fischer, A. A. (1987). Pressure algometry over normal muscles. Standard values, validity and reproducibility of pressure threshold. *Pain, 30*(1), 115-126.
- Gibbons, P., & Tehan, P. (2000). *Manipulation of the spine, thorax and pelvis*. London: Harcourt publishers Ltd.
- Gunn, C. C., & Milbrandt, W. E. (1976). Tennis elbow and the cervical spine. *Canadian Medical Association Journal, 114*(9), 803.
- Hopkins, W. G. (2002). A scale of magnitudes for effect statistics [Electronic version]. *A new view of statistics: Internet society for sports science*.
- Kelley, J. D., Lombardo, S. J., Pink, M., Perry, J., & Giangarra, C. E. (1994). Electromyographic and cinematographic analysis of elbow function in tennis players with lateral epicondylitis. *The American Journal of Sports Medicine, 22*(3), 359.
- Kraushaar, B. S., & Nirschl, R. P. (1999). Current concepts review-tendinosis of the elbow (tennis elbow). clinical features and findings of histological, immunohistochemical, and electron microscopy studies. *The Journal of Bone and Joint Surgery, 81*(2), 259.
- Lassere, M. N., van der Heijde, D., Johnson, K., Bruynesteyn, K., Molenaar, E., Boonen, A., et al. (2001). Robustness and generalizability of smallest detectable difference in radiological progression. *The Journal of Rheumatology, 28*(4), 911.
- Licciardone, J. C., & Russo, D. P. (2006). Blinding protocols, treatment credibility, and expectancy: methodologic issues in clinical trials of osteopathic manipulative treatment. *JAOA: Journal of the American Osteopathic Association, 106*(8), 457.

- Maigne, J.-Y., & Vautravers, P. (2003). Mechanism of action of spinal manipulative therapy. *Joint Bone Spine*, 70(5), 336-341.
- Noteboom, T., Cruver, R., Keller, J., Kellogg, B., & Nitz, A. J. (1994). Tennis elbow: a review. *The Journal of orthopaedic and sports physical therapy*, 19(6), 357.
- Pienimäki, T. T., Kauranen, K., & Vanharanta, H. (1997). Bilaterally decreased motor performance of arms in patients with chronic tennis elbow. *Archives Of Physical Medicine And Rehabilitation*, 78(10), 1092-1095.
- Radpasand, M., & Owens, E. (2009). Combined multimodal therapies for chronic tennis elbow: pilot study to test protocols for a randomized clinical trial. *Journal of manipulative and physiological therapeutics*, 32(7), 571-585.
- Scher, D. L., & Wolf, M. (2009). Lateral epicondylitis. *Orthopedics*, 32(4), 276-282.
- Smidt, N., van der Windt, D. A., Assendelft, W. J., Mourits, A. J., Devillé, W. L., de Winter, A. F., et al. (2002). Interobserver reproducibility of the assessment of severity of complaints, grip strength, and pressure pain threshold in patients with lateral epicondylitis. *Archives of Physical Medicine and Rehabilitation*, 83(8), 1145-1150.
- Stratford, P., Lavy, D., & Gowland, C. (1993). Evaluative properties of measures used to assess patients with lateral epicondylitis at the elbow. *Physiotherapy Canada*, 45, 160-160.
- Trudel, D., Duley, J., Zastrow, I., Kerr, E. W., Davidson, R., & MacDermid, J. C. (2004). Rehabilitation for patients with lateral epicondylitis: a systematic review. *Journal of Hand Therapy*, 17(2), 243-266.
- Vicenzino, B. (2003). Lateral epicondylalgia: a musculoskeletal physiotherapy perspective. *Manual Therapy*, 8(2), 66-79.
- Vicenzino, B., Collins, D., Benson, H., & Wright, A. (1998). An investigation of the interrelationship between manipulative therapy-induced hypoalgesia and sympathoexcitation. *Journal Of Manipulative And Physiological Therapeutics*, 21(7), 448-453.
- Vicenzino, B., Collins, D., & Wright, A. (1996). The initial effects of a cervical spine manipulative physiotherapy treatment on the pain and dysfunction of lateral epicondylalgia. *Pain*, 68(1), 69-74.
- Vicenzino, B., Paungmali, A., Buratowski, S., & Wright, A. (2001). Specific manipulative therapy treatment for chronic lateral epicondylalgia produces uniquely characteristic hypoalgesia. *Manual Therapy*, 6(4), 205-212.
- Vicenzino, B., Smith, D., Cleland, J., & Bisset, L. (2009). Development of a clinical prediction rule to identify initial responders to mobilisation with movement and exercise for lateral epicondylalgia. *Manual Therapy*, 14(5), 550-554.
- Werle, S., Goldhahn, J., Drerup, S., Simmen, B. R., Sprott, H., & Herren, D. B. (2009). Age- and gender-specific normative data of grip and pinch strength in a healthy adult Swiss population. *Journal of Hand Surgery (European Volume)*, 34(1), 76.
- Wright, A., Thurnwald, P., O'Callaghan, J., Smith, J., & Vicenzino, B. (1994). Hyperalgesia in tennis elbow patients. *Journal of Musculoskeletal Pain*, 2(4), 83-97.

Appendices

Appendix A: Figures

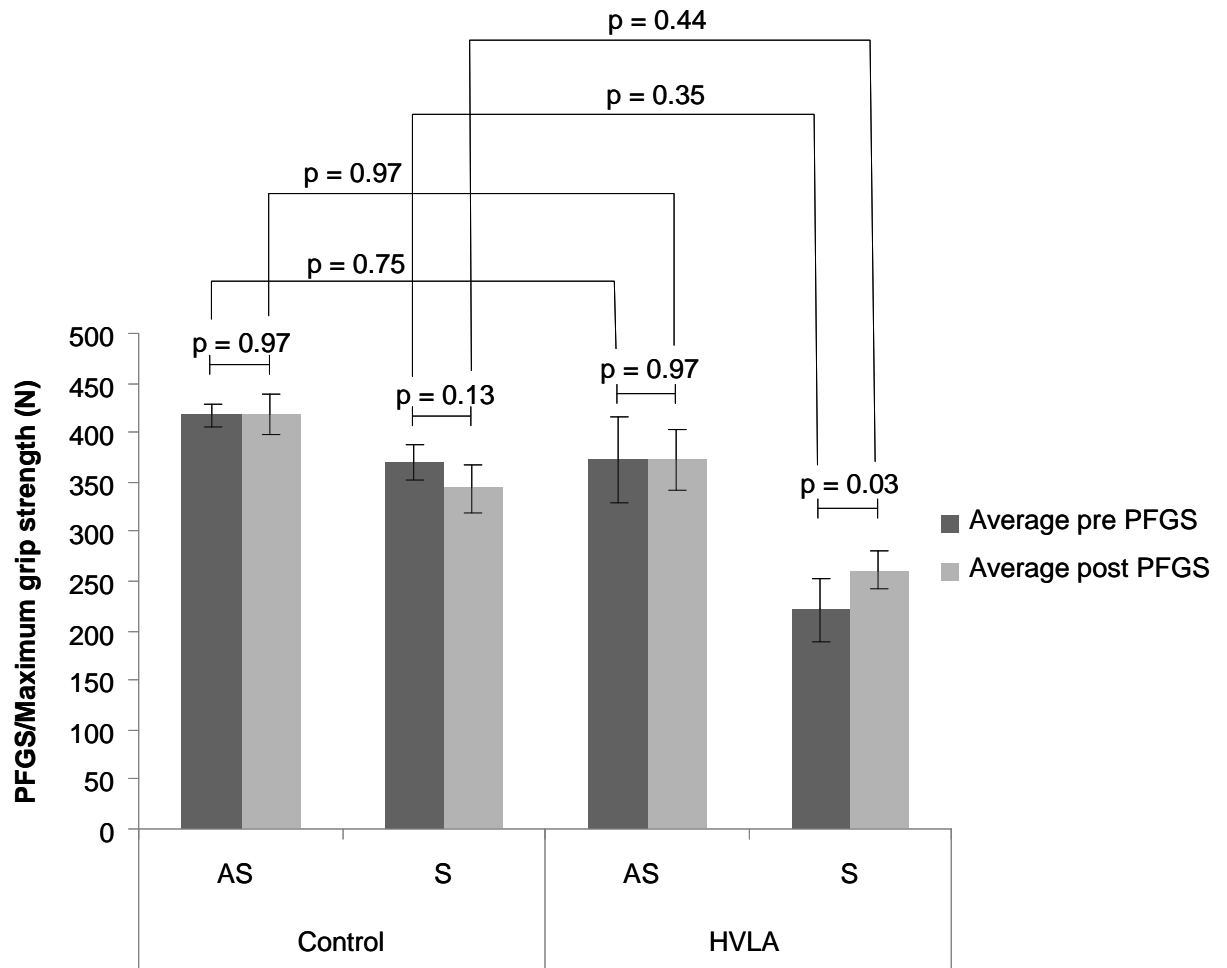


Figure 2 Mean group grip strength (n=10)

Key AS = asymptomatic arm and represents maximal grip strength. S = symptomatic arm and represents pain free grip strength. PFGS= Pain free grip strength, HVLA= High velocity low amplitude; P= p values (shown above graphs).

Error bars represent ± 1SD.

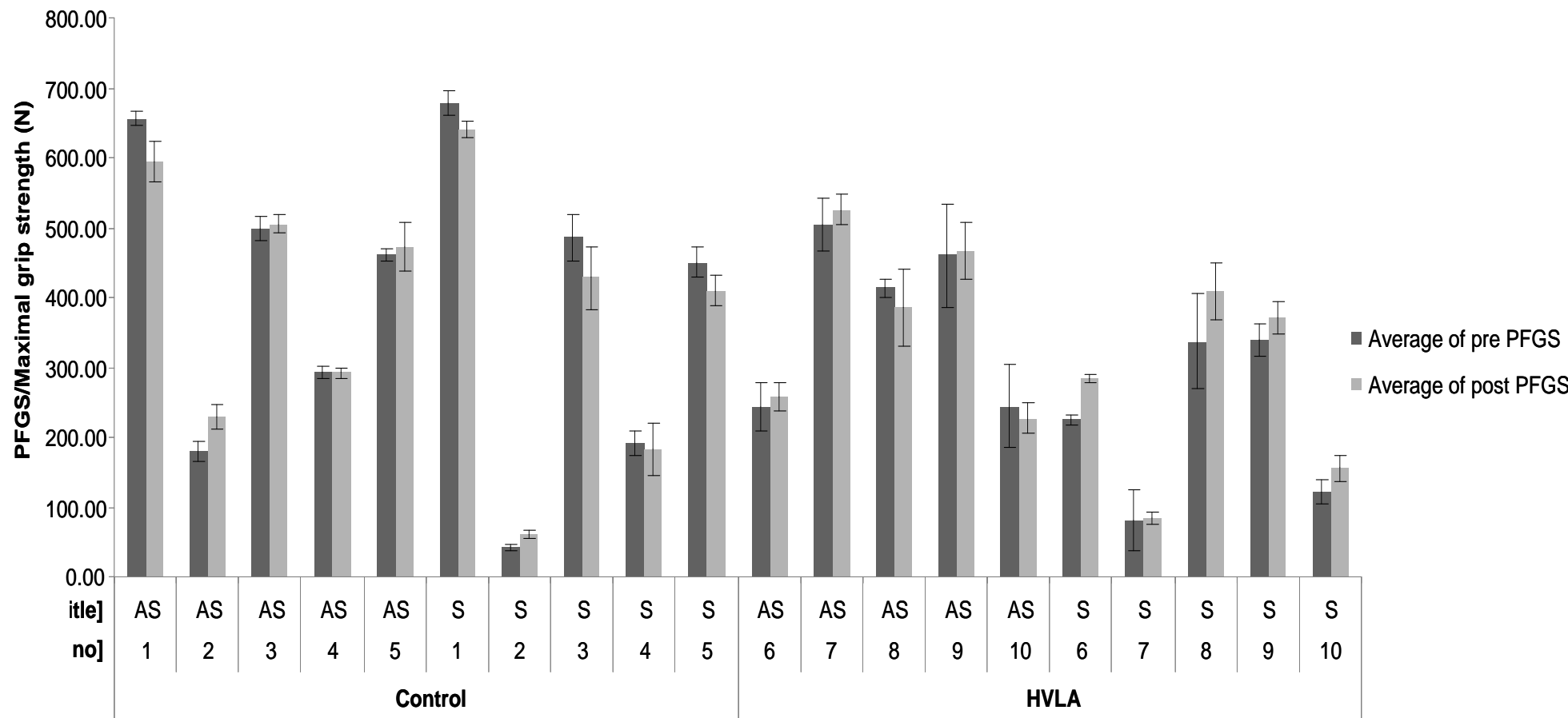


Figure 3 Mean individual grip strength.

Key AS = asymptomatic arm and represents maximal grip strength. S = symptomatic arm and represents pain free grip strength. PFGS= Pain free grip strength; HVLA= High velocity low amplitude. Error bars represent $\pm 1SD$.

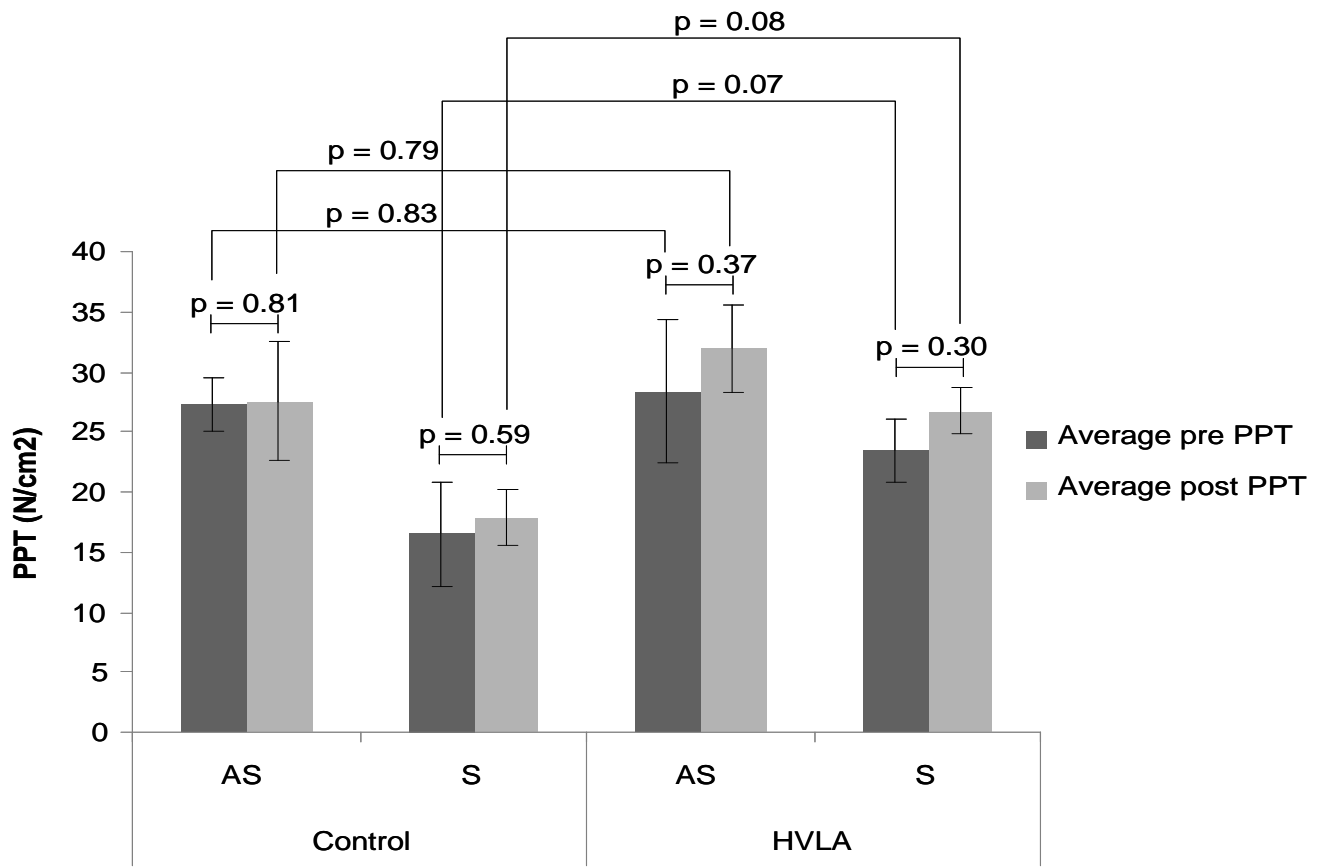


Figure 4 Mean group pressure pain thresholds
 Key AS = asymptomatic arm, S = symptomatic arm, PPT= Pressure pain threshold, HVLA= High velocity low amplitude, P= p values (shown above graphs). Error bars represent ± 1SD

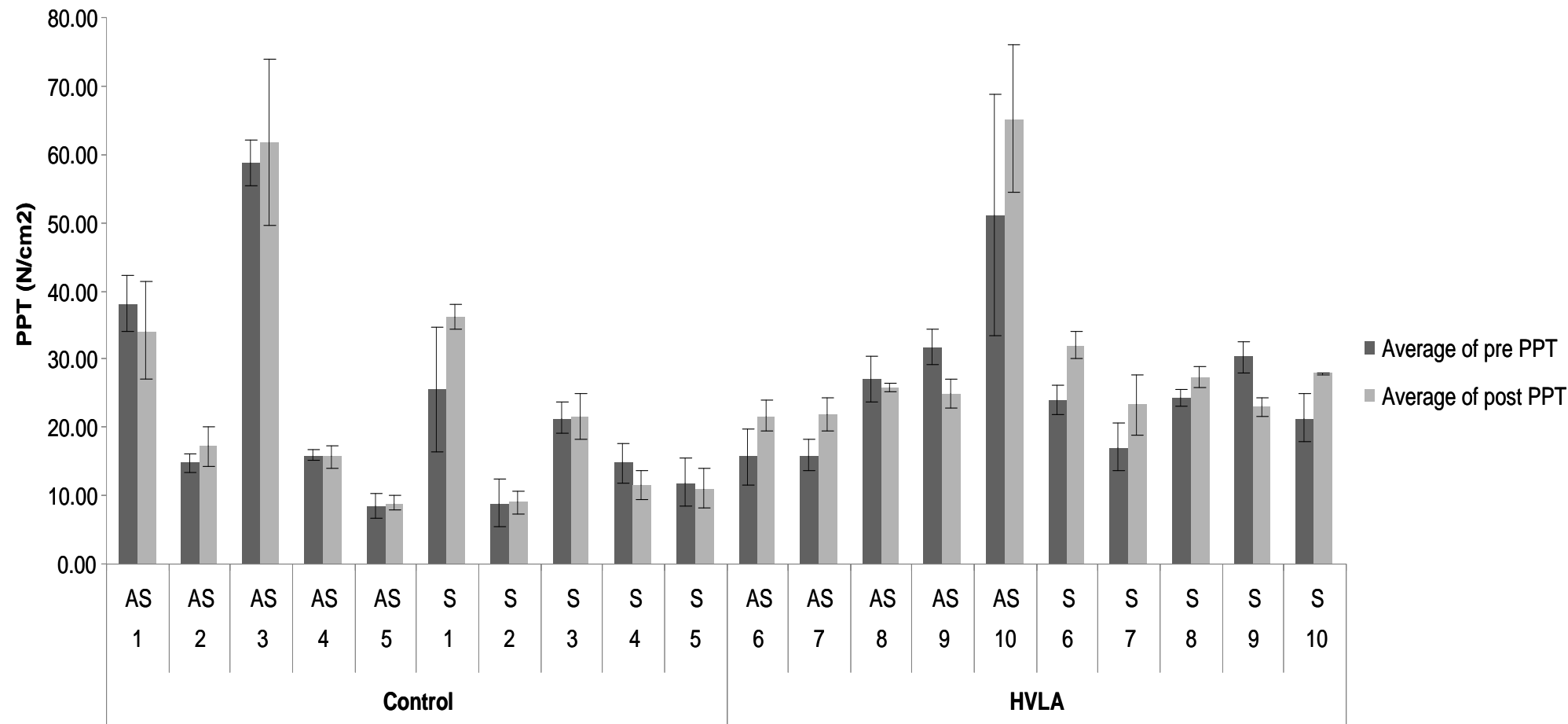


Figure 5 Mean individual pressure pain thresholds.

Key AS = asymptomatic arm. S = symptomatic arm, PPT= Pressure pain threshold, HVLA= High velocity low amplitude. Error bars represent $\pm 1SD$.

Appendix B: Raw Data

subject	group	symptom	Average of pre PPT	Average of post PPT	Average of pre PFGS	Average of post PFGS
1	control	AS	38.13	34.13	656.27	594.33
2	control	AS	14.80	17.23	179.40	228.17
3	control	AS	58.80	61.87	499.10	504.70
4	control	AS	15.97	15.67	292.33	291.53
5	control	AS	8.50	8.93	460.73	472.93
1	control	S	25.50	36.07	678.57	640.17
2	control	S	8.90	9.07	42.37	60.40
3	control	S	21.40	21.73	486.47	427.77
4	control	S	14.77	11.53	191.43	182.93
5	control	S	11.87	10.97	450.03	409.67
6	HVLA	AS	15.67	21.67	242.57	257.10
7	HVLA	AS	15.97	21.93	503.97	525.50
8	HVLA	AS	27.00	25.80	413.57	385.00
9	HVLA	AS	31.77	24.90	459.43	467.03
10	HVLA	AS	51.13	65.23	243.87	226.87
6	HVLA	S	24.03	32.07	224.83	284.60
7	HVLA	S	17.13	23.40	81.00	83.27
8	HVLA	S	24.33	27.30	337.57	408.57
9	HVLA	S	30.30	22.97	339.27	370.97
10	HVLA	S	21.40	27.83	121.67	155.83

Appendix C: Instructions for pain free Grip strength

“The purpose of this study is to test your pain-free maximum hand grip strength. You will be asked to repeat this three times with each side beginning with your (LEFT/RIGHT) side. Please hold the grip strength meter in a comfortable position and when you are ready squeeze the handle as hard as you are able to the point where your pain starts, if you do not experience pain please grip as hard as possible. After one squeeze relax your hand and I will record your measurement.” (Radpasand & Owens, 2009, p. 574)

Radpasand, M., & Owens, E. (2009). Combined multimodal therapies for chronic tennis elbow: pilot study to test protocols for a randomized clinical trial. *Journal of manipulative and physiological therapeutics*, 32(7), 571-585.

Appendix D: Ethics Resources



Immediate effects of cervical spine manipulation on pressure pain threshold and grip strength in subjects with lateral epicondylitis

Information for participants

You are invited to take part in a research project being undertaken as part of the Masters of Osteopathy Degree. The research involves investigating cervical manipulation as a means of temporarily reducing pain for those suffering from lateral epicondylitis (tennis elbow). This information sheet aims to provide information regarding the nature of this research and what will happen should you choose to participate.

The researchers

The researcher is Alastair Treacher, with supervision from Dr Craig Hilton and Rob Moran.

Who can be involved?

We are seeking participants between the ages of 18-45 years who are currently experiencing lateral elbow pain. Unfortunately, if you have pain in both elbows, have undergone previous surgery on either elbow or have a medical condition such as haemophilia or cancer you cannot be included in this research.

What participation will mean for you

Participants will attend one session lasting approximately one hour. The session will involve a brief physical examination of your elbow and a questionnaire regarding relevant medical history to ensure that you meet the inclusion criteria for this study. Following this you will be informed of what happens in the research. After you have had time to consider participating, you will then be invited to sign a consent form. Once this is done the experimental procedure will begin.

Participants will be placed in standardized position then will be asked to grip a handle and gradually squeeze until the first sensation of pain in the elbow is perceived. Following this a device will be placed on the outside of your elbow and gradual pressure will be applied until the point where pain is first experienced. Each of these tests will be repeated three times and results will be recorded. Participants will then either receive the intervention or be asked to wait for a period of approximately 15 minutes before being retested. The intervention for this study is cervical manipulation. This technique is commonly used as part of osteopathic treatment and will be carried out by an experienced osteopath.

Potential risks to participants

There are potential risks of adverse effects occurring as a result of cervical spine manipulation ranging from mild headaches and neck stiffness to stroke or spinal fractures. However, the actual risk of severe complication is considered to be extremely low (in order of between 1/400,000 and

1/10,000,000) (Coulter et al 1996). In order to minimise any potential risk a pre-treatment medical screen will be undertaken to ensure participants are suitable to proceed. If any factors that place participants at increased risk are identified manipulation of the neck will not be performed. All manipulations in this study will be carried out by an experienced osteopath.

Confidentiality:

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

- All consent forms and completed questionnaires will be seen only by researchers
- All hard copies and information will be stored in a locked file in a secure room. Only the researchers will have access to this file.
- Only anonymous data will be presented in reports relating to this research.
- Electronic files will be protected with a password
- Information gathered during this research will be held for 5 years before being destroyed.

You have the right not to participate, or to withdraw from this research project until the day of final data collection. This can be done by contacting Alastair Treacher or Dr Craig Hilton via telephone or email.

Contact:

If you require further information or have concerns please contact the researchers via phone or email.

Alastair Treacher
School of Health and Community Studies
Unitec New Zealand
Telephone (09) 940 4464
Mobile 021 166 0254
Email altreacher@hotmail.com

Dr Craig Hilton
School of Health and Community Studies
Unitec New Zealand
Telephone (09) 815 4321 ext 5194
Mobile 021 268 2076
Email chilton@unitec.ac.nz

UREC REGISTRATION NUMBER: (2009-1056)

This study has been approved by the UNITEC Research Ethics Committee from (24/03/2010) to (23/03/2011). If you have any complaints or reservations about the ethical conduct of this research, you may contact the Committee through the UREC Secretary (ph: 09 815-4321 ext 6162). Any issues you raise will be treated in confidence and investigated fully, and you will be informed of the outcome.



Immediate effects of cervical spine manipulation on pressure pain threshold and grip strength in subjects with lateral epicondylitis

Consent Form

This research project investigates the effectiveness of an osteopathic technique on reducing levels of pain experienced at the lateral elbow. The research is being undertaken by Alastair Treacher from Unitec New Zealand, and will be supervised by Dr Craig Hilton and Robert Moran.

Name of Participant:.....

I have seen the Information Sheet dated.....for people taking part in the study, titled. **“Immediate effects of cervical spine manipulation on pressure pain threshold and grip strength in subjects with lateral epicondylitis”** I have had the opportunity to read the contents of the information sheet and to discuss the project with the researcher and I am satisfied with the explanations I have been given. I understand that taking part in this project is voluntary (my choice) and that I may withdraw up until the point at which data analysis is started (approximately 10 days after the data collection session) and this will in no way affect my access to the services provided by Unitec New Zealand or any other support service.

I understand that I can withdraw from the study up until the point at which data analysis is started, if for any reason I want to do this.

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.

Signature..... Participant (Date)

Project explained by.....

Signature..... (Date)

Participant information

Subject number: _

Date: _____

NAME: _____ D.O.B. _____

Side of elbow pain (Please circle) **Left / Right**

Approximate duration of current episode: _____

Approximate intensity of pain:

(Place an X on the line below at the point that best represents the current level of your pain)

0 _____ 10

(No Pain)

(Worst pain imaginable)

Do you have any medical conditions that may put you at risk from participating in this study?

Including: Rheumatoid arthritis, osteoarthritis, osteoporosis, haemophilia, cancer.

(Please circle) **Yes / No**

Dates and outcomes:

Have you ever suffered whiplash or a fracture of the cervical spine?

(Please circle) **Yes / No**

Dates and outcomes:

Have you ever suffered from any of the following? (Circle as many as applicable)

Dizziness Blackouts Double vision Blurred vision Ringing in the ears

Facial numbness Difficulty swallowing Difficulty Speaking Nausea

Dates and outcomes:

To be completed by the researcher:

Point tenderness of lateral epicondyle: **Yes / No**

Reproduction of familiar pain with resisted extension **Yes / No**

Signs of VBI (Nystagmus, Horners syndrome, gait disturbance) **Yes / No**

Intervention **Y / N**

Dominant arm: **Left / Right**

Notes:

Pressure Pain threshold (Pre test)					
	1	2	3	Average	%CV
Left					
Right					

Pressure Pain Threshold (Post test)					
	1	2	3	Average	% CV
Left					
Right					



ARE YOU EXPERIENCING ELBOW PAIN?

Seeking volunteers to take part in an osteopathic study

We are currently undertaking a study that explores the potential of osteopathic techniques to reduce the level of pain experienced by people who suffer from tennis elbow.

Are you interested in taking part?

We are seeking participants between the ages of 18-45 years who are currently experiencing lateral elbow pain (tennis elbow).

This study is being conducted at the Unitec osteopathic clinic and will take approximately one hour.

Unfortunately you are not eligible to take part if you experience pain in both elbows or have undergone previous surgery on either elbow.



This study has been approved by the
UNITEC Research Ethics Committee
UREC REGISTRATION NUMBER: 2009-1056
24/03/2010 – 23/03/2010

**If you are interested in participating,
or would like more information
please contact:**

Alastair Treacher

P: 021 166 0254

E: altreacher@hotmail.com

