The efficacy of surface electromyographic biofeedback assisted stretching for the treatment of chronic low back pain: A case-series

Aimee M. Moore

Thesis submitted in partial fulfilment of the requirements for the degree of Masters of Osteopathy Unitec Institute of Technology, 2013
DECLARATION

Name of candidate: Aimee Moore

This Thesis entitled ‘The efficacy of surface electromyographic biofeedback assisted stretching for the treatment of chronic low back pain: A case-series’, is submitted in partial fulfilment for the requirements for the Unitec degree of Master of Osteopathy.

Candidate’s declaration

I confirm that:

- This Thesis represents my own work;

- 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: 2011-1193

Candidate Signature: ……………………..          Date: ……………………..

Student number: 1220008
ACKNOWLEDGEMENTS

I would like to thank all those who volunteered their time to be part of my project. Thank you to Martin for living this with me. Thank you also to my supervisors Rob Moran and Jamie Mannion. Thank you to friends, family and all the others who offered their time, help and support.
**PREFACE**

This thesis is submitted in partial fulfilment of the requirements for the Masters of Osteopathy degree at Unitec Institute of Technology.

The following thesis is divided into three sections:

1. The literature review, with emphasis on:
   - Low back pain prevalence and its impact on society
   - Chronic low back pain and its classification
   - Investigation of Flexion Relaxation mechanisms, a common neuromuscular phenomenon which is predictably absent in individuals with back pain
   - Low back pain treatment effects on absent Flexion Relaxation
   - The effect of surface electromyography assisted stretching programmes in affecting Flexion Relaxation in individuals with chronic low back pain.

2. A manuscript in the format specified for submission to the *Journal of Bodywork and Movement Therapies*, investigating the effects of a surface electromyography assisted stretching programme on impaired Flexion Relaxation, pain, disability and range of motion in individuals with chronic low back pain.

3. Appendices including ethical approval, participant information sheets, consent forms, questionnaires, additional results information and the guidelines for authors to the *Journal of Bodywork and Movement Therapies*. 
**THESIS ABSTRACT**

Chronic low back pain is a major problem to both the individual and society. The negative impact of Chronic Low back pain (LBP) includes its large direct and indirect treatment costs, and associated disability and suffering. Low back pain is discussed in the literature review, including information of the various models of diagnosis and classification. As it is well recognised that chronic pain is a multidimensional issue, models of mechanical, neurological and biopsychosocial influences are presented. Common motor control impairments are briefly explored.

Flexion Relaxation (FR) is a commonly observed muscle pattern of lumbar paraspinal relaxation (electrical silence) near the end range of flexion. Flexion Relaxation is observable to some degree in most asymptomatic individuals. Impaired FR, displayed as continued muscle activation at maximal voluntary flexion (MVF), is commonly identified in those with chronic LBP.

The study presented in section two of the thesis, investigated the effects of a surface electromyographic assisted stretching (SEMGAS) programme on impaired FR patterns in individuals with chronic LBP. Nine volunteers with chronic LBP that displayed impaired FR were recruited from the general public, and took part in a biofeedback SEMGAS intervention including an at-home stretching component over five weeks. Outcome measures included FR, Oswestry Disability Index, Numeric Pain Rating Scale and Sit and Reach, and were recorded pre and post-intervention as well as at a four to six-week follow-up. The aim was to investigate if improved FR is associated with improved range of motion, pain intensity and disability in individuals with chronic LBP. Of the nine participants included, three improved FR to statistically significant levels. All three also achieved a clinically important change in pain intensity scores. The results suggest SEMGAS may provide benefits to some individuals with chronic LBP and impaired FR, although larger scale investigation of SEMGAS as a unimodal therapy in a larger population is indicated.
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<th>Description</th>
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<tbody>
<tr>
<td>AUD</td>
<td>Australian Dollars</td>
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<tr>
<td>CI</td>
<td>Confidence Interval (95% unless stated otherwise)</td>
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<tr>
<td>dB</td>
<td>Decibel (unit)</td>
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<tr>
<td>FIR</td>
<td>Finite Infinite Response (filter)</td>
</tr>
<tr>
<td>FR</td>
<td>Flexion Relaxation</td>
</tr>
<tr>
<td>kHz</td>
<td>Kilohertz (unit)</td>
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<td>LBP</td>
<td>Low Back Pain</td>
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<td>M</td>
<td>Median</td>
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<td>MCID</td>
<td>Minimum Clinically Important Difference</td>
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<td>mm</td>
<td>Millimetre</td>
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<td>MVF</td>
<td>Maximal Voluntary Flexion</td>
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<td>NPRS</td>
<td>Numeric Pain Rating Scale</td>
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<td>NZD</td>
<td>New Zealand Dollars</td>
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<td>ODI</td>
<td>Oswestry Disability Index</td>
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<td>p</td>
<td>Statistical Probability</td>
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<td>PGIC</td>
<td>Patient Global Impression of Change</td>
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<td>Cohens d</td>
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<td>RMS</td>
<td>Root Mean Square</td>
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<td>Range of Motion</td>
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<td>SEMG</td>
<td>Surface Electromyography</td>
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<td>SEMGAS</td>
<td>Surface Electromyography Assisted Stretching</td>
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<td>SD</td>
<td>Standard Deviation</td>
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<td>SR</td>
<td>Sit and Reach</td>
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<td>VAS</td>
<td>Visual Analogue Scale</td>
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<tr>
<td>μ</td>
<td>Mean</td>
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Section One: Literature Review
**INTRODUCTION**

Chronic low back pain is a major problem to both the individual and society, due to its large direct and indirect treatment costs, as well as associated disability and suffering (Lehmann, Spratt, & Lehmann, 1993). Low back pain is discussed in the initial part of this literature review, including information of the various models of diagnosis and classification. As it is well recognised that chronic pain is a multidimensional issue (Dankaerts & O’Sullivan, 2011), models of mechanical, neurological and biopsychosocial influences are presented. Common motor control impairments are briefly explored. The absence of the Flexion Relaxation response, manifested as continued muscle activation at a posture where muscles typically relax is commonly identified in those with chronic low back pain (Watson, Booker, Main & Chen, 1997). Proposed mechanisms for the presence and absence of Flexion Relaxation are discussed in detail. The focus and challenge of recent research in the field of Flexion Relaxation has been to identify improvements of impaired Flexion Relaxation following various interventions, as well as investigating associations with pain and disability.
LOW BACK PAIN

Low back pain (LBP) is considered to be the most common, costly and disabling musculoskeletal condition (Lehmann et al., 1993). Defined topographically as pain occurring between the lower margins of the 12th rib and the gluteal folds (Johnson, Adegoke, & Ogunlade, 2010), LBP is an expensive issue due to the necessary spending towards repeated treatment, as well as the need for additional professional and personal support. In New Zealand, the estimated cost to the economy as a result of LBP is (NZD)$500 million annually (McBride, Begg, Herbison, & Buckingham, 2004), with Australia recently estimating over (AUD)$9 billion spent per year (Dagenais, Caro, & Haldeman, 2008). This high expenditure is principally due to the large number of lost workdays, considered an indirect cost, as well as the direct treatment costs (Dagenais et al., 2008; Krismer & van Tulder, 2007).

The high prevalence of back pain is another factor influencing cost. With an estimated 70-90% of any adult population experiencing at least one episode over their lifetime, LBP is the most prevalent of all musculoskeletal problems (Hoy, Brooks, Blyth, & Buchbinder, 2010; Johnson et al., 2010; Walker, 2000). In New Zealand, an estimated 20-25% of all workplace injuries are LBP related (Firth, Herbison, McBride, & Feyer, 2002). Over the last two decades, the prevalence of back pain and its associated costs have been increasing considerably (Gregg, Hoffman, Hall, McIntosh, & Robertson, 2011).

Contemporary healthcare generally considers back pain to be a multidimensional problem with a multi-causal aetiology (Dankaerts & O’Sullivan, 2011; O’Sullivan, 2005; Waddell, 1999). Low back pain can therefore present with a variety of symptoms, physical limitations, psychological features and consequences, all of which make effective treatment difficult (Dankaerts & O’Sullivan, 2011; Deyo & Phillips, 1996; Pedersen, 1981).

CLASSIFICATIONS

Although some evidence suggests that approximately 80% of acute back pain1 may spontaneously resolve irrespective of treatment (Andersson, 1999; Deyo & Phillips, 1996); a finding which is reflected in the current LBP guidelines (Accident Rehabilitation & Compensation Insurance Corporation of New Zealand, 2004), more recent investigation acknowledges an initial back pain episode considerably increases the chance of pain recurrence (Pengel, Herbert, Maher, & Refshauge, 2003). Between 24-87% of those who experience LBP, report symptomatic episodes over, and extending past, one year (termed ‘recurrent pain’). A further 6-10% are estimated to experience persistent pain, becoming a chronic problem (Carey, Garrett, & Jackman, 2000; Lalanne, Lafond, & Descarreaux, 2009; Pengel et al., 2003). Recent research has reported as many as 12-17% New Zealanders suffering with an episode of acute LBP, report it progressing to a chronic problem (Curia Research, 2012). The development of chronicity is associated with the largest costs to health services and highest cost to

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1 Low back pain is commonly defined simply on a temporal basis; pain that lasts less than six-weeks is defined as ‘acute’, ‘sub-acute’ pain is defined as pain that lasts between six-weeks to three-months (Krismer & van Tulder, 2007). ‘Chronic’ pain is defined as pain that lasts more than three-months (Accident Rehabilitation & Compensation Insurance Corporation of New Zealand, 2004).
the country in lost productivity, accounting for up to 80% of the healthcare compensation costs incurred (McBride et al., 2004; Quinn, 2002). Therefore the transition from acute pain to chronicity, as well as chronic LBP therapy has received much attention, with research aiming to reduce the prevalence and costs associated with chronic LBP (Carey et al., 2000; Dubois, Piché, Cantin, & Descarreaux, 2011; Klinger et al., 2010; O’Sullivan, 2005).

**NON-SPECIFIC CHRONIC LOW BACK PAIN**

A substantial volume of research has been undertaken in an attempt to explain and categorise back pain (Carey et al., 2000; Dubois et al., 2011; Klinger et al., 2010; Mannion, Käser, et al., 2001; O’Sullivan, 2005). Some have correlated pain with tissue based dysfunction, sometimes termed ‘pathoanatomical’, ‘structural’ or ‘mechanical’ back pain. However, pathoanatomical diagnoses appears controversial (Dagenais, Tricco, & Haldeman, 2010; Paalanne et al., 2011; Takatalo et al., 2009), due to a lack of objective clinical tests, poor inter-rater reliability in identification and lack of an accepted pathophysiological model of chronic muscular pain (Dagenais et al., 2010; Geissier, Alschuler, Donaldson, & Smith, 2007). A valid pathoanatomical diagnosis is estimated to be made in only 5-10% of LBP cases (Krismer & van Tulder, 2007).

Many of the objective indicators of pathology detected by imaging tools used by clinicians for diagnosis (including disk degeneration, herniation and other degenerative changes) can also be identified in asymptomatic individuals, thereby confounding diagnosis (Takatalo et al., 2009). Therefore, it is suggested that LBP is multifactorial and different chains of causation make it difficult to isolate risk factors (Gilkey, Keefe, Peel, Kassab, & Kennedy, 2010).

Low Back Pain is a general term, referring to a symptom rather than a diagnosis. In an estimated 85-95% of chronic LBP cases, no specific pain generating structure can be identified or definitive diagnosis established, and are therefore labelled ‘non-specific chronic LBP’ (Apeldoorn et al., 2010; Deyo & Weinstein, 2001; Dillingham, 1995; Krismer & van Tulder, 2007). This lack of identifiable causation makes it difficult for both practitioner and patient to direct effective management (Padfield, Chesworth, & Butler, 2002).

Chronic LBP is commonly associated with some level of functional limitation (or disability), including limitations in leisure activities, work productivity and activities of daily living (Bogduk, 2006). Disability can be attributed or influenced by different factors including: physical impairment (accounting for 40% of disability), psychosocial distress (23%), and illness behaviour (8%) (Waddell, Newton, Henderson, Somerville, & Main, 1993). This distribution leaves almost one-third of disability causality unexplained. Despite involving unidentified components, early disability potentially poses the greatest negative influence to rehabilitation, limiting recovery and predicting those progressing to chronicity (Accident Rehabilitation & Compensation Insurance Corporation of New Zealand, 2004). Given the widespread consequences of chronic LBP, one model which includes reference to the various systems involved is the biopsychosocial model.
BIOPSYCHOSOCIAL MODEL

The biopsychosocial model was first proposed in 1977 to acknowledge the unique psychological, social and behavioural aspects of illness (Engel, 1977). This model contrasts the purely pathoanatomical explanation, which aims to identify specific anatomical structures that are the source of low back symptoms (Slade, Troup, Lethem, & Bentley, 1983). In chronic LBP, physical findings seldom correlate with an individual’s experience of pain and disability (Main, Richards, & Fortune, 2000; Moseley, 2007). A large variety of factors, other than objective pathology and the musculoskeletal system may be involved in non-specific back pain. It is consistently reported in the literature that psychological and social factors influence the experience and course of back pain (Linton, Buer, Vlaeyen, & Hellsing, 2000; Thomas & France, 2008; Vlaeyen, Kole-Snijders, Boeren, & van Eek, 1995; Waddell et al., 1993). In some patients, psychosocial factors, fear, and catastrophising play central roles in their pain presentation (Saner, Kool, De Bie, Sieben, & Luomajoki, 2011).

One of the more important predictors of the development of chronic LBP is high fear of pain (kinesiophobia) and associated fear-avoidance behaviour is reported to correctly predict 66% of chronic pain sufferers (Kleenerman et al., 1995). The fear-avoidance model of chronic pain suggests the development of a cycle of decreased physical activity and an increase in pain perception (Slade et al., 1983). According to this model, those who perceive pain as a potential sign of tissue damage, are more inclined to avoid movements or behaviours, thus prolonging return to normal activities and delaying recovery (Linton et al., 2000; Thomas & France, 2008; Trost, France, Sullivan, & Thomas, 2012).

A common physical manifestation of fear-avoidance is a muscular ‘bracing’ or ‘splinting’ of the affected area, initially in order to prevent pain provocation (Main & Waddell, 1991; Sullivan et al., 2001). This splinting is commonly seen in acute pain situations, acting as an initial protective measure in order to prevent further damage to the injured area and promote initial healing (McGorry & Lin, 2012), by limiting extreme movements (Lund, Donga, Widmer, & Stohler, 1991; Sihvonen, 1997). The biological splint, is commonly attributed to neuromuscular or physiological changes occurring with the onset of pain perception (van Dieën, Selen, & Cholewicki, 2003; Zedka, Prochazka, Knight, Gillard, & Gauthier, 1999). Although splinting of the affected area is initially protective, over time the response may become habitual, in relation to perceived pain preserved by fear of pain, rather than nociceptive signals (Neblett, 2007). Fear of pain and associated avoidance of movements considered pain provoking has been correlated with reduced spinal range of motion (Geisser, Haig, Wallborn, & Wiggert, 2004; Thomas & France, 2007; Trost et al., 2012), associated with prolonged disability (Linton et al., 2000), and has even been implicated in maladaptive patterns of motor control as back pain develops (Dankaerts & O'Sullivan, 2011; Trost et al., 2012).

MOTOR CONTROL IMPAIRMENT

In a review exploring motor control impairment, Dankaerts and O’Sullivan (2011) suggested inherent maladaptive movement patterns, rather than pain avoidance, are implicated in developing chronic pain and act as a potential on-going peripheral nociceptive driver. Commonly identified motor control alterations may be a
result of the increased motor activity of the stabilising muscles of the spine (splinting), potentially resulting in increased and abnormal loading forces across pain sensitive structures (Dankaerts & O'Sullivan, 2011).

Three main mechanisms are proposed in the literature to explain this splinting behaviour: 1) the pain-spasm model; 2) the pain-adaptation model; and 3) reduced modulation depth.

**Pain-SPASM Model**

As early as 1942, Travell and associates (Travell, Rinzler, & Herman, 1942) suggested that the presence of pain, irrespective of mechanical alterations, would increase muscle tension, leading to muscular hyperactivity referred to as muscular ‘spasm’. This proposed spasm could increase pain and perpetuate a pain-spasm cycle, consistently increasing muscle contraction at both rest and with activity (van Dieën et al., 2003). Maintained and intensified muscle activity is attributed to hyper-excitability of the alpha motor neuron pool, due to increased muscle spindle sensitivity (van Dieën et al., 2003).

**Pain-ADAPTATION Model**

As the spasm model predicts increased muscle activation in a stereotypical manner (irrespective of task), the pain-adaptation model proposed by Lund et al. (1991) addresses the more variable changes identified in response to pain. Lund et al. (1991) state that the presence of pain will decrease the activation of muscles when active as agonists (concentrically contracting) and increase activation when the muscle is active as an antagonist (eccentrically lengthening). These muscular changes due to the presence of pain, result in reduced movement velocity, force and range (De Luca & Kline, 2012; Farina, Arendt-Nielsen, & Graven-Nielsen, 2005; Hodges & Tucker, 2011; van Dieën et al., 2003).

In acute pain situations, the increase in muscle activity via biological splinting mechanism may be functionally beneficial. However, after a prolonged increase in muscle activity, pain may actually increase owing to an accumulation of nociceptor stimulating substances such as arachidonic acid, bradykinin, potassium and lactate (Kaufman, Longhurst, Rybicki, Wallach, & Mitchel, 1983; Mense, 1993).

Both the pain-spasm and pain-adaptation models predict rather stereotypical responses to pain, and although supported by some research, numerous studies have reported results inconsistent with these models (Del Santo, Gelli, Spidalieri, & Rossi, 2007; Sessle, 1999; Svensson, Houe, & Arendt-Nielsen, 1997).

**Reduced Modulation Depth**

A more appropriate model of adaptation to pain, which encompasses both previously discussed models (Lund et al., 1991; Travell et al., 1942), may involve a reduction in the modulation depth of the muscle, resulting in more baseline muscle activity and less maximal activity (Dubois et al., 2011; Svensson et al., 1997; Zedka et al., 1999). Some researchers have shown increased baseline muscle activity in participants with local painful stimulation as well as in response to psychological stress (DeGood, Stewart, Adams, & Dale, 1994; Dubois et al., 2011; Flor, Birbaumer, Schugens, & Lutzenberger, 1992; Kravitz, Moore, & Glaros, 1981; Ohrbach et al., 1996; Zedka et al., 1999). As with the pain-spasm and pain-adaptation models, there is little muscle recovery time allowed, not only
for the painful muscle, but also the surrounding muscle. Although it is thought that the reduced modulation depth supplies the painful structure with increased stability in order to prevent further activity-induced injury, the mechanism may contribute to the gradual spread of pain to muscles in the vicinity of the original lesion (Zedka et al., 1999).

Although the underlying mechanism is uncertain, adaptation is commonly manifested in painful low back muscles as reduced activity of the paraspinal muscles during the movement phases of trunk flexion and re-extension, and increased activity with quiet standing and at the point of terminal flexion (Sihvonen, Huttunen, Makkonen, & Airaksinen, 1998; Watson et al., 1997; Zedka et al., 1999). This is commonly observed in chronic LBP patients and reported as an impaired Flexion Relaxation response (Ahern, Follick, Council, Laser-Wolston, & Litchman, 1988; Geisser et al., 2005; Silver & Floyd, 1955; Watson et al., 1997).

Impaired Flexion Relaxation has been reported to consistently identify those with back pain from asymptomatic individuals (sensitivity = 0.89; specificity = 0.81) (Geisser et al., 2005), and preliminary evidence suggests Flexion Relaxation can be restored following an intervention, which may correlate with decreased pain and disability suffered (Neblett, Alscher, Wiggert, Haig, & Geisser, 2009; Neblett, Mayer, Brede, & Gatchel, 2010).

**Flexion Relaxation**

Flexion Relaxation (FR) is a commonly observed muscle pattern, of lumbar paraspinal relaxation (electrical silence) near the end range of flexion (Neblett et al., 2010; Schultz, Sinkora, Warwick, & Haderspeck-Grib, 1985; Silver & Floyd, 1955). Flexion Relaxation is observable to some degree in most asymptomatic individuals. Impaired FR, displayed as continued muscle activation at maximal voluntary flexion (MVF), is commonly identified in those with chronic LBP (Figure 1) (Ahern et al., 1988; Triano & Schultz, 1987; Watson et al., 1997).

![Figure 1: Normal and Impaired Flexion Relaxation](image)

**Figure 1: Normal and Impaired Flexion Relaxation**

Note the significant reduction in surface electromyographic (SEMG) activity at maximum voluntary flexion (MVF) in an asymptomatic individual on the left, compared to the continued muscle activation in the individual with chronic LBP on the right. (Authors original data from attached manuscript). During the normal response, activity of the paraspinal muscles is generally low during quiet standing. As the individual begins to bend forwards, muscle activity increases as low back muscles contract eccentrically in order to support the trunk, controlling the speed of motion and accommodating the increasing effect of gravity. Close to MVF, muscle activity drops significantly, often to a level lower than at quiet standing, as the spinal passive elements support the load of the trunk and the contractile tissues relax. When the individual returns to standing, the spinal muscles contract concentrically and muscle activity resumes.
**MECHANISM**

Two main explanations have received support to explain FR. The mechanical model implicates a load sharing mechanism allowing paraspinal muscle activity to decrease, and the neural model attributes FR to neural reflexive responses (Floyd & Silver, 1951; Golding, 1952; Silver & Floyd, 1955). The amount of influence each mechanism has on the behaviour of the lumbar paraspinal muscles during trunk flexion is not certain.

The normal decrease in muscle activity is thought to be due to the extensor muscles being relieved of their active supporting role, by passive tissues of the area which provide adequate resistance to gravity (Colloca & Hinrichs, 2005; Demoulin, Crielaard, & Vanderthommen, 2007; Kippers & Parker, 1984). As well as this, vertebral structure compression occurs and re-extension torque of the posterior vertebral elements is produced. Although the lumbar paraspinal muscles appear silent during MVF as they no longer actively contract, they are still generating forces elastically through stretching of passive elements. Activation of muscles other than the paraspinals (i.e. the quadratus lumborum and deep lateral muscles) also occurs at MVF, providing additional support to the passive spinal elements (Andersson, Oddsson, Grundstrom, Nilsson, & Thorstensson, 1996; Dolan, Mannion, & Adams, 1994).

Furthermore, various studies have investigated a mechanism of reflex-inhibition relating to the electrical silence observed at MVF (Colloca & Hinrichs, 2005; Solomonow, Baratta, Banks, Freudenberger, & Zhou, 2003; Youssef et al., 2008). Stretch receptors located in the posterior spinal elements (ligaments and discs) are sensitive to increased joint angles and capsular tension. These receptors are involved in the ligamento-muscular reflex (Solomonow et al., 2003) and with the aim of increasing joint stability, may be excitatory or inhibitory. In the case of FR, it is proposed the increased tension of the posterior elements with flexion, elicits an inhibitory reflex, signalling the spinal muscles to relax (Gupta, 2001; McGill & Kippers, 1994). Therefore load transfer is achieved, resulting in an equilibrium between upper body weight and gravity, as the support is economically maintained by viscoelastic elements (McGill & Kippers, 1994; Schultz et al., 1985).

Several factors have been investigated which alter the normal FR response. Modifying factors of loading, movement speed, fatigue and movement range have resulted in altered FR in asymptomatic individuals (Ahern, Hannon, Goreczny, Follick, & Parziale, 1990; Descarreaux, Lafond, Cantin, Jeffrey-Gauthier, & Centomo, 2010; Gupta, 2001; Lee, Yoo, An, Kim, & Oh, 2011; McGill & Kippers, 1994). Most notably, those with LBP commonly present with impaired FR.

**CHRONIC LOW BACK PAIN AND FLEXION RELAXATION**

Absent FR has been shown to be a pattern that is constant and predictable in individuals with chronic LBP. Although at MVF muscle activity normally reduces (up to a 90% reduction), individuals with chronic LBP commonly display little or no reduction in muscle activity (Ahern et al., 1988; Kaigle, Wessberg, & Hansson, 1998; Watson et al., 1997). Geisser et al.’s (2005) meta-analysis including a pooled sample of n=227 LBP participants and n=115 controls, reported that FR could discriminate between individuals with and without LBP, reported to correctly identify 86-89% of chronic LBP patients from asymptomatic individuals (Ahern et al., 1988; Watson et al., 1997).
Clinicians often recognise the presence of pain is associated with increased muscle activity during periods where relaxation is typically observed (MVF), and decreased activity during periods of active contraction (flexion and re-extension) altering movement dynamics (De Luca & Kline, 2012; Lund et al., 1991; van Dieën et al., 2003). The aforementioned protective spasm response elevates myoelectric activity and alters motor control leading to persistent activation of the paraspinal muscles (Lalanne et al., 2009). Normally, this heightened muscle activity decreases in response to reduced pain with healing (Solomonow, Hatipkarasulu, Zhou, Baratta, & Aghazadeh, 2003). However, in those with chronic pain this normal reduction may not occur.

The mechanism for impaired FR has been investigated and abnormal FR in chronic LBP patients has since been attributed to different factors including an effort to protect damaged passive structure, fear-avoidance behaviours, muscle spasm, exaggerated stretch reflexes, or reduced spinal range of motion (Ahern et al., 1988; Colloca & Hinrichs, 2005; Demoulin et al., 2007; Descarreaux, Lafond, Jeffrey-Gauthier, Centomo, & Cantin, 2008; Gupta, 2001; Hashemirad, Talebian, Olyaei, & Hatef, 2010; Watson et al., 1997).

Sihvonen et al. (1998) reported absent FR was more common in those with current pain, rather than those being pain free at the time of testing as nociceptive stimulation alters stretch receptor sensitivity, disrupting stiffness regulation and motor control. In an attempt to provide additional stability, activity of the afferent receptors and local output increases, manifesting as continued paraspinal activity, potentially limiting movement (Johansson & Sojka, 1991; Matre, Sinkjaer, Svensson, & Arendt-Nielsen, 1998). Supporting evidence is provided in both animal and human investigations (Djupsjöbacka, Johansson, Bergenheim, & Wenngren, 1995; Masri, Ro, & Capra, 2005; Thunberg et al., 2001).

A study by Zedka et al. (1999) aimed to test the hypothesis that painful input to the central nervous system leads to muscular hypertonicity, through increased spindle sensitivity to stretch or increased gain of central transmission. By injecting hypertonic saline into the lumbar muscles of healthy participants, muscular pain was provoked and muscle-activation patterns were recorded. The induced pain resulted in reduced voluntary movement patterns, similar to those observed in patients with LBP. Increased muscle activation was observed in asymptomatic individuals injected with hypertonic saline, producing muscular pain despite no underlying injury (Zedka et al., 1999). Therefore changes in modulation depth appear to be in response to ‘perceived’ rather than actual tissue injury. As Zedka et al.’s (1999) findings suggest cutaneous pain did increase the amplitude of the long-latency response; they hypothesised facilitation of poly-synaptic reflex pathways by cutaneous receptors, concluding the changes in muscle activity are not associated with increased gain of the spinal muscle stretch reflex, but rather suggest a more complex mechanism.

**Range of Motion and Flexion Relaxation**

As FR is likely to be mediated to some degree by a mechanical reflex, absent FR has been attributed to lumbar motion restriction, where individuals do not achieve flexion to the degree necessary to evoke the reflex (Colloca & Hinrichs, 2005; Hashemirad, Talebian, Hatef, & Kahlae, 2009; Kaigle et al., 1998). Strong evidence indicates that chronic LBP is associated with a reduction in range of motion (ROM) (Hansen et al., 1955; Neblett, Mayer, Brede, & Gatchel, 2009; Thomas & France, 2008; Triano & Schultz, 1987). Impaired range of motion (ROM) is
associated with high fear of pain and is potentially due to heightened resting potentials of the paraspinal muscles and neuromuscular spasm (Arena, Sherman, Bruno, & Young, 1991; Geisser et al., 2005).

Although normal ROM is reported in most individuals achieving FR, impaired FR is commonly present even with the patient achieving a normal flexion range (Neblett, Alschuler, et al., 2009; Neblett, 2007; Neblett, Mayer, et al., 2003). Reduced lumbar ROM therefore does not fully explain absent FR, as even individuals with chronic LBP have been shown to flex past the angle at which FR commonly occurs in asymptomatic individuals (Kaigle et al., 1998; Kippers & Parker, 1984; Thomas & France, 2008). Although some authors report that FR will typically occur at a point after 70 degrees of trunk flexion (Kaigle et al., 1998; Kippers & Parker, 1984; Wolf, Basmajian, Russe, & Kutner, 1979), more recent studies indicate that the required angle is more likely based on individual flexibility than a mechanically pre-determined angle (Shin, Shu, Li, Jiang, & Mirka, 2004).

In order to exclude a pain-mediated reduction in ROM from affecting FR recordings, Zedka et al. (1999) measured the forward flexion movement in asymptomatic participants, then had participants perform the same movement with the use of a mechanical, guiding arm, following the administration of hypertonic saline to the lumbar muscles. With the provocation of pain, participants displayed decreased range of only 60-90% of the control range, and displayed reduced modulation depth. When producing movements identical to when pain free, participants still displayed elevated muscle tone in painful situations, despite overcoming muscular splinting (Zedka et al., 1999). As changes involve more than just a strategy to reduce the extent of movement, it therefore seems unlikely that improved FR can be attributed exclusively to changes in ROM.

**QUANTIFYING FLEXION RELAXATION**

Although the underlying mechanism behind altered lumbar FR is uncertain, the reliability of this measure to differentiate between chronic LBP and pain free individuals has made this phenomenon a highly investigated topic, and a commonly utilized outcome measure in back pain literature.

Researchers have utilised different methods of interpreting, understanding and examining muscle activation levels and FR in individuals with chronic LBP. This discrepancy makes it difficult to compare results of studies investigating FR. Measurements vary from static to dynamic measures and can be analysed individually or as composites. Appropriate relaxation has been categorized by the expression of an improved ratio (Marshall & Murphy, 2006a, 2008; Watson et al., 1997), the attainment of specified voltage levels (Neblett, Mayer, et al., 2003; Neblett et al., 2010) and visual inspection alone of the signal phases (Mannion, Taimela, Müntener, & Dvorak, 2001).

The method most commonly used to present FR is by calculating a ratio from formulas utilising various surface electromyography (SEMG) measures. The FR ratio was developed to provide a reliable, repeatable and objective method to quantify FR (Watson et al., 1997). Calculation of the ratio may include comparing the mean maximal SEMG during one of the four phases of the flexion movement, with that from another phase (Ambroz, Scott, Ambroz, & Talbott, 2000; Neblett, Alschuler, et al., 2009; Owens, Gudavalli, & Wilder, 2011; Watson et al., 1997).
Due to the technical differences in equipment, methods of collecting data, as well as individual human characteristics, studies have quantified the best way of normalizing and presenting FR data (Neblett, Alschuler, et al., 2009; Owens et al., 2011). A 2009 study investigating the most reliable methods of ratio calculation revealed the ratio from the maximum SEMG during re-extension to the average SEMG at MVF (termed the extension-relaxation ratio), was most highly associated with clinical and musculoskeletal aspects of back pain compared to other calculations (Neblett, Alschuler, et al., 2009). This ratio is significantly associated with disability, pain related fear and clinical status. It is also the only ratio sensitive to self-reported pain intensity, which has been poorly associated with FR changes in the past (Geisser et al., 2004; Neblett, Alschuler, et al., 2009).

As much back pain lacks identifiable (therefore un-measurable) structural pathology, SEMG and the FR response offer a simple method of measuring and monitoring back pain objectively, by evaluating abnormal muscle function (Angoules et al., 2008; Geisser et al., 2005). As abnormal FR is consistently identified in chronic LBP patients, researchers and clinicians have used FR as an objective outcome measure in interventions aimed at treating back pain.

Although there are numerous studies employing FR as an outcome measure related to chronic LBP interventions, much of the literature is low quality due to the heterogeneous samples, technical variation of electromyographic equipment, data analysis and sample sizes utilized. As well as differing ways to normalise and report electromyographic data, there are also differences in calculations of significant change that makes it difficult for comparison of results across studies (Marshall & Murphy, 2008; Neblett et al., 2010; Watson et al., 1997). Although some investigators report normal FR as attaining specific muscle activity thresholds (Neblett, Mayer, et al., 2003; Neblett et al., 2010), FR is not a dichotomous phenomenon, with no definitive cut-off point defining presence or absence (Mannion, Taimela, et al., 2001; Marshall & Murphy, 2006b; Owens et al., 2011).

**Effects of Treatment on Flexion Relaxation**

There are a small number of high quality studies evaluating the effects of various interventions on chronic LBP which utilise FR as an outcome measure. There have been mixed results, with some studies identifying improvements in FR following interventions (Geisser et al., 2005; Marshall & Murphy, 2006a; Neblett, Mayer, et al., 2003; Watson et al., 1997) and others not (Mannion, Taimela, et al., 2001; Ritvanen, Zaproudina, Nissen, Leinonen, & Hänninen, 2007).

Marshall and Murphy (2008) evaluated FR in chronic LBP patients (n=50), before and after a three-month exercise programme. FR measures were reported as an extension-relaxation ratio and significant change was determined by statistical analysis. The results showed a significant improvement in FR (effect size = -1.60, 95% CI = -2.25 to -0.94; $p = 0.01$), and significant reductions in subjective disability measures ($p = 0.023$).

Also reporting a significant improvement in FR was Lalanne et al. (2009), comparing within session FR measures (flexion relaxation ratio) after a single session manipulation (high velocity thrust) or sham treatment in individuals with chronic LBP (n=27). The manipulation group achieved a statistically significant improvement in
FR compared to the sham treatment group (effect size = -1.40, 95% CI -2.24 to -0.56; *p* =0.007). Although it is reported that pain and disability measures were taken at baseline, post intervention data is not reported. These findings suggest FR-related measures have some ability to discriminate functional improvement following rehabilitation programmes.

In contrast, two other studies found no significant differences in FR, following a traditional bone setting intervention versus physical therapy (n=61) (Ritvanen et al., 2007) or a physical therapy, strengthening or aerobics programme (n=132) (Mannion, Taimela, et al., 2001). Limitations of the studies included post intervention scores taken one month following treatment (Ritvanen et al., 2007), and FR grading being by visual analysis alone (Mannion, Taimela, et al., 2001). Although Mannion et al. (2001) reported no change in FR and no association with ROM, they did identify increased maximum SEMG recordings indicating increased muscle force, as well as improved pain intensity measures.

It is important to note that these studies have not specifically targeted FR with the intervention, instead utilising FR as an outcome measure only. Therefore FR is likely indirectly affected by treatments aimed primarily at affecting other factors associated with back pain. Recently, Neblett et al. (2010) published the first study to directly affect FR with an intervention including SEMG biofeedback assisted stretching in chronic LBP patients.

**BIOFEEDBACK**

The Association for Applied Psychophysiology and Biofeedback (2013), describes biofeedback as a process that enables an individual to learn how to change physiological activity, for the purposes of improving health and performance. Information about physiological processes is conveyed to the patient in real time, commonly via visual or auditory cues. Surface electromyography, which measures muscle activation levels, is the most commonly used biofeedback tool in musculoskeletal rehabilitation, in part due to the electrodes being non-invasive and providing a simple, reliable tool to measure myoelectrical activity of the muscle it is placed over (Donaldson, Donaldson, & Snelling, 2003; Neblett, Alschuler, et al., 2009; Neblett, Mayer, et al., 2003). Additionally, SEMG is sensitive to influences from both physical and psychosocial aspects of pain, consistent with the bio-psychosocial model of pain (Robinson & Riley, 1998).

Evidence suggests pain modulation with biofeedback in chronic pain patients may be achieved via de-catastrophizing and learning techniques to lower arousal (Angoules et al., 2008; Jones & Wolf, 1980; Vlaeyen et al., 2002). This may prevent the maintenance of sympathetic drive, alter improper muscle activation and blood flow patterns and counter the effect of central sensitisation (Crider, Glaros, & Gevirtz, 2005; McNulty, Gevirtz, Berkoff, & Hubbard, 1994; Sherman, 2003; Tan et al., 2007). Biofeedback has been highly investigated in clinical cases of musculoskeletal pain with favourable results, underlining the potential of electromyographic feedback for pain relief (Angoules et al., 2008; Crider et al., 2005; Henschke et al., 2010).

The aim of biofeedback is to the patient to consciously reduce muscular tension, anxiety and subsequently, pain, commonly achieved with the inclusion of stretching (Henschke et al., 2010). The benefit in combining biofeedback with stretching is that it influences both the musculoskeletal system and the neurological system.
Biofeedback assisted stretching aims to teach participants how to relax their muscles during movement. Participants are able to see or hear cues to inform them when their muscles are activated over a pre-determined threshold. Encouragement can then be given to relax the specific muscles to below the threshold, influencing the visual or audio feedback. When a practitioner utilizes feedback during a stretch, it is with the aim to have the patient put the muscle on stretch, without significantly increasing the stretch receptor drive on the alpha motor system (Cram & Kasman, 1998). Patients are able to receive objective indication of what the muscles are doing in real time and able to see if their muscle activity is considered normal or abnormal.

In 2010, a Cochrane review exploring behavioural treatments included a meta-analysis of low back electromyographic biofeedback treatment, compared to wait list controls for chronic LBP (Henschke et al., 2010). The three randomised controlled trials included a pooled total of n = 34 patients in intervention groups and n = 30 in control groups (Newton-John, Spence, & Schotte, 1995; Nouwen, 1983; Stuckey, Jacobs, & Goldfarb, 1986). The biofeedback group displayed a statistically significant standardised mean difference in short term pain intensity (-0.80, 95% CI -1.32 to -0.28) compared to the controls. Flexion Relaxation was not discussed.

Not only is SEMG (biofeedback) Assisted Stretching (SEMGAS) a training tool to promote clinical changes, it also provides a rationale for the patient to effectively relax during a stretch as they have been provided with objective signs of dysfunction (Geisser et al., 2004; Neblett, Gatchel, & Mayer, 2003). It has been reported SEMGAS can increase flexibility in the low back, as well as in other parts of the body, including the upper extremity, knee, hips and neck (Cram & Kasman, 1990, 1998; Jones & Wolf, 1980; Neblett, Gatchel, et al., 2003). Recent studies have also shown that SEMGAS may have an effect on FR (Neblett et al., 2010; Neblett, 2007; Neblett, Mayer, et al., 2003). In relation to impaired FR, patients can identify abnormally high muscle tension, and are provided with strategies to reduce the muscular activity at MVF.

**SURFACE ELECTROMYOGRAPHY ASSISTED STRETCHING AND FLEXION RELAXATION**

Investigation of SEMGAS and FR appears to have been undertaken by only one research group. This research group has provided information on theory, rationale and clinical applications, as well as reporting quantified improvement in FR post intervention (Neblett, 2002, 2007; Neblett, Gatchel, et al., 2003; Neblett et al., 2010).

The first occasion FR and SEMGAS were investigated together was in Neblett et al.’s 2003 paper (n = 46). This study was later replicated on a larger scale (chronic LBP n = 104, control n = 30) in 2009 utilising similar chronic LBP populations, outcome measures and intervention (Neblett, Mayer, et al., 2009). These studies investigated FR, ROM and Million VAS (pain and disability 150 point scale) as outcome measures in chronically disabled occupational lumbar disorder patients, versus asymptomatic controls following a comprehensive multimodal intervention (functional restoration), employing SEMGAS as a component.

The two studies (Neblett, Mayer, et al., 2009) reported very similar results, with number of participants achieving FR increasing significantly from baseline to post intervention (41% to 94%; $p = <0.001$ in 2003 and 31% to 74%; $p = <0.001$ in 2009). The predictive value of FR predicting the presence of ‘disease’ (abnormal ROM) was
high in both studies with specificity of 98 to 100%. However both reported lower sensitivity (25-79%) as some participants achieved FR despite displaying abnormal motion (false negatives) Additionally, pain and disability as measured by the Million VAS scale significantly improved in both studies post intervention ($p = <0.01$). As the improvement in pain was associated with the patient’s ability to achieve FR, Neblett et al. (2003) suggested diminution of pain inhibition and fear-avoidance occurred, concluding FR is responsive to functional improvements including changes in ROM and self-reported scales of pain and function (Neblett, Mayer, et al., 2003, 2009).

Following from this, Neblett et al. (2010) recently published the first study investigating changes in FR following an intervention (rather than employing it as an outcome measure), comparing an asymptomatic control group ($n = 30$) with two groups of chronic LBP patients ($n = 94$) involved in an intervention. Both treatment groups were involved in the same functional restoration programme, similar to the one utilised in the 2003 and 2009 studies. Functional restoration typically involved 160 to 240 hours of training over two to four months and included stretch training, cardiovascular exercise, educational classes, cognitive behavioural therapy and stress management training (Neblett et al., 2010). One of these treatment groups ($n = 71$) was additionally involved in a SEMGAS programme to relax the paraspinal muscles during flexion, directly aimed to influence FR.

Both treatment groups significantly improved in FR and ROM measures ($p = <0.001$). In the SEMGAS group, the number of participants achieving FR increased from 34% at pre-intervention, to 86% at post intervention (compared with 17 to 26% in the functional restoration only group). The improvement in this group was such that the chronic LBP group displayed FR comparable (not statistically different) at post intervention with an asymptomatic population ($p = 0.534$), as was ROM ($p = 1$). This suggests that the addition of the SEMGAS can lead to normalization of FR and range rather than just an improvement (Neblett et al., 2010). Pain and disability were not investigated in this study. Researchers hypothesised that although other training methods may improve ROM, without specific SEMGAS training, participants may still be unable to achieve normal muscular relaxation (Neblett et al., 2010).
**Conclusion**

Although FR is a useful tool for objective measurement of functional improvements in individuals with chronic LBP, only one study (Neblett et al., 2010) has investigated the effect of an intervention on FR specifically. Although the results from this study are positive, the intervention required a large time commitment and measures of pain intensity and disability were not assessed.

Further research is required to investigate the exclusive use of SEMGAS in a typical clinical setting, and to explore associations between FR and pain intensity and disability in individuals with chronic LBP.


Section Two: Manuscript

Although this manuscript is written in the style described for the *Journal of Bodywork and Movement Therapies* [Appendix J], tables and figures have been placed throughout the body (rather than on a separate document) for ease of examination. References to the appendices throughout the following manuscript (presented in square brackets) have also been included for examination purposes only, and are not intended for manuscript submission.
The efficacy of surface electromyographic biofeedback assisted stretching for the treatment of chronic low back pain: A case series
The efficacy of surface electromyographic biofeedback assisted stretching for the treatment of chronic low back pain: A case series

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<table>
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<th>Abbreviation</th>
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<tr>
<td>CI</td>
<td>95% Confidence Interval</td>
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<tr>
<td>Cohens $d$</td>
<td>Effect Size</td>
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<td>dB</td>
<td>Decibel</td>
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<tr>
<td>FIR</td>
<td>Finite Infinite Response</td>
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<td>FR</td>
<td>Flexion Relaxation</td>
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<td>Hz</td>
<td>Hertz</td>
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<td>kHz</td>
<td>Kilohertz</td>
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<tr>
<td>LBP</td>
<td>Low Back Pain</td>
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<tr>
<td>L2, L4</td>
<td>Lumbar levels two and four</td>
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<td>M</td>
<td>Median</td>
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<tr>
<td>MVF</td>
<td>Maximal Voluntary Flexion</td>
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<td>NPRS</td>
<td>Numeric Pain Rating Scale</td>
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<td>NZ</td>
<td>New Zealand</td>
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<td>ODI</td>
<td>Oswestry Disability Index</td>
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<td>$p$</td>
<td>Statistical Probability</td>
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<td>P</td>
<td>Participant</td>
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<tr>
<td>PGIC</td>
<td>Patient Global Impression of Change</td>
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<td>RMS</td>
<td>Root Mean Square</td>
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<td>ROM</td>
<td>Range of Motion</td>
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<tr>
<td>SEMGAS</td>
<td>Surface Electromyography Assisted Stretching</td>
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<tr>
<td>SD</td>
<td>Standard Deviation</td>
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<tr>
<td>SR</td>
<td>Sit and Reach</td>
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<td>T</td>
<td>Wilcoxon-rank Test</td>
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ABSTRACT

Flexion Relaxation (FR) is a pattern of lumbar paraspinal muscle relaxation near the end range trunk flexion observed in most asymptomatic individuals, however is typically reduced or absent in those with chronic low back pain (LBP).

This study aimed to investigate the effects of a surface electromyographic assisted stretching (SEMGAS) programme, on impaired FR, pain intensity, disability and flexibility in individuals with chronic LBP. Nine chronic LBP patients recruited from the general public, who displayed impaired FR, took part in a biofeedback SEMGAS intervention, with measures taken pre and post intervention and at a follow up. Of the nine participants, three improved pain intensity and FR scores to clinically significant levels with a group mean significant improvement in FR (p = 0.027) observed from baseline to follow up, suggesting SEMGAS may provide benefits to some individuals with chronic LBP and impaired FR. A larger scale investigation is indicated.

Keywords: Flexion Relaxation; Chronic low back pain; Surface electromyography; SEMG-assisted stretching; Biofeedback; Pain intensity; Disability; Range of motion; Maximum voluntary flexion
Flexion Relaxation (FR) is a commonly identified muscle activation pattern where lumbar paraspinal muscle activity decreases near maximal voluntary flexion (MVF). The phenomenon was first described by Floyd and Silver (1951) and is consistently observable in pain free individuals (Kippers & Parker 1984; Silver & Floyd 1955).

Flexion Relaxation is commonly attributed to the redistribution of load bearing structures of the spine; from the muscles which contract eccentrically to control the flexion movement, to the passive structures, including spinal ligaments, discs and fascia (Colloca & Hinrichs 2005; Kippers & Parker 1984). As the posterior passive ligaments become increasingly tensioned during flexion, stretch receptors located in those posterior elements produce a reflex, which acts to inhibit the paraspinal muscles (McGill & Kippers 1994; Schultz et al 1985).

Individuals with chronic low back pain (LBP) commonly display abnormal FR, maintaining substantial muscle activity at MVF. Absent FR can reportedly identify 86-89% of chronic LBP patients from asymptomatic individuals (Ahern et al 1988; Watson et al 1997). While an abnormal FR appears to present as a positive adaptive response to acute injury, it also poses potential risk for continued neuromuscular maladaptation in chronic conditions. Continued muscle activation is thought to contribute to pain, in part due to the accumulation of nociceptor stimulating substances. Evidence indicates that FR can be restored with the resolution of pain symptoms (Ahern et al 1990; Haig et al 1993; Hides et al 1996; Silver & Floyd 1955). In this context, lumbar FR has been utilized to objectively measure functional improvements after rehabilitation programmes including exercise (Marshall & Murphy 2006; 2008), spinal manipulation (Lalanne et al 2009), and functional restoration (Marshall & Murphy 2006; Neblett et al 2003; 2010).

Recent investigation by Neblett et al (2010) has provided evidence that FR can be normalised following a multimodal intervention with a Surface Electromyography Assisted Stretching (SEMGAS) component, aiming to directly affect FR. Following the intervention, individuals with chronic LBP displayed lumbar paraspinal myoelectric activity which was not statistically different ($p = 0.534$) from the asymptomatic control group (86% achieving FR at post intervention, compared to 35% achieving at baseline) (Neblett et al 2010). However, SEMGAS has not been evaluated as a multimodal treatment for improving impaired FR in individuals with chronic LBP, nor has SEGMAS been assessed in a conventional clinical setting with an at-home component.

The present study aims to evaluate the effectiveness of a SEMGAS programme at improving FR, pain intensity and disability. Specifically, the aims of the study were: 1) To evaluate if a SEMGAS programme run over five weeks including an at-home stretching component will improve abnormal FR patterns in individuals with chronic LBP; and 2) To evaluate if a change in FR is associated with improved range of motion, pain intensity and disability in individuals with chronic LBP.
METHODS

Design

A case series was undertaken to measure the effects of a SEMGAS biofeedback programme to treat chronic LBP.

Participants

Participants with chronic LBP were recruited over a 12-month period from the general public. Advertising included posters, online advertising (through researchstudies.co.nz, an online participant recruitment website) and an advertorial in local suburban newspapers [Appendix A].

Participants were included in the study if they were between 16 and 65 years of age; had experienced chronic LBP for a minimum of three months; and displayed impaired FR (as identified at initial consultation, visually determined by observation of ‘more than usual’ activity occurring during terminal flexion when the participant was asked to relax and allow the body to ‘hang’).

Participants were not eligible if they had a history of muscular or spinal pathology (including spinal stenosis, spinal surgery, neurological symptoms, osteoporosis, lower limb musculoskeletal injuries or surgeries or disc injuries), Body Mass Index over 35 kg/m², or were currently involved in another physical rehabilitation intervention.

Informed consent was received from all participants [Appendix B]. This study was approved by the Unitec Research Ethics Committee, UREC reference: 2011-1193 [Appendix C].

Outcome Measures

Upon enrolment a standardised history and basic physical examination was undertaken. Baseline outcome measurements of FR, disability, flexibility and pain intensity were recorded. The intervention commenced immediately, consisting of a daily home stretching programme and weekly SEMGAS biofeedback sessions over 5-weeks. Weekly measures of FR, sit and reach (Predrag et al 2010), the numeric pain rating scale (Childs et al 2005) and the Oswestry questionnaire (Fairbank & Pymsent 2000) were collected throughout the study [for additional information on outcome measures see Appendix D], and again 4 to 6 weeks after intervention completion (follow-up at week 9 to 11). At follow-up, an additional measure, the Patient Global Impression of Change (Watson et al 2005), was also completed [Appendix E].

Flexion Relaxation Instrumentation

Electromyographic data was collected using 20 mm diameter, self-adhesive foam electrodes (Meditrace; Covidien Mansfield, MA), placed bilaterally on the muscle bellies of the lumbar paraspinal muscles at the L2 and L4 levels and approximately 2 cm lateral of the midline. This placement is consistently reported in the literature (Lalanne et al 2009; Neblett et al 2003; Watson et al 1997). The skin was gently abraded with medical abrasion tape (trace
dot prep; 3M, St Paul, MN) and wiped with an alcohol swab, in order to reduce skin impedance to below 5 kilohms. The participant was asked to bend forward approximately 40 degrees whilst the four self-adhesive electrodes were placed, and additionally secured with medical tape, allowing for skin stretch during the bending movement. A reference electrode was placed over the bony prominence of the left olecranon.

Electromyographic activity was recorded with a Power Lab/8SP and Octal bio amp (AD Instruments, Dunedin, NZ). Data was recorded with LabChart 7.2.1 (AD Instruments, Dunedin, NZ) at a sampling rate of 2 kHz and Finite Infinite Response (FIR) filters were used on the raw EMG data. Data was band-pass filtered between 20 Hz (high-pass FIR filter with a half-amplitude frequency of 20 Hz and transition width of 15 Hz) and 500 Hz (low-pass FIR filter with a half-amplitude frequency of 500 Hz and transition width of 100 Hz). When the power of 50 Hz (Fast Fourier Transformation) exceeded twice that of any other frequency band during the re-extension phase, a 50 Hz second-order notch filter with 32 dB attenuation was employed to filter noise artefact.

**Flexion Relaxation Procedure**

The movement required to assess FR consisted of the participant standing with arms relaxed, barefoot, and feet shoulder-width apart. The researcher explained and demonstrated the forward bending movement to the participant. The instruction included starting in a comfortable standing position and maintaining straight, but unlocked knees. Participants were encouraged to maintain a regular breathing pattern as well as free hanging arms throughout the movement. Participants were required to bend forwards towards their toes as far as they could go before feeling pain more than a ‘mild discomfort’ in their low back. This movement was completed to a three-second count. Participants were then required to hold the position of maximal flexion for three seconds, and were encouraged to relax and let their body ‘hang’ if they were able to do so without significant pain. The re-extension movement returned the participant to the standing position over the final three seconds.

Following three practice trials, data was recorded for three further consecutive repetitions. The movement was defined by standard phases for analysis, including standing, flexion movement, maximal voluntary flexion (MVF) and return to standing (re-extension movement).

**Treatment**

Participants took part in a 5-week programme including a daily at-home stretching protocol, concurrent with five sessions of 20-minute biofeedback involving SEMGAS, scheduled one per week with the researcher.

*Surface electromyographic assisted stretching (Biofeedback)*

Following electrode placement on the lumbar paraspinal muscles, the participant was directed to slowly flex the neck and bend forward with their knees slightly bent, as if to touch their toes. When they felt a gentle stretch in their low back, they were to stop the bending movement and concentrate on relaxing the muscles in their back. The stretch was held for 30 seconds and completed three times, with data being recorded in real time.
The researcher prompted the patient to relax with phrases such as: “relax completely into the stretch”, “allow gravity to do the work and relax your body”, “focus on passive relaxation, rather than forcing the movement”. Feedback included verbal feedback, visual feedback (slightly delayed as participants were unable to view the computer display from the fully flexed position), as well as verbal coaching and encouragement, as previously reported (Neblett et al 2003).

The raw SEMG data recorded was discussed as feedback with the participant. Participants were shown their elevated muscle activity trace during the target stretch, compared to normal FR. Participants were reassured the stretch was safe, were encouraged not to be fearful of the movement, and were regularly reminded of the objective to achieve minimal activity of their lumbar muscles during the stretch.

Home Stretching

The home stretching programme was run concurrently over the 5-week intervention phase. Participants were asked to complete three repetitions of the forward flexion low back muscle stretch each day, holding the stretch for between 30 and 120 seconds. Participants were encouraged to be conscious of relaxing the muscles identified in the biofeedback sessions.

Participants were provided with a stretch diary utilising a tick-sheet where they were asked to indicate when they had successfully completed their three stretches for the day. This diary provided a self-reported record of adherence to the programme [Appendix G].

Follow-Up

At the completion of the intervention, the participants were asked to return between 4 to 6 weeks later (week 9 to 11) in order to repeat all of the outcome measures to identify medium term changes. Although the tick sheet provided to participants did not extend past the intervention phase, participants were encouraged, but not required, to continue with the regular stretching over the time between the final intervention and follow-up.

DATA ANALYSIS

SEMG Data Reduction

A ratio was calculated from the raw SEMG data collected to allow for comparison between time points. Many different methods of quantifying FR have been reported in the literature (Neblett a et al 2009; Owens et al 2011), however, the measure using maximal activity from re-extension and MVF (extension relaxation ratio) was chosen as it has been reported to be highly correlated with clinical and musculoskeletal aspects of back pain (Neblett a et al 2009).

Approximately 2 seconds (visually determined) of raw data from the middle of each movement phase was imported into a custom spreadsheet in Microsoft Excel 2010 for processing. Data was rectified and averaged using a moving one-second window Root Mean Square (RMS) calculation, where each window included 2000
raw data points and each successive window incremented by a single data point. The greatest one-second RMS value from the re-extension and MVF phases was used to calculate the extension relaxation ratio. At each session, the procedure was repeated and recorded three times, and the mean ratio of both left and right sides from the three recorded trials were used for further comparison and analysis.

Quantitative Analysis

The initial measures from each participant were used as baseline data. Comparisons were then made between baseline, post-intervention and follow-up using the reported minimally clinically important difference [for additional information on outcome measures see Appendix D]. Clinically relevant change was therefore; 2 points for the Numeric Pain Rating Scale (NPRS) (Childs et al 2005); 10 points for the Oswestry Disability Index (ODI) (Fritz et al 2009); and 4 cm for the Sit and Reach (SR) (López-Miñarro et al 2012).

Change in FR was analysed using the Two Standard Deviation Band method that is calculated from the baseline data of all participants. The baseline measures had a mean extension relaxation ratio of 1.81 and standard deviation (SD) of 0.6. Therefore a two SD change of 1.2 in FR measures was considered significant. This procedure was used as it has the advantage of being sensitive to changes in variability across the phases of a single-system design (Nourbakhsh & Ottenbacher 1994).

To assist visual interpretation of the results presented in tables and figures, the negative sign (-) was used to indicate an unfavourable result (i.e. an increase in pain or disability or a decrease in FR or flexibility) and changes considered clinical improvements are reported as positive values.

Overall group change was analysed using the Wilcoxon Signed-Rank Test to identify changes between baseline and post intervention scores, as well as between baseline and follow-up scores for each of the four outcome measures. Statistical significance was set at the $p < 0.05$ level. Group means ($\mu$) and medians (M) are reported, and where possible, standard deviations (SD), 95% confidence intervals (CI) and effect sizes (Cohen’s $d$) are presented.
RESULTS

Of the 40 applicants responding to advertising, 16 met initial inclusion criteria, and of those, nine individuals displayed a visually detectable abnormal FR response and were invited to participate in the study (Table 1)[ for additional case history information see Appendix I]. All nine participants completed the five intervention sessions. However, only seven participants completed all follow-up measures. Scheduling conflicts (P3) and a scheduled knee surgery (P4) were reported barriers for attending follow-up. The two participants unable to attend the follow-up session completed online versions of the ODI, NPRS and the Patients Global Impression of Change scale [weekly data is plotted in graphs found in Appendix H].

Table 1: Participant Demographics

<table>
<thead>
<tr>
<th>Participant</th>
<th>Gender</th>
<th>Age (years)</th>
<th>BMI (kg/m²)</th>
<th>Pain Duration (years)</th>
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</thead>
<tbody>
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<td>55</td>
<td>33</td>
<td>10</td>
</tr>
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<td>2</td>
</tr>
<tr>
<td>9</td>
<td>F</td>
<td>59</td>
<td>23</td>
<td>20</td>
</tr>
</tbody>
</table>

Mean (SD) 48.4 (12.7) 27 (5.3) 9.4 (7.8)

Note: Where F=Female, M=Male, BMI=Body Mass Index
Overall Outcome Measures over Intervention

Participant 8 was the only participant who made important improvements in all outcome measures from pre to post-intervention intervention (maintaining all changes at follow-up. See Table 2). Over the intervention period, Participant 2 achieved minimal clinically important change (MCID) in all measures except for flexibility. Three participants (P3, P6, P7) improved in 2 of the 4 outcome measures. Participants 1 and 4 improved only in flexibility measures. Participant 9 and Participant 5 did not achieve MCID in any outcomes measures over the intervention period. No participants deteriorated by clinically important levels over the intervention period.

Table 2: Summary of Important Change from Baseline to Post Intervention

<table>
<thead>
<tr>
<th>Participant</th>
<th>Flexion Relaxation</th>
<th>Pain Intensity</th>
<th>Disability</th>
<th>Sit and Reach</th>
<th>Total Outcomes Improved</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>N/C</td>
<td>N/C</td>
<td>N/C</td>
<td>I</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>N/C</td>
<td>3</td>
</tr>
<tr>
<td>3</td>
<td>N/C</td>
<td>N/C</td>
<td>I</td>
<td>I</td>
<td>2</td>
</tr>
<tr>
<td>4</td>
<td>N/C</td>
<td>N/C</td>
<td>N/C</td>
<td>I</td>
<td>1</td>
</tr>
<tr>
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<td>N/C</td>
<td>N/C</td>
<td>N/C</td>
<td>N/C</td>
<td>0</td>
</tr>
<tr>
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<td>N/C</td>
<td>I</td>
<td>N/C</td>
<td>I</td>
<td>2</td>
</tr>
<tr>
<td>7</td>
<td>I</td>
<td>I</td>
<td>N/C</td>
<td>N/C</td>
<td>2</td>
</tr>
<tr>
<td>8</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>I</td>
<td>4</td>
</tr>
<tr>
<td>9</td>
<td>N/C</td>
<td>N/C</td>
<td>N/C</td>
<td>N/C</td>
<td>0</td>
</tr>
</tbody>
</table>

Total Improved 3 4 3 5 15

N/C indicates No Change; I indicates a change that exceed the minimum clinically important Improvement.
Flexion Relaxation

Over the intervention period, three of the nine participants (P2, P7, and P8) achieved a significant improvement in FR (Table 3). At follow-up, only one participant (P8) maintained a significant change. Participant 1 made a gradual, but insignificant, improvement over the intervention, and achieved a significant change at follow-up.

When results for all participants are pooled, FR measures showed a non-significant improvement (Wilcoxon-rank test $T = 0, p = 0.139, d = 0.49$) from baseline ($\mu = 1.81, SD = 0.6; M = 1.7, CI = 1.3 - 2.0$) to post-intervention ($\mu = 2.75, SD = 1.7; M = 2.4, CI = 1.5 to 3.8$). However, a significant overall improvement ($T = 1, p = 0.027, d = 0.834$) was observed from baseline to follow-up ($\mu = 3.3, SD = 2.2; M = 2.8, CI = 1.5 to 5.7$).

Table 3: Flexion Relaxation Scores (Extension Relaxation Ratio)

<table>
<thead>
<tr>
<th>Participant</th>
<th>Baseline Score</th>
<th>Post-Intervention Score</th>
<th>Baseline vs. Post Change</th>
<th>Follow Up Score</th>
<th>Baseline vs. Follow Up Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>3.2</td>
<td>4</td>
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<td>2.5*</td>
</tr>
<tr>
<td>2</td>
<td>1.9</td>
<td>3.5</td>
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<td>3</td>
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<td>0.8</td>
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<td>n/a</td>
</tr>
<tr>
<td>4</td>
<td>1.7</td>
<td>2.4</td>
<td>0.7</td>
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<td>n/a</td>
</tr>
<tr>
<td>5</td>
<td>1.8</td>
<td>1.3</td>
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<td>2.8</td>
<td>1</td>
</tr>
<tr>
<td>6</td>
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<td>2.1</td>
<td>0.4</td>
</tr>
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<td>7</td>
<td>1.3</td>
<td>2.5</td>
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<td>0.2</td>
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<tr>
<td>8</td>
<td>1.2</td>
<td>6.5</td>
<td>5.3*</td>
<td>6.9</td>
<td>5.7*</td>
</tr>
<tr>
<td>9</td>
<td>1.3</td>
<td>1.6</td>
<td>0.3</td>
<td>1.3</td>
<td>0</td>
</tr>
</tbody>
</table>

Mean (SD) 1.81 (0.6) 2.75 (1.7) 1.3 (1.6) 3.3 (2.1) 1.5 (2)

An * indicates a significant change of 1.6 or more (two or more Standard Deviations [SD] from baseline group average of 0.6)
Pain Intensity

Four of the nine participants, (P2, P6, P7, P8) achieved a clinically important change in pain intensity (two or more points) over the course of the intervention (Table 4). All four participants maintained these improvements at follow-up. Two additional participants (P1, P5) had achieved a MCID at follow-up despite reporting no change over the intervention period. Only one participant (P4) showed an important deterioration in pain intensity at follow-up compared to baseline and post intervention measures. However, at the time of the follow-up measures, this participant was awaiting a meniscal operation for his knee, which had recently increased in pain.

When the results from all participants are pooled, average pain intensity compared with baseline ($\mu = 4.7$, SD 1.3; M = 5, CI = 4 - 6), was significantly lower ($T = 2, p = 0.017, d = 0.8$) at post-intervention ($\mu = 2.89$, SD 2.3; M = 3, CI = 1 to 5) and even lower ($T = 1, p = 0.039, d = 0.69$) at follow up ($\mu = 2.7$, SD 2.0; M = 2, CI = 1.5 to 4).

Table 4: Pain Intensity (NPRS scores)

<table>
<thead>
<tr>
<th>Participant</th>
<th>Baseline Score</th>
<th>Post-Intervention Score</th>
<th>Baseline vs. Post Change</th>
<th>Follow Up Score</th>
<th>Baseline vs. Follow Up Change</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
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<td>1</td>
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<td>0</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
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<td>1</td>
<td>7</td>
<td>-2*</td>
</tr>
<tr>
<td>5</td>
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<td>0</td>
<td>4</td>
<td>2*</td>
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<tr>
<td>6</td>
<td>5</td>
<td>1</td>
<td>4*</td>
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<td>4</td>
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<tr>
<td>9</td>
<td>4</td>
<td>3</td>
<td>1</td>
<td>3</td>
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</tr>
</tbody>
</table>

Mean (SD) 4.7 (1.3) 2.9 (2.3) 1.8 (1.6) 2.8 (2) 1.9 (2)

An * indicates a clinically important change of 2 or more points
Functional Disability

Clinically important improvements in ODI were achieved in three of the nine participants (P2, P3, P8) at post intervention, compared to baseline scores (Table 5). These three participants maintained improvements at follow-up.

Two participants (P7, P9) reported more disability post-intervention compared to baseline (although this change did not achieve MCID). Participant 4 deteriorated from post-intervention to follow-up to the level of MCID, however, this was the aforementioned participant scheduled for knee surgery.

The pooled results of all participants showed average ODI scores were statistically significantly higher ($T = 1, \rho = 0.035, d = 0.70$) at baseline ($\mu = 20.4, SD 9.2; M = 22, CI = 12 to 29$) than at post-intervention ($\mu = 12, SD 7.7; M = 12, CI = 6 to 17$). However, differences between baseline and follow up were not statistically significant ($\mu = 13.8, SD 9.8; M = 14, CI = 7 to 21, T = 1, \rho = 0.141, d = 0.49$).

Table 5: Disability (Oswestry scores)

<table>
<thead>
<tr>
<th>Participant</th>
<th>Baseline Score</th>
<th>Post-Intervention Score</th>
<th>Baseline vs. Post Change</th>
<th>Follow Up Score</th>
<th>Baseline vs. Follow Up Change</th>
</tr>
</thead>
<tbody>
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<td>16</td>
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<td>14</td>
<td>8</td>
</tr>
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<td>32</td>
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<td>24*</td>
<td>14</td>
<td>18*</td>
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<td>22</td>
<td>26</td>
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<td>-8</td>
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</tbody>
</table>

Mean (SD) 20.4 (9.2) 12 (7.7) 10.2 (8.9) 13.8 (9.8) 6.7 (11.2)

An * indicates an important change of 10 or more points
Flexibility

Over the intervention period, five participants (P1, P3, P4, P6, P8) achieved a clinically important change in flexibility as defined by a 4 cm change in SR scores (Table 6). Of these five, three participants (P1, P6, P8) maintained an important improvement at follow-up. The two remaining participants (P3, P4) were not available for follow-up measures. Compared to the baseline score, participant nine showed a clinically important deterioration in flexibility.

When the results of all participants were pooled, flexibility improved significantly ($T = 0, p = 0.021, d = 0.77$) from baseline ($\mu = 19.2, SD = 13.9; M = 19, CI = 6 to 32$), to post-intervention ($\mu = 24.6, SD 11.8; M = 27, CI = 13.5 to 36.3$), as well as at follow-up ($\mu = 28.2, SD 9.0; M = 25, CI = 24 to 36, T = 1, p = 0.207, d = 0.48$).

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Table 6: Sit and Reach scores (cms)

<table>
<thead>
<tr>
<th>Participant</th>
<th>Baseline Score</th>
<th>Post-Intervention Score</th>
<th>Baseline vs. Post Change</th>
<th>Follow Up Score</th>
<th>Baseline vs. Follow Up Change</th>
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<td>18</td>
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<td>14</td>
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</tbody>
</table>

Mean (SD) 19.2 (13.9) 24.6 (11.7) 5.4 (5.3) 28.2 (9) 3.7 (6.9)

An * indicates an important change of 4 cm or more
**Patients Global Impression of Change**

At the completion of the study, four of the nine participants (P2, P3, P6, P8) reported their progress over the intervention as having at least a ‘moderate and noticeable change’ in symptoms, scoring five or over on the Patients Global Impression of Change scale (PGIC). Each of the individual four participants achieved MCID for at least two of the four outcome measures. Only two of the participants reporting at least a moderate change showed significant improvement in FR measures over the study (P2 and P8).

Of the five participants who reported a change in PGIC that was not meaningful, participant seven showed FR that was significantly improved over the study period and two participants (P1, P5) significantly improved FR from post-intervention to follow-up.

**Adherence to At-Home Stretching**

All participants reported at least 90% adherence to the daily stretching over the five week intervention period, with five participants reporting 100%. The most days missed over the intervention period by any participant was 3 days (out of approximately 35 days).
DISCUSSION

The primary aim of this study was to investigate if a SEMGAS programme would improve FR in individuals with chronic LBP. The second aim was to investigate if improved FR was associated with improved pain intensity, disability and sit and reach measures. The results from the present study were not uniform across all of the participants, however, over the course of the intervention three of the nine participants significantly improved FR, with all three achieving clinically important changes in pain intensity scores and two improving in perceived disability.

Flexion Relaxation and Pain Are Related

Impaired FR has been attributed to a mechanism of continuous paraspinal contraction, which is common in patients with current pain (Geisser et al 2004; Sihvonen et al 1998; Thomas & France 2008). It was hypothesised that the continued paraspinal muscle activity may actually contribute to the pain, by preventing adequate muscle recovery. Therefore, it was expected that decreased pain intensity scores would accompany improved FR. The current study supports the positive association between pain intensity and FR, as all participants improving FR also improved pain intensity scores. It is not possible to determine if changes in FR caused a decrease in pain intensity or if the intervention had indirectly affected pain and subsequently caused an improvement in FR. Common models of pain adaptation predict that increased muscle activity will be persistent in chronic LBP patients (Travell et al 1942; van Dieën et al 2003; Zedka et al 1999). Limited recovery time from continued contraction is implicated in pain production, presumably owing to the accumulation of muscle by-products from the continued contraction (Kaufman et al 1983; Mense 1993). Utilising SEMGAS and training the muscles to relax effectively may permit more appropriate paraspinal relaxation. Pain intensity potentially reduces as muscle function normalises. Although the current study included a small sample (n=9), an association between improved FR and improved pain intensity appears operant in responding individuals, in that all three participants that improved FR also improved pain intensity measures. These results reflect the recent studies by Neblett and colleagues that observed significant improvements in pain intensity in larger populations, when SEMGAS was employed concurrently with a comprehensive functional restoration programme (Neblett b et al 2009; Neblett et al 2003).

Although improved pain intensity was consistently observed with improved FR, one participant improved in pain intensity scores but not FR. Although this finding contrasts the protective splinting behaviour, (Main & Waddell 1991; Sullivan et al 2001), the finding is more in line with other reports of low associations between improved FR and improved pain measures, especially in individuals with chronic LBP (Ahern et al 1990; Neblett a et al 2009; Triano & Schultz 1987). However, researchers have typically utilised other rehabilitation modalities that have not specifically addressed FR, and have instead employed FR as an outcome measure (Marshall & Murphy 2008; McGorry & Lin 2012; Neblett a et al 2009; Neblett et al 2003; Owens et al 2011; Sihvonen et al 1991; Watson et al 1997).
Directing an intervention to specifically affect FR has only recently been explored. The current study and Neblett et al’s 2010 study are the only investigations aimed at directly improving FR. As SEMGAS aims to directly affect FR, the current results suggest that improved FR may influence pain intensity scores.

Clinical Relevance

Over the five week intervention period, three participants achieved a clinically important improvement in disability measures, two of which also improved in FR measures. Participant 7 improved FR however disability measures did not improve. Interestingly all three improving disability, also reported important improvement in PGIC measures.

The intervention appeared to be effective in reducing perceptions of disability for some (3/9) of the participants in the study. Interestingly, improvements in FR were not apparent for all three of these participants to achieve this improvement, as only two of the three displayed an improved FR. These results reflect the multifactorial complexity of disability (Bogduk 2006), in that improvement in a single outcome measure can seldom be expected to exclusively correlate with an improved disability.

The participant reporting the largest improvement over the intervention (P8), significantly improved in all measured outcomes, was the youngest participant involved in the study, and had symptoms for the shortest duration (two years). Participant 8 also started with the lowest baseline FR ratio value, indicating a strong maladaptive neuromuscular response to pain. However, in the small sample of participants involved in this study, factors of baseline FR, age and pain duration did not appear to strongly influence outcome measures for most other participants in this, or other studies.

In clinical practice, it is fundamental to determine whether a patient has improved, deteriorated or stayed the same. Using a Global Impression of Change scale, patients are able to indicate what they consider important, rather than measuring a pre-determined aspect of health (Kamper, 2009). Half (2/4) of the participants who felt the intervention achieved a meaningful change in their chronic LBP (as measured with the PGIC), significantly improved in FR, while three of the four participants improved SR scores. This may suggest two plausible mechanisms implicated in improvement following a SEMGAS programme: 1) improved range of motion (ROM), irrespective of maintaining abnormal neuromuscular control (abnormal FR); or 2) altering the function of the neuromuscular system, resulting in improved FR.

Range of Motion and Flexion Relaxation

Due to the inclusion of a stretching component in the SEMGAS programme, it was anticipated ROM would improve in participants following the intervention. Flexibility was improved by clinically relevant levels in the majority (5/9) of participants over the intervention period. However, improvements in SR appeared to be poorly associated with any other outcome measures in the current study.

It is hypothesised that persistent muscle activation in response to pain leads to decreased ROM (Thomas & France 2008). Subsequently this may prevent initiation of the stretch reflex (Colloca & Hinrichs 2005;
Hashemirad et al (2009). It was anticipated that an improvement in ROM from the intervention may allow for the initiation of the stretch reflex and myoelectric reduction at MVF (Kaigle et al 1998). Initially, four of the nine participants had normal (23 to 28 cm) SR scores (López-Miñarro et al 2009). These participants were therefore likely to have achieved sufficient flexion to initiate the stretch reflex, however persistent muscle activation continued. In these cases, rather than being influenced by the degree of lumbar motion, the ability to achieve FR was likely influenced by altering neural adaptation.

In the current study all those achieving FR, achieved normal SR scores following the intervention (P2 and P7 at post intervention and P8 at follow up), although not all participants with normal ROM displayed normal FR. Recent literature supports this observation, with one study reporting 100% of participants displaying normal FR, also achieved normal ROM (Neblett et al 2003). The authors, however, reported considerably lower sensitivity, as some participants displayed impaired FR despite achieving normal ROM (Neblett b et al 2009; Neblett et al 2003). Participants with normal ROM but abnormal FR somewhat contradict the ‘protective’ role of the increased paraspinal activity, as despite improved ROM, participants still fail to reduce muscular ‘splinting’. In their investigation, Zedka et al (1999) found elevated muscle tone in painful situations, in spite of controlling for splinting behaviour and producing movements. It therefore seems likely that improved FR was not attributed to changes in ROM alone for all participants, but rather influenced by the neuromuscular aspects of the SEMGAS programme.

Neuromuscular Changes and Flexion Relaxation

Restricted ROM associated with pain could be attributed to exaggerated stretch reflexes (Cram & Kasman 1990; 1998). Therefore treatment theoretically resulted in a lower level of activity to reach the stretch receptor threshold, as the effect of training could have resulted in a decreased receptor drive on the motor system during the stretch.

A 12-week, supervised swiss-ball exercise intervention described by Marshall and Murphy (2008) was intended to influence spindle output, similar to the aim of a SEMGAS programme. Stimulation of type III and IV afferents, which are important pain generators (Johansson & Sojka 1991; Mense 1993) have the net effect of resetting the sensitivity of the receptors over time. The altered receptor sensitivity decreases the local output, contributing to decreased activity at MVF (Lowndes et al 1979; Pickar & Wheeler 2001).

As biofeedback has the unique ability to influence both the physical and psychosocial aspects of pain, improvement following SEMGAS may be due to influencing the central nervous system (Robinson & Riley 1998). As Zedka et al (1999) identified impaired FR following pain production involving no actual damage to spinal structures, as well as when overcoming movement limitations, they proposed a centrally mediated state, or “pain mode”, involving non-voluntary muscle guarding. Although the positive results may be explained by the natural progression of chronic LBP, it appears SEMGAS may affect both the musculoskeletal and central nervous systems, resulting in reduced paraspinal activity at MVF.
Dose

Effective treatment dose is of interest as chronic LBP is notoriously difficult and costly to treat (Lehmann et al 1993). Contact hours required to treat LBP are commonly high, inefficient and costly. In order to contain costs and improve efficiency of treatment, it is beneficial to identify the lowest effective dose for any given treatment.

Other studies investigating the effect of various interventions on FR measures have ranged in dose from a single session (Lalanne et al 2009), up to 240 hours (Neblett et al 2010). The present intervention was specifically designed to be brief, involving a total of only two hours of contact time over a five-week period. The results of this study found that three of nine participants had significant improvements in FR, as well as in the majority of outcome measures over the intervention. Therefore a less intensive intervention such as the one described which requires little consultation time commitment may be effective in improving FR, pain intensity, disability and sit and reach in some patients with chronic LBP. Recent literature supports these findings. In Marshal and Murphy’s (2008) study, researchers were able to identify FR improvement after week-four of involvement in active exercise, which plateaued by week-eight. Neblett et al’s 2010 study, normalised FR in chronic LBP patients in an average of 2.4 (SD 1.4) SEMGAS sessions, with a maximum of six sessions required to attain FR (session duration was not described). However, 10 of the 47 participants were unable to achieve FR despite multiple sessions.

CONCLUSION

This case series demonstrates that when used alone and with a limited dose, SEMGAS may be effective at improving FR in some individuals with chronic LBP. The results also suggest improved FR is associated with improved pain intensity and measures of disability. It appears that improvements in FR are not exclusively attributable to improved ROM, and a more neurological mechanism was likely affected. Future research in the form of small-scale clinical trials, or a randomised control trial with larger samples, would improve the generalizability of the results.

Although the results of this small-scale case series cannot be generalized to the larger chronic LBP population, preliminary evidence from this study does suggest that SEMGAS can provide clinical benefits to some individuals with chronic LBP and impaired FR.


Section Three: Appendices
APPENDIX A: RECRUITMENT ADVERTISING
Does Back Pain Interfere With Your Everyday Life?

You are invited to participate in a research project. The research aims to investigate the effectiveness of a programme at retraining muscle patterns in the low back and may help alleviate back pain.

You must be aged between 16-65 and experience back pain that makes it difficult to do everyday activities on more than half the days in the last 3 months.

You may be required to attend 6 weekly 1 hour training sessions at UNITEC (Carrington Rd).

These sessions will involve being guided through evidence based stretching movements, while the activity of your low back muscles is being painlessly recorded.

If you are interested in receiving more information regarding this study, please visit www.backpain.getparticipants.com or contact me on the details provided.

Aimee Moore  email- backpain@getparticipants.com  mobile- 027 6722275
Unitec researchers looking for people with low back pain

Low back pain is a common occurrence in today's society, with approximately 85% of the population experiencing back pain at some stage of their lives. These back pain symptoms may range from mild nagging pain, to severe on-going disability. Back pain can interfere with work, leisure and social and family activities. For many people, the fear of exacerbating their back pain is enough to deter them the doing the things they love most.

Sometimes we know what caused back pain, such as an old sports injury, however for many individuals the cause of their pain is unknown. More often than not, the pain resolves over a couple of weeks. For about 5% of back pain sufferers, the pain may become chronic and interfere with a fulfilling and satisfying life. Unfortunately, despite the high incidence of low back pain, we are still a long way from knowing how to cure many types of back pain.

Understanding the mechanisms of back pain and exploring the ways in which it can be treated through research is the only way to help improve and treatment options for those suffering with pain. Currently, Osteopathic researchers at Unitec, Auckland, are exploring a couple of novel treatment options for low back pain and are currently looking for volunteers.

Unitec, New Zealand, would like to invite sufferers of low back pain to participate in their research. Participants need to have had back pain for at least three months and have not had surgery on their spine.

If you think you may be eligible and are willing to donate some of your time to participate in one of their studies, then please visit www.getparticipants.com/unitec
Appendix B: Information Sheet and Consent Form
Evaluation of the Efficacy of a Surface Electromyography assisted biofeedback program at correcting muscle activation patterns and reducing pain

Researcher: Aimee Moore. UNITEC New Zealand.

You are invited to participate in our research investigation. Please read carefully through this information sheet before you make a decision about volunteering.

My name is Aimee Moore and I am a Masters student in Osteopathy at UNITEC. As part of this degree I am undertaking a research project leading to a thesis.

The project I am undertaking is evaluating if a surface electromyography (sEMG) assisted training programme is effective at correcting muscle patterning in the low back and if this reduces pain.

Why this information is important:

Low back pain (LBP) affects 70-95% of the adult population, with 6-10% of cases, progressing to chronicity. It has been found in previous studies that there is an altered muscle-firing pattern in people with LBP compared to those without.

Flexion-relaxation (FR) is a stereotypical muscle pattern where low back spinal muscles relax at maximal voluntary trunk flexion (touching your toes). This is consistently observable, in most pain free patients.

Absent FR, where there is increased muscular activity at full bending, has been shown to be a constant and predictable pattern in patients with chronic LBP.

A recent study has identified sEMG biofeedback training as a way to retrain the low back muscles to relax while bending, correcting the FR response. I am aiming to add to this evidence, and to discover if there is a link between changing FR and decreasing pain scores.

What will happen as part of this study?

If you volunteer to take part in this experiment, you will be asked to expose your lower back and the sEMG equipment will be applied. The device looks like this:

Four electrodes will be placed on the skin over muscles on either side of the low back. These will be held against the skin with some tape. These electrodes connect to a laptop which records information about the tone of the underlying muscle. Participants will be directed through a stretching protocol by the researcher, and will receive instantaneous information via the sEMG equipment. The researcher will encourage you to change the tone of the low back muscles in an attempt to retrain the muscles to activate correctly. The stretching protocol requires the participant to bend forward towards their toes, then return to standing.
Taking part in this study will require you to attend 8 sessions at the Osteopathic clinic at UNITEC on Carrington road. These sessions will take place weekly and will last approximately 1 hour. You will also be asked to partake in a daily stretching routine at home, which will take approximately 15 minutes, for the six-week duration of the study. That is; eight, one-hour sessions at UNITEC, plus six weeks of 15 minutes of daily stretching at home.

**Who may participate?**

You are eligible to participate if you:

- Are aged between 16 and 65 years of age.
- Have suffered with chronic low back pain for 3 months or longer, and experience some back pain on more than half the days.
- Are willing to give informed written consent.
- Are able to partake in the training programme regularly (Once a week at the UNITEC osteopathy clinic located in Mt Albert).
- Demonstrate LBP symptoms within a physical examination (undertaken by the researcher).
- Present with impaired Flexion Relaxation response. (This will be confirmed by the researcher, with readings from the sEMG equipment.)
- Present with mild disability from back pain. (This information will be obtained from a questionnaire you will be asked to complete as part of the data collection.)

Unfortunately you are unable to participate if you:

- Have any known or suspected spinal or muscular pathologies (including spinal stenosis, spinal surgery, neurological symptoms, osteoporosis, lower limb musculoskeletal injuries or surgery, disc injuries).
- Are pregnant or have given birth within the last 6 months.
- Are unable to regularly attend the stretching protocol with the researcher.

Please inform the researcher if any of the above pertains to you.

**You have the right to withdraw your data from this project at any time, until one week after your final data collection. This can be done by contacting the researcher below.**

A summary of the final report will be available to you if you are interested.

**Information and concerns**

If you require any further information about this project, please contact me by phone or email.

**Researcher information:**

<table>
<thead>
<tr>
<th>Aimee Moore</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mobile: 0276722275</td>
</tr>
<tr>
<td>Email: <a href="mailto:backpain@getparticipants.com">backpain@getparticipants.com</a></td>
</tr>
</tbody>
</table>
Evaluation of the efficacy of a surface electromyography assisted biofeedback program at correcting muscle activation patterns and reducing pain

This research project aims to evaluate if a surface electromyography (sEMG) assisted training programme is effective at correcting muscle patterning in the low back and if this reduces pain.

Name of Participant: ____________________  D.O.B : _____ / _____ / ______

I have been given and have understood an explanation of this research project.

I confirm that I have read and understand the information sheet.

I have had the opportunity to consider the information, ask questions and have had these answered satisfactorily.

I understand that I may withdraw myself (or any information I have provided) from this project (before data collection and analysis is complete) without having to give reasons or without penalty of any sort.

I understand that any information I provide will be kept confidential to the researcher and the supervisor.

I understand the published results will not use my name, and that no opinions will be attributed to me in any way that will identify me.

I understand that the data I provide will not be used for any other purpose or released to others without my written consent.

I understand that I can see the finished research document.

I have had time to consider everything and I give my consent to be a part of this study.

I know whom to contact if I have any questions or concerns about this study.

I agree to take part in the above research study.

Participant Signature: ________________  Date: _____ / _____ / ______

Study explained by: ________________

Signature: __________________________  Date: _____ / _____ / ______
APPENDIX C: ETHICAL APPROVAL
Aimee Moore  
2/11 Fir Street  
Waterview  
Auckland 1026

24.8.2011

Dear Aimee,

Your file number for this application: 2011-1193
Title: Evaluation of the efficacy of a surface electromyography assisted biofeedback programme at correcting muscle activation patterns and reducing chronic low back pain.

Your application for ethics approval has been reviewed by the Unitec Research Ethics Committee (UREC) and has been approved for the following period:

Start date: 11.8.2011  
Finish date: 11.8.2012

Please note that:

1. The above dates must be referred to on the information AND consent forms given to all participants.

2. You must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.

Yours sincerely,

Scott Wilson  
Deputy Chair, UREC

cc: Rob Moran  
Cynthia Almeida
Dear Aimee,

Re: Request for amendments

Your file number for this application: 2011-1193
Title: Evaluation of the efficacy of a surface electromyography assisted biofeedback programme at correcting muscle activation patterns and reducing chronic low back pain.

Your request for amendments to your research project has been reviewed by the Unitec Research Ethics Committee (UREC) and has been approved for the following period:

Start date: 22.3.12
Finish date: 11.8.12

Please note that:

1. The above dates must be referred to on the information AND consent forms given to all participants.

2. You must inform UREC, in advance, of any ethically-relevant deviation in the project. This may require additional approval.

You may now commence your research according to the protocols approved by UREC. We wish you every success with your project.

Yours sincerely,

Scott Wilson
Deputy Chair, UREC

cc: Rob Moran
Cynthia Almeida
APPENDIX D: SUPPLEMENTARY INFORMATION OF OUTCOME MEASURES
Pain Intensity

The 11-point Numeric Pain Rating Scale (NPRS) was used to monitor pain intensity. The scale ranges from 0 (“no pain”) to 10 (“worst pain imaginable”). Each measure required the participant to rate the intensity of their pain over the past week on this scale. Minimal clinically important difference (MCID) for the NPRS is consistently reported as two points (Childs, Piva, & Fritz, 2005; Ostelo et al., 2008; Wyrwich, Tierney, & Wolinsky, 1999).

Functional Disability

The Revised Oswestry Disability Index (ODI) was used to measure functional status. This self-reported questionnaire consists of 10 different sections relating to different activities of daily living. Higher scores correspond to greater disability. The revised ODI, which replaced the original versions question of sex-life to employment/homemaking, has been shown to be reliable and valid in patients with low back pain (Fairbank & Pynsent, 2000; Fritz & Irrgang, 2001). The MCID for the ODI is reportedly between 6-15 points (Burns, Mintken, Austin, & Cleland, 2011; Fritz & Irrgang, 2001; Maughan & Lewis, 2010; Ostelo et al., 2008). A threshold of 50% improvement on the ODI has also been proposed to be a valid measure for defining a successful outcome for patients with chronic LBP (Childs et al., 2005; Fritz, Hebert, Koppenhaver, & Parent, 2009; Ostelo et al., 2008). For this study, a 10-point change is used to show clinically relevant change.

Flexibility

The Sit and Reach (SR) test was employed to assess the combined flexibility of the lumbar and hamstring muscles. It has a high validity and has been found to be comparable to more expensive laboratory equipment when assessing flexibility (Predrag, Nemanja, Bobana, Nenad, & Ivan, 2010).

A ruler was attached to the top of the box with the markings on the ruler positioned so that the 23 cm mark (a standard position) represented the point at which the participants’ fingertips were in line with their toes. All measures were therefore recorded as a positive figure.

Participants were seated on the floor with knees fully extended and ankles in neutral against the box. Participants were asked to slowly reach forward along the top of the box as far as possible while the knees remained extended. The score was recorded in centimetres (to the nearest 0.5 cm) as the final position of the tips of the fingers against the ruler. Previous studies suggest a MCID for SR to be a 4 cm change (López-Miñarro, Muyor, Belmonte, & Alacid, 2012; López-Miñarro, Rodrigues-Garcia, & Andujar, 2009).

Patients Global Impression of Change Scale

At the completion of the follow-up measures, participants completed the Patients Global Impression of Change Scale (PGIC) form to assess changes in their general wellbeing over the intervention. PGIC was measured with a 7-point scale: 1) No change (or condition has become worse); 2) Almost the same, hardly any change at all; 3) A little better, but no noticeable change; 4) Somewhat better, but the change has not made any real difference; 5)
64

Moderately better and a slight but noticeable difference; 6) Better and a definite improvement that has made a real and worthwhile difference; 7) A great deal better, a considerable improvement that has made all the difference.

PGIC scales are considered to be highly reliable ($r = 0.72$ to $r = 0.90$) (Stratford, Binkley, Solomon, Gill, & Finch, 1994; Watson et al., 2005) and sensitive with high face validity (Davidson & Keating, 2002; Kamper, 2009; Kovacs et al., 2007, 2008). In relation to clinical relevance, strong correlations with patient satisfaction measures have been reported (Spearman correlation coefficients of 0.56 to 0.77) (Fischer, Stewart, & Bloch, 1999), and it may also correlate with physical performance (Osoba, Rodrigues, Myles, Zee, & Pater, 1998). The scale is simple to use, and allows the patient to decide for themselves what they consider to be a meaningful or significant change (Kamper, 2009).

The PGIC directly measures the opinion of the patient on their level of change (including improvement, deterioration or no change) at the end of a trial (Kamper, 2009). A score of five or greater is considered an improvement (Field, Newell, & McCarthy, 2010; Hurst & Bolton, 2004).

References


APPENDIX E: PATIENTS GLOBAL IMPRESSION OF CHANGE
Patients' Global Impression of Change (PGIC) scale.

Name: ___________________________ Date: _______________ DOB: ______

Chief Complaint: ______________________________________

Since beginning treatment at this clinic, how would you describe the change (if any) in ACTIVITY LIMITATIONS, SYMPTOMS, EMOTIONS and OVERALL QUALITY OF LIFE, related to your painful condition? (tick ONE box).

- No change (or condition has got worse) [ ] 1
- Almost the same, hardly any change at all [ ] 2
- A little better, but no noticeable change [ ] 3
- Somewhat better, but the change has not made any real difference [ ] 4
- Moderately better, and a slight but noticeable change [ ] 5
- Better, and a definite improvement that has made a real and worthwhile difference [ ] 6
- A great deal better, and a considerable improvement that has made all the difference [ ] 7

In a similar way, please circle the number below, that matches your degree of change since beginning care at this clinic:

<table>
<thead>
<tr>
<th>Much Better</th>
<th>No Change</th>
<th>Much Worse</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
<td></td>
<td></td>
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</tbody>
</table>

Patient’s signature: ___________________________ Date: _______________

APPENDIX F: OUTCOME MEASURES FORM
Welcome and thank you for participating in this study.

Please take care to read the questions carefully and answer them truthfully. If you are unsure of how to answer any of the questions, please mark with a (?), and we will clarify in the interview.

The following Questions have been designed to give your therapist information as to how your back pain affects your ability to manage everyday life. Please answer every question by placing a mark in the one box that best describes your condition today. We realize you may feel that 2 of the statements may describe your condition, but please mark only the box that most closely describes your current condition.

### Pain Intensity

- I can tolerate the pain I have without having to use pain medication
- The pain is bad, but I can manage without having to take pain medication
- Pain medication provides me with complete relief from pain
- Pain medication provides me with moderate relief from pain
- Pain medication provides me with little relief from pain
- Pain medication has no effect on my pain

### Personal Care (e.g., Washing, Dressing)

- I can take care of myself normally without causing increased pain
- I can take care of myself normally, but it increases my pain
- It is painful to take care of myself, and I am slow and careful
- I need help, but I am able to manage most of my personal care
- I need help every day in most aspects of my care
- I do not get dressed, I wash with difficulty, and I stay in bed
Lifting

- I can lift heavy weights without increased pain
- I can lift heavy weights, but it causes increased pain
- Pain prevents me from lifting heavy weights off the floor, but I can manage if the weights are conveniently positioned (e.g., on a table)
- Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned
- I can lift only very light weights
- I cannot lift or carry anything at all

Walking

- Pain does not prevent me from walking any distance
- I have pain while walking, but I can still walk my required normal distance
- Pain prevents me from walking long distance
- Pain prevents me from walking intermediate distance
- Pain prevents me for walking even short distances
- Pain prevents me from walking at all

Sitting

- I can sit in any chair as long as I like
- I can only sit in my favourite chair as long as I like
- Pain prevents me from sitting for more than 1 hour
- Pain prevents me from sitting for more than 1/2 hour
- Pain prevents me from sitting for more than 10 minutes
- Pain prevents me from sitting at all

Employment / Homemaking

- My normal homemaking / job activities do not cause pain
- My normal homemaking / job activities increase my pain, but I can still perform all that is required of me
- I can perform most of my homemaking / job duties, but pain prevents me from performing more physically stressful activities (e.g., lifting, vacuuming)
- Pain prevents me from doing anything but light duties
- Pain prevents me from doing even light duties
- Pain prevents me from performing any job or homemaking chore
Sleeping

- Pain does not prevent me from sleeping well
- I can sleep well only by using pain medication
- Even when I take medication, I sleep less than 6 hours
- Even when I take medication, I sleep less than 4 hours
- Even when I take medication, I sleep less than 2 hours
- Pain prevents me from sleeping at all.

Social Life

- My social life is normal and does not increase my pain
- My social life is normal, but it increases my level of pain
- Pain prevents me from participating in more energetic activities (e.g., sports, dancing)
- Pain prevents me from going out very often
- Pain has restricted my social life to my home
- I have hardly any social life because of my pain.

Travelling

- I can travel anywhere without increased pain
- I can travel anywhere, but it increases my pain
- My pain restricts my travel over 2 hours
- My pain restricts my travel over 1 hour
- My pain restricts my travel to short necessary journeys under 1/2 hour
- My pain prevents all travel except for visits to the physician/therapist or hospital

Standing

- I can stand as long as I want without increased pain
- I can stand as long as I want, but it increases my pain
- Pain prevents me from standing for more than 1 hour
- Pain prevents me from standing for more than 1/2 hour
- Pain prevents me from standing for more than 10 minutes
- Pain prevents me from standing at all

In the past week how bothersome has your Low Back Pain been?

(0–10, where 0 = no pain, 10 = worst pain imaginable)
APPENDIX G: HOME STRETCH DIARY
STRETCH PROTOCOL

Find a comfortable stretching area with enough space to move freely.
Stand with feet shoulder width apart. Slowly roll forward, first bringing the chin to
the chest, and continue down, reaching your hands towards the floor. When you feel
a gentle stretch in the low back, stop the movement and maintain the
position.
Concentrate on deep breathing and relaxing the muscles being lengthened.
Let your arms hang loose, with your knees staying soft.
If it is comfortable to do so, hold the stretch for at least 30 seconds, no more
than 2 minutes.

TIPS

If you feel pain in the back of your legs, bend your knees slightly.
Breathe deeply, using your diaphragm, letting the abdomen expand as you inhale.
As you exhale, consciously relax your neck, torso, shoulders and low back.
Slowly and gently further your stretch, reaching towards the ground, if you begin
to feel a decrease in tension in the low back.

NOTE: Do not stretch if you feel any pain.
The ideal stretching amount is individual and depends on your level of flexibility.
If you feel pain, please decrease your stretch. If pain persists, please stop
stretching and find a comfortable position.

STRETCHING

The aim of this treatment is to interrupt this cycle, by replacing the muscle over-reaction,
with a more adequate muscular response.
When a muscle is stretched, so is the muscle spindle (which, from within the muscle,
regulates the tension in the muscle).
The muscle spindle is involved in the stretch reflex, which attempts to resist the change in
muscle length, by causing the stretched muscle to contract, especially if stretched quickly. This is
to protect the spine from moving too fast past the limits of its movement. It is thought that with
low back pain, an overactive reflex is initiated, called the reflex spasm cycle. Stretch leads to
spasm, which leads to reduced range of motion and muscular shortening, increasing stretch
and spasm and so on...

A useful metaphor liken's this reflex to letting an anchor out from a boat. As gravity pulls the
anchor to the ground, a deckhand or machine will slowly lower the anchor out. This reduces the
sudden 'jolt' when the anchor reaches the ground. The low back muscles work like the
deckhand, supporting the spinal column and allowing controlled and gradual movement.

HOW IT WORKS

Sustained stretch resets the muscle spindles, which gradually becomes accustomed to its
new length, it reduces its signaling, therefore decreasing the stretch reflex, which leads to
muscle relaxation, and lengthening.
Stretching muscle that requires lengthening and relaxing has been shown to be an
effective way to decrease the pain cycle.
However stretching a muscle that does not show an unusual pattern, has not been
shown to be as beneficial.
We have identified unusual muscle patterns in the test we have had you do, where your
muscles remain contracted, rather than relaxing. Therefore a stretch for a specific
muscle in your back should be effective in returning your muscle patterns to normal.

The normal pattern in forward bending is that your low back muscles relax when you bend
forward as far as you can. This is because the spine is supported by the passive elements,
like the boney structure and ligaments.

In some low back pain, the back muscles don't relax, and the muscles surrounding the
spine remain contracted, acting as a spinal 'spine', stopping the individual from reaching a
relaxed, full range of motion.
This is commonly thought to be a pain avoiding mechanism, where the muscles contract, to avoid being put in a position of
expected pain (whether the movement would be painful or not).
This is shown in the basic cycle above.

STRETCHING

The aim of this treatment is to interrupt this cycle, by replacing the muscle over-reaction,
with a more adequate muscular response.
When a muscle is stretched, so is the muscle spindle (which, from within the muscle,
regulates the tension in the muscle).
The muscle spindle is involved in the stretch reflex, which attempts to resist the change in
muscle length, by causing the stretched muscle to contract, especially if stretched quickly. This is
to protect the spine from moving too fast past the limits of its movement. It is thought that with
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deckhand, supporting the spinal column and allowing controlled and gradual movement.
APPENDIX H: WEEKLY OUTCOME DATA
PARTICIPANT 1

Figure A1: Weekly scores in Outcome Measures for Participant 1
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.

PARTICIPANT 2

Figure A2: Weekly scores in Outcome Measures for Participant 2
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.
PARTICIPANT 3

Figure A 3: Weekly scores in Outcome Measures for Participant 3
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.

PARTICIPANT 4

Figure A 4: Weekly scores in Outcome Measures for Participant 4
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.
PARTICIPANT 5

Figure A 5: Weekly scores in Outcome Measures for Participant 5
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.

PARTICIPANT 6

Figure A 6: Weekly scores in Outcome Measures for Participant 6
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.
PARTICIPANT 7

Figure A 7: Weekly scores in Outcome Measures for Participant 7
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.

PARTICIPANT 8

Figure A 8: Weekly scores in Outcome Measures for Participant 8
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.
PARTICIPANT 9

Figure A9: Weekly scores in Outcome Measures for Participant 9
NPRS = Pain Intensity. Includes line of best fit (dotted line) over the intervention period.
APPENDIX I: ADDITIONAL DEMOGRAPHIC INFORMATION
Data summarized from initial case history.
APPENDIX J: JOURNAL OF BODYWORK AND MOVEMENT THERAPIES: INSTRUCTIONS FOR AUTHORS
Journal of Bodywork and Movement Therapies

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