MUSCLE ACTIVITY AND LUMBAR PA STIFFNESS

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DEDICATION

This work is dedicated to my son, David Shirley, whose enthusiasm for science and ability to always see another perspective provided endless inspiration throughout this work.

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ABSTRACT

This thesis examines the contribution of muscle activity to lumbar postero-anterior (PA) stiffness in people with and without symptoms. Lumbar PA stiffness was measured with a mechanical device purpose built to simulate the manual assessment of PA stiffness in the clinic. Muscle activity was measured using surface electromyography. The contribution of muscle activity to PA stiffness was examined under a number of active and passive conditions.

The lumbar spine PA response demonstrates time dependent behaviour within 5 loading cycles with PA forces where the first cycle is significantly less stiff than the following four cycles. This behaviour should be considered a normal response to loading with PA forces. The effects due to the time dependent behaviour reverse within a short period of time and PA stiffness is stable when tested over time intervals greater than 5 minutes and up to several months.

Voluntary activation of the lumbar extensor muscles significantly increases lumbar PA stiffness even when activation is as low as 10% of a maximal voluntary contraction. This suggests that low levels of voluntary activity increase stiffness and led to the proposal that activity occurring in response to assessment with PA forces might also increase stiffness.

The intensity of low back pain during an episode was significantly predicted by the level of PA stiffness at 0.05 Hz loading and by muscle activity. Furthermore, there was a strong trend for measures of stiffness and muscle activity taken during an episode of LBP to predict the presence of pain several months later. These results indicate that during an episode of LBP high pain levels are associated with high stiffness but low levels of initial

stiffness and muscle activity are associated with ongoing pain. Theses results have implications for the treatment of acute low back pain.

This thesis also describes a new method of calculating instantaneous stiffness, which demonstrates excellent reliability. This new method is highly correlated with the method of calculating stiffness between 30 and 90N that has previously been used in studies investigating PA stiffness. The new method is particularly useful in situations where there is variable maximum force within and between different stiffness tests eg during tasks involving dynamic breathing.

Lumbar PA stiffness varies throughout the breathing cycle. During breathing tasks involving breath holding there was no increase in muscle activity for volumes up to tidal volume but stiffness increased with increasing respiratory and expiratory efforts. There is no increase in stiffness with breath holding at different inspiratory volumes when the glottis is closed but stiffness increases with increased inspiratory effort when the glottis is held open. Trunk and respiratory muscle activity as well as intra-abdominal and trans-diaphragmatic pressures are correlated with increased stiffness. Lumbar PA stiffness is greater at L2 then L4 during respiratory manoeuvres. These results provide evidence that the diaphragm and trunk muscles contribute to PA stiffness.

PREFACE

This thesis examines the relationship between lumbar postero-anterior (PA) stiffness and muscle activity. The first chapter reviews the literature and provides the rationale for the experimental studies that follow. Chapters Two to Six are written as independent studies. At the time of submission of this thesis, Chapter Two has been published in Manual Therapy and Chapter Three has been published in Physical Therapy. Chapter Five is being prepared for submission to Manual Therapy and Chapter Six as been submitted to Journal of Applied Physiology. These chapters have been presented in a similar format to that used in their respective papers. The experimental studies were approved by the Human Ethics Committees of the institutions involved and all subjects gave written consent to participate.

Chapter Two was undertaken to determine the normal responses to application of postero-anterior (PA) forces to the spine and to determine the behaviour of the PA response over time. Understanding the normal response of PA stiffness was necessary so that the results of the later studies could be interpreted appropriately.

Chapter Three examined the effect of voluntary activation of the lumbar erector spinae on PA stiffness. Of particular interest was the result that activation of erector spinae as low as 10% of maximum voluntary contraction increased stiffness. However, it was not known whether this increase in stiffness was clinically relevant.

After determining that small amounts of voluntary muscle activity increase stiffness, Chapter Four investigated the relationship between stiffness and muscle activity in the presence of low back pain. Muscle activity was not found to the related to PA stiffness in subjects with LBP. The absence of a relationship could be because the very small

amount of spontaneous muscle activity provoked by PA forces was not great enough to produce changes in stiffness, or other muscles that were not measured may contribute to PA stiffness.

Chapters Five and Six examine the effect respiratory manoeuvres on PA stiffness. Chapter Five investigated the effect of breath holding at various inspiratory volumes and Chapter Six extended the findings by examining inspiratory and expiratory manoeuvres. In Chapter Five there was no difference in PA stiffness at different volumes of breath holding. Stiffness was increased during dynamic manoeuvres where there was higher respiratory muscle activity suggesting a possible role of the diaphragm in PA stiffness. Chapter Six was undertaken to explain the results of Chapter Five by determining the effect of breath holding with the glottis open and closed as well as comparing stiffness at L2 and L4.

Chapter Seven discusses the implications of the main findings of this thesis and makes suggestions for future directions for research in this area.

PUBLICATIONS

The work arising from these PhD studies has resulted in the following publications.

Refereed Journals

SHIRLEY D, LEE M, AND ELLIS E. (1999) Relationship between submaximal activity of the lumbar extensor muscles and lumbar postero-anterior stiffness. *Physical Therapy*. **79**(3): 278-285.

SHIRLEY D, LEE M AND ELLIS E (2002) The behaviour of postero-anterior lumbar stiffness with repeated loading. *Manual Therapy*. **7**(1): (In press)

SHIRLEY D, HODGES PW, ERIKSSON AEM AND GANDEVIA S C. (2002) Spinal stiffness changes throughout the respiratory cycle. Submitted to *Journal of Applied Physiology*.

Published Abstracts

SHIRLEY D, LEE M, (1992) An investigation of the relationship between lumbar spine stiffness and low back pain. *Proceedings of the International Federation of Orthopaedic and Manipulative Therapy 5th International Congress*. June 1-5, 1992. Vail, Colorado, USA.

SHIRLEY D, LEE M, MORAN C, (1993) An investigation of the relationship between lumbar EMG activity and lumbar stiffness. *Proceedings of the 8th Biennial Conference of the Manipulative Physiotherapists Association of Australia*. Perth, Western Australia.

SHIRLEY D, LEE M, ELLIS E. (1995) Muscle activity as a possible contributing factor to Lumbar PA stiffness. *Proceedings of the 9th MPAA Biennial Conference, Clinical Solutions*. November 22-25, Gold Coast, Queensland, Australia.

SHIRLEY D, LEE M, MORAN C. (1995) The electromyographic response of lumbar extensor muscles to an applied PA force. *Proceedings of the 12th International Congress Of the World Confederation for Physical Therapy*. June 25-30, 1995. Washington DC, USA.

SHIRLEY D, LEE M, ELLIS E. (1997) Clinical implications of the behaviour of properties of lumbar spine PA stiffness with repeated loading? *Proceedings of the Manipulative Physiotherapists Association of Australia 10th Biennial Conference, Focusing Ahead.* November 26-29. Melbourne, Australia.

SHIRLEY D, LEE M, ELLIS E. (1999) The response of the lumbar spine to repeated postero-anterior loading. *Proceedings of the 13th International Congress Of the World Confederation for Physical Therapy*. May 23-28, 1999. Yokohama, Japan.

SHIRLEY D, ERIKSSON AEM AND HODGES PW (2000) Spinal stiffness changes throughout the respiratory cycle. *Proceedings of the 7th Scientific Conference of the International Federation of Orthopaedic Manipulative Therapists*. November 6-10. Perth, Western Australia.

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SMILE ! YOUR HAVING A GREAT DAY

CHAPTER ONE

LITERATURE REVIEW

INTRODUCTION

Manually applied postero-anterior (PA) forces are commonly used in the assessment and treatment of acute low back pain (LBP). Low back pain is a significant health problem and the associated disability and costs are rapidly increasing (Waddell 1996). Clinical guidelines advocate manual therapy as appropriate for use in acute LBP (Bigos et al. 1994; CSAG 1994; Waddell et al. 1996). The use of manual therapy in treatment relies on judgements made by the clinician during the application of PA forces in assessment. At the present time it is not possible to optimise the use of PA forces as the mechanisms of effectiveness are not well understood and the subgroups that manual therapy may be most effective for have not been identified.

Postero-anterior (PA) stiffness is the term used by physiotherapists to describe the resistance to movement felt when a force is applied in the PA direction to the spinous process of a vertebra in the lumbar spine. Postero-anterior forces are commonly used in the assessment of patients with low back pain by practitioners of manual therapy (Grieve 1984; Maitland et al. 2001). When a PA force is applied the therapist obtains information about the reproduction of the patient's symptoms, any increase in PA stiffness and whether any muscle spasm is present (Maitland et al. 2001). This information is then used to determine the most appropriate technique to use for treatment. A common method of choosing treatment is to use the passive accessory technique that most closely reproduces the patient's symptoms during assessment. It is believed that applying the technique, as a treatment, will result in a decrease in pain, PA stiffness and muscle activity, although this belief has not been fully investigated.

LOW BACK PAIN

An old problem or a new epidemic

Low back pain (LBP) is a major cause of suffering, disability and social costs (Carey et al. 1996) in much of the world today. Back pain has been a problem throughout history (Allan and Waddell 1989) and is described as universal, irrespective of culture, race or gender (Waddell 1996). It is estimated that 80% of the population will experience LBP at some time during their life (CSAG 1994; Koes et al. 1995), some of these will go on to have recurrent episodes (Waddell 1996) and about 10% will be chronically incapacitated due to their pain (Lattimer 1997).

Most episodes of LBP (80%) are self limiting and resolve within 4-6 weeks (Coste et al. 1994) with minimal intervention (Little et al. 1996; Lattimer 1997). The natural history of LBP suggests that for most patients their symptoms will improve and they will return to work either with or without pain, however, for many there will be a recurrence of their symptoms (Von Korff 1994; MacDonald et al. 1997; Schiottz-Christensen et al. 1999; Waxman et al. 2000). Recurrence rates are high with reports of 31% of people suffering LBP in Sweden having one or more recurrences of pain within twelve months (Bergquest-Ullman and Larson 1977) and in North America up to 45% experience a further episode within five years (Spengler et al. 1986; Zwerling et al. 1993; Garcy et al. 1996). In addition, recurrent episodes may last longer and become more disabling (Waxman et al. 2000).

Low back pain is a relatively common condition and estimates suggest a prevalence of LBP of 12-33% at any one time (CSAG 1994; Walker 2000), with similar figures reported in Australia, the United Kingdom, the United States, Canada and Europe (Von Korff et al. 1988; Carey et al. 1996; Cassidy et al. 1998; Walker 2000). Low back pain is a significant problem in Australia with a point prevalence of between 15 and 17% (Strauss et al. 1993; Ebrall 1994). There are concerns that the problem of LBP is increasing in the community. However, while LBP has always been a health problem, the prevalence of LBP does not appear to be increasing (Leino et al. 1994). Although the prevalence is not increasing, the marked increase in disability (Waddell 1996) and the associated escalation of costs with LBP, is of major concern. As the prevalence of LBP is not increasing it is most likely that the increasing costs are related to the disability associated with the LBP condition rather than the prevalence of the condition.

Costs

The costs of LBP are high in all western countries with time lost from work and disability forming the majority of the costs involved (Spengler et al. 1986). In NSW alone in 1995/96 there was a loss of 117,255 weeks of work. Eighteen percent of the total cost of back injuries was made up of costs related to the time off work for 61% of back injuries (Lattimer 1997). The average cost per claim was $16,195 with half of the claims costing $2,901 or less. The total cost of back injuries for 1995/96 in NSW was $212.5 million, almost double the cost for 1992/93 (Lattimer 1997). Back injuries also accounted for $700 million in health system costs in 1993-94 in

Australia, which is equivalent to 23% of total musculoskeletal costs (Mathers and Penm 1999). Statistics from other countries indicate that there are also significant high costs for LBP throughout the western world (Nachemson 1992; CSAG 1994).

One of the significant costs associated with LBP is treatment by health professionals. Most people who experience low back pain consult a health care provider at some time about their problem (Carey et al. 1996). In Australia, treatment by allied health professionals cost $151 million in 1993-94 (Mathers and Penm 1999). Appropriate management of LBP from the early stages may be one of the factors that will lead to a decrease in disability associated with LBP and therefore a reduction in the overall costs.

Evidence based management of low back pain

The escalation in costs associated with LBP has led to many randomised controlled trials (RCT) and systematic reviews of the literature to determine the most effective treatments. In the past, management has focused on diagnosis by identifying the structure at fault, which would then dictate treatment options. However, identifying the source of LBP is extremely difficult and, in fact, in 80% of patients no specific source of pain can be identified (Spitzer 1987). Therefore, most cases of LBP can be classified as simple LBP, or in other words, non specific LBP of musculoskeletal origin (Waddell 1996) and patients are managed conservatively.

Current evidence suggests management of simple LBP includes early return to normal activity, which leads to less chronic disability and time off work, education about the benign nature of LBP and spinal manipulative therapy (Waddell 1996). Spinal manipulative therapy (SMT) has been demonstrated to be more effective than placebo for acute low back pain and there is also moderate evidence for the use of SMT to treat chronic low back pain (van Tulder et al. 1997). Clinical guidelines for the management for acute LBP recommend that SMT is of benefit for low back pain of less than six weeks duration (Bigos et al. 1994; CSAG 1994; Waddell et al. 1996).

SPINAL MANIPULATIVE THERAPY

Historical Perspective

Spinal manipulative therapy has featured as a remedy for back problems throughout recorded history. Spinal manipulative therapy involves techniques applied manually by the therapist and includes both mobilisation and manipulation (Maitland et al. 2001). Manipulative techniques and various forms of traction are reported from the time of Hippocrates (Schiotz and Cyriax 1975) and are evident in most ancient cultures. These manual techniques appear to have been used and replicated with little evolution for many centuries. Eventually, towards the end of the nineteenth century three main streams of SMT emerged; conventional medicine, chiropractic and osteopathy (Schiotz and Cyriax 1975).

Spinal manipulative therapy as practiced by physiotherapists emerged from conventional medicine and involves key features, which can be attributed to the orthopaedic surgeons, Mennell and Cyriax. Mennell (1960) describes the importance of history taking in the diagnosis of LBP by stating that "there are few conditions in which the history plays a more important role than in LBP" (p 39). A comprehensive history allows the clinician to determine if the LBP is of musculoskeletal origin and to ensure that there are no contraindications to manual therapy (Maitland 1986). Mennell (1960) also placed emphasis on passive movements of the joints called joint play movements, which are like Maitland's passive accessory movements (Maitland 1986). In Australia, Maitland (1986) was a pioneer of manipulative physiotherapy and was one of the first practitioners to place such a strong emphasis on a systemic examination to guide treatment by incorporating the features of history taking and examination with passive movements. Mobilisation and manipulation are key techniques using passive movements that are advocated by Maitland (1986) for the assessment and treatment of musculoskeletal pain.

Mobilisation and Manipulation

Passive mobilisation is the application of passive movements to the spine in a rhythmical oscillating fashion. These movements are under the voluntary control of the patient and can be prevented by the patient (Maitland et al. 2001). Manipulation is a high velocity, low amplitude thrust performed with a minimum of force applied at or near the end of available range of movement. Manipulation is not under the

voluntary control of the patient (Maitland et al. 2001). Mobilisations are described by grades of movement (ie Grade I – IV) depending on where they are performed in range. A manipulation could be described as a larger grade of mobilisation (eg grade V) (Maitland et al. 2001).

The manual application of forces to the spine is part of the Maitland approach, which relies on systematic assessment and monitoring of signs and symptoms to guide treatment and assess progress (Maitland 1986). The theory of a "permeable brick wall" was proposed and suggested that the therapist focuses on the presenting signs and symptoms to choose management strategies and monitor ongoing treatment while considering the possible influence of pathology. This concept is not unlike the diagnostic triage proposed by Waddell et al. (1996) whereby most patients would be classified as having simple LBP, however it is emphasised that serious pathology must be recognised. It is important to note that neither of these approaches focuses on structural diagnosis but rather aims to identify those patients with simple LBP.

In order to make clinical decisions the therapist aims to provoke the patient's symptoms and then uses the provocative techniques as a means of reducing symptoms. The provocative techniques include various active and passive movement tests. Passive movement tests are described as physiological (those which can be voluntarily performed by the patient) and accessory (those which cannot be performed voluntarily) (Maitland et al. 2001).

One passive accessory technique commonly used for the provocation of symptoms is the PA pressure (Maitland et al. 2001). This technique involves the therapist

applying an oscillating force in a postero-anterior (PA) direction to the spinous or transverse processes of the lumbar spine. When applying this PA force the therapist is seeking information about increased spinal stiffness, the pain response and the presence of muscle spasm. Practitioners of manipulative therapy have operated on the assumption that there is a relationship between increased spinal stiffness and muscle spasm in patients with LBP, however, this assumption has not been substantiated.

Mechanisms

The mechanisms by which manipulative techniques are effective are poorly understood. The proposed mechanisms include mechanical effects and neurophysiological effects (Zusman 1986). Mechanical effects could involve a permanent or temporary change in length of connective tissue structures, such as the joint capsule of the zygapophyseal joints, ligaments and muscle. It seems unlikely that any observed changes in mobility associated with mobilisation are due to permanent changes in the length of connective tissue. To achieve permanent changes in a single treatment session the force used would have to be great enough to cause microfailure of the tissue (Threlkeld 1992). Threlkeld (1992) suggests that the forces used in manual therapy techniques are not great enough to result in microfailure of tissue and are more likely to cause temporary length changes due to creep which is reversible over time. Therefore, mechanical effects on the passive tissues do not adequately explain the dramatic changes in mobility that are said to be observed in the clinic and maintained between treatments.

Neurophysiological mechanisms have also been postulated to account for changes in PA mobility observed in response to the application of PA forces. One of these neurophysiological mechanisms may be modulation of the afferent input such that the perception of pain is diminished (Zusman 1986). Muscle activity is thought to occur in response to pain (Mennell 1960; Travell and Simons 1983) and increased muscle excitability in response to joint pathology or pain provocation has been demonstrated in animal models (Ferrell et al. 1988; Qing-Ping and Woolff 1995). If increased muscle activity occurs in response to pain then it would be expected that muscle activity might reduce if the level of pain reduces (Katavich 1998) and PA stiffness may be decreased.

LUMBAR POSTERO-ANTERIOR (PA) STIFFNESS

Response of the vertebral column to applied PA forces

Early texts of manual therapy suggest that when a PA force is applied to the spine the resulting movement is primarily between two adjacent vertebrae. Accordingly, these techniques were called passive intervertebral accessory movements (Maitland 1986). In contrast to this belief, more recent investigations of the application of PA forces to the lumbar spine have indicated that a complex movement of the spine occurs (Lee et al. 1996). The predominant movement involves the lumbar spine moving anteriorly into extension, with movement also occurring many levels distant from the applied force, and only a small amount of translation of one vertebra relative to another (Lee and Moseley 1991; Lee et al. 1995; Lee and Evans 1997). In

addition, when a PA force is applied to the spine the pelvis rotates anteriorly (Lee et al. 1994b) and the ribcage is compressed (Lee et al. 1994a) and there is also likely to be compression of the overlying soft tissues. This information indicates that the early hypothesis that only intervertebral movement occurs when PA pressures are applied to the lumbar spine is incorrect. In fact, PA forces applied to the lumbar spine result in a complex motion involving extension of the spine, anterior rotation of the pelvis and only a small amount of intervertebral movement.

The response of lumbar spine tissues to loading with external forces has been described in both living subjects and cadaveric specimens as consisting of a non linear phase early in range followed by a linear phase later in range (Yahia et al. 1991; Lee and Evans 1992). The response of the tissues of the lumbar spine to applied PA forces has been described as non linear at low forces (<30N). The stiffness response is linear from 30 - 100N (Lee and Svensson 1990) although there is a tendency for stiffness to increase with increasing force up to 200N (Latimer et al. 1998). The response to PA forces is typically depicted by a force displacement relationship (Figure 1.1) and is generally thought to reflect the normal mechanical behaviour or compliance of the soft tissues of the spine (Threlkeld 1992).

The contribution of individual soft tissue structures to lumbar PA stiffness has been determined by sequential dissection of lumbar intervertebral segments of cadaveric specimens in which the lumbar spine was dissected away from the surrounding tissue (Lee 1995). The intervertebral disc makes the largest contribution to restriction of motion in a PA direction while the supraspinous ligaments, interspinous ligaments, ligamentum flavum and the zygapophyseal joints only play a

small part in resistance of motion in a PA direction (Lee 1995). Although the intervertebral disc was found to be the main structure resisting PA movement in sequential dissection, there is no indication of the magnitude of contribution of the IVD disc in relation to other external factors, which could influence stiffness such as muscle activity. In the clinical situation, however, skin and subcutaneous tissue, in addition to the structures mentioned previously, could have some influence on PA stiffness detected by manual examination.

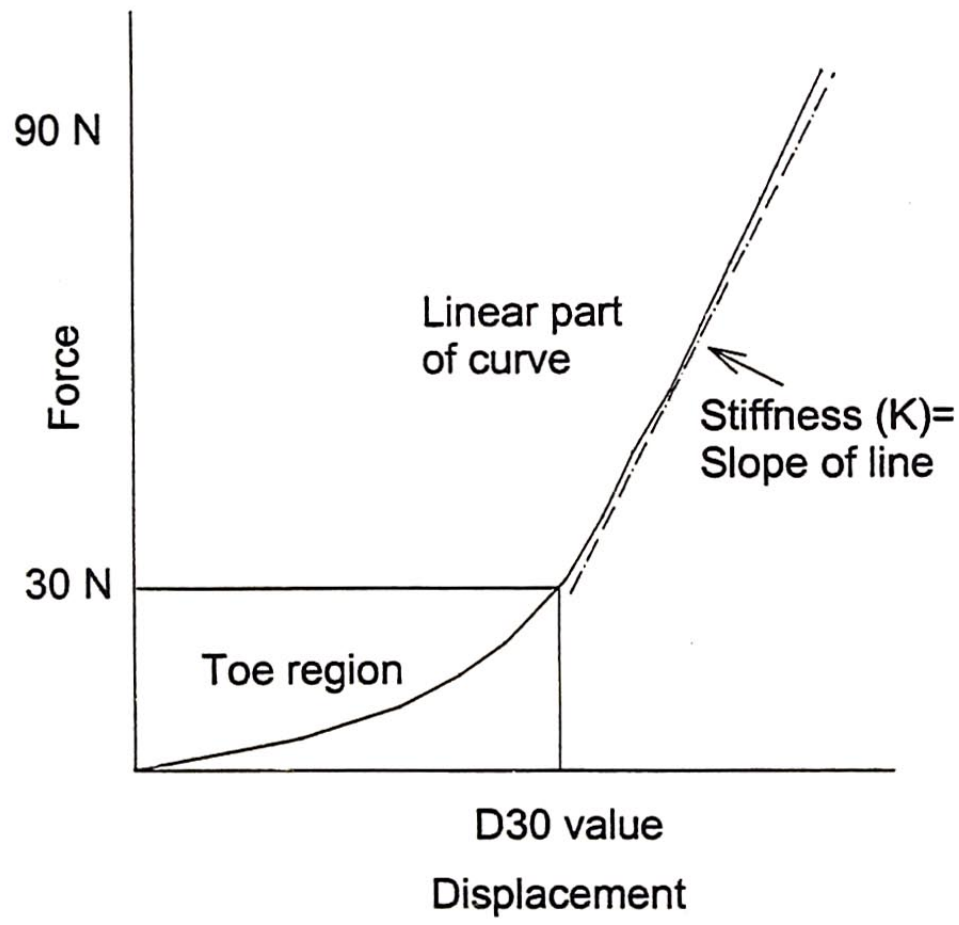

Figure 1.1: Schematic Diagram of part of a Force Displacement Curve (loading to 90N) showing Stiffness Coefficient K and D30. Stiffness Coefficient K is the slope of a regression line fitted to the curve between 30 and 90N. Displacement D30 is the displacement between 2 and 30N of force.

Manual Assessment

The assessment of the response of the spine to applied PA forces is generally carried out manually in the clinic, however, mechanical devices have also been devised to produce an objective measure of the spine's response. Clinicians assess the spine manually with PA forces to obtain an impression of the status of the soft tissues and compliance (stiffness) of the spine. The information about compliance of the spine is based on a subjective judgement of the spine's response when PA forces are applied.

Reliability of stiffness judgements

Manual assessment has been shown to be unreliable in determining whether there is increased stiffness (Matyas and Bach 1985; Maher and Adams 1994). An early study determined that students are unreliable in judging stiffness but that they are reliable in determining the pain response (Matyas and Bach 1985). This work has been criticised because the subjects making stiffness judgements were physiotherapy students who were inexperienced in the use of these techniques. Further studies using experienced manipulative physiotherapists have found similar results indicating that experience does not improve reliability of stiffness judgements. Experienced manipulative physiotherapists demonstrated good reliability for making pain judgements but were unreliable with stiffness judgements on patients with low back pain being tested with the range of forces used by physiotherapists in clinical practice (Maher and Adams 1994, Binkley et al 1995). The value of identifying painful segments is highlighted by the ability of some therapists to accurately detect

symptomatic levels of the cervical or lumbar spine compared to spinal level diagnosis with segmental nerve blocks when manual examination is accompanied by the patient's verbal response (Jull et al. 1988; Phillips and Twomey 1996). In contrast, manipulative physiotherapists demonstrate excellent agreement when identifying symptomatic segments of the cervical spine without feedback of the pain response from the patient suggesting that pain may not be the only important factor (Jull et al. 1994). However, in some cases symptomatic segments were reliably identified in asymptomatic individuals, perhaps indicates that verbal responses from the patient are important.

The ability to make judgements about spinal PA stiffness is a complex task that is influenced by many variables. Standardising some of the variables that affect the judgement may result in better reliability of this process. These variables can be described in the following categories: the perception of stiffness; variations in technique; and patient variations.

Perception of PA stiffness

Judgements about PA stiffness of the spine are made from a subjective interpretation of the information perceived when applying PA forces to the lumbar spine. However, it is not known exactly which information the therapist focuses on when making judgements of PA stiffness. Stiffness judgements may involve the therapist focusing on either abnormal tissue stiffness, which could be due to muscle reactivity, qualities of the response through range (Jull et al. 1994) or other patient or therapist related factors.

Perception of stiffness could relate to the responses of the spine early in range or the stiffness characteristics later in range. Patients with LBP have decreased displacement of the spine early in range as well as increased stiffness later in range during an episode of LBP (Latimer et al. 1996c), which suggests that the responses of the spine throughout the entire range of force application are worthy of further consideration. Although the importance of the nonlinear phase is still not fully understood, the addition of a nonlinear toe region did not alter the ability to discriminate stiffness (Nicholson et al. 1998) compared to linear elastic stiffness alone (Nicholson et al. 1997; Nicholson et al. 1998) when using non-biological elastic stimuli. It is possible that different therapists focus on different aspects of the response when they apply a PA force to make their stiffness judgement. Variations in the focus of attention of the spine's response to PA loading could lead to different judgements of stiffness and therefore contribute to the poor inter-rater reliability.

Variations in technique

Variation in therapist technique during manual application of a PA force can also influence perception and reliability of spinal PA stiffness. Variations in therapist technique include the direction of force application, the vertebral level tested, the contact grip used, the frequency of force application, and the magnitude of the applied force and vision.

Direction of force

Physiotherapists report that they apply PA forces in such a way that they take into account the individual spinal curves of patients which means varying the direction for different vertebral levels. The mean angle used by physiotherapists to apply a PA pressure at different vertebral levels is -13.8° at L5, -4.8° at L4, -1.2° at L3, 3.1° at L2 and 6.4° at L1 (Viner and Lee 1995). The negative angles indicate PA pressures directed towards the subject's feet (i.e. in the caudal direction) and the positive angles indicate pressures were applied directed towards the subject's head (i.e. in the cephalad direction). The angles determined by Viner and Lee (1995) are similar to the angles of the vertebral bodies in standing subjects determined by Stagnara et al. (1982), which have been used in the measurement of lumbar PA stiffness with mechanical devices.

Varying the angle of the force to a more caudad or cephalad direction to fully explore the spine's response is recommended practice in assessment of LBP (Maitland et al. 2001). Spinal stiffness varies according to the angle of application of the PA force when measured with a mechanical device (Caling and Lee 2001). Spinal stiffness is greater at the angles determined by Viner and Lee (1995) than when applied either 10 degrees more caudad or cephalad (Caling and Lee 2001). Also stiffness is less when the force is applied in the vertical direction compared with perpendicular to the spine at L5 (Allison et al. 1998). However, the differences were small (approximately 9%) and may not be clinically significant. Therefore, as the angle of application of the PA force affects the stiffness response it will influence the judgement of stiffness made by the therapist.

Vertebral level tested

To make a judgement of PA stiffness the therapist decides whether the movement is increased or decreased compared to their perception of "normal" and compared to adjacent vertebral levels (Maitland et al. 2001). Decreased movement in a PA direction is generally considered to reflect increased PA stiffness. Lumbar PA stiffness increases between L1 and S1 when the force is applied to individual vertebral levels (Lee and Liversidge 1994; Viner et al. 1997). Therefore, the validity of comparing stiffness between adjacent vertebral levels is questionable, as stiffness varies among vertebral levels. In addition, slight alterations in direction of force application will alter PA stiffness.

Contact Grip

Two techniques are commonly used for applying PA forces to the spine. The most common method is for the therapist to place the pisiform area of the hand over the spinous process with the spinous process resting just distal to the pisiform bone (Maitland et al. 2001). The second method involves placing the tips of the thumbs on the skin over the spinous process and applying the PA pressure (Maitland et al. 2001). In the lumbar spine region most therapists use the pisiform grip, as it is more comfortable for both the therapist and patient. Although the ability to discriminate stiffness does not vary with the technique is used (Maher and Adams 1996a), it has been shown that the stiffness judgement differs depending on which method is used. Stiffness judgements made using the thumbs technique are on average greater than from the pisiform grip (Maher and Adams 1996a).

When stiffness judgements are made using the thumbs the therapist is relying on information from a smaller contact area than with the pisiform grip. When PA stiffness was measured with a mechanical device comparing contact areas similar to that of a pisiform and thumb tip grip it was increased when a PA force was applied using a smaller contact area (Squires et al. 2001). Therefore, the results of the increase in perceived stiffness when the thumb tip grip was used (Maher and Adams 1996a) may be related to an interaction between the type of grip used and the response of the spine to the size of the contact area. It is important for therapists to be aware that their contact grip will influence their judgement of stiffness and therefore, it is important to use a consistent technique.

Frequency of force application

The frequency of force application also influences lumbar PA stiffness. Several studies have demonstrated higher PA stiffness with faster frequency of force application when measured with mechanical devices (Lee and Svensson 1993; Squires et al. 2001). These findings suggest that if the frequency of force application is varied during clinical assessment it may affect the perceived stiffness response and highlights the importance of standardising frequency for making judgements about change in stiffness.

Magnitude of force

The magnitude of the applied force influences lumbar PA stiffness, with larger forces resulting in greater stiffness values (Latimer et al. 1998). Latimer et al.

(1998) applied 200N forces to asymptomatic subjects and analysed the stiffness in various ranges within that force. While the force displacement curves were predominantly linear between 30 and 200N, the findings indicated that the stiffness values were greater in the higher ranges of force. These results suggest that the spine became stiffer with increases in the applied force. Previous studies of PA stiffness that only measured stiffness up to 90N of force have described a linear response between 30 and 90N of force (Latimer et al. 1996a). Therefore, therapists who apply larger forces during stiffness assessment are likely to report higher stiffness values.

Vision

Another factor that effects the perception of stiffness is whether the therapist looks at the spine during assessment of PA stiffness. Although occluding vision does not impair the therapist's ability to make stiffness judgements, when vision is occluded a bias is introduced such that stiffness stimuli are judged to be stiffer than when vision is not occluded (Maher and Adams 1996b). This implies that in order to improve the perception of stiffness, therapists must standardise whether they have visual contact with the spine.

Breathing cycle

Another factor that may influence reliability of stiffness judgements is the effect of the breathing cycle. In the clinical situation spinal stiffness is usually measured with the patient breathing in the relaxed state with no specific instructions about breathing

(normal tidal breathing). In contrast, most studies measuring spinal stiffness with a mechanical device have measured spinal stiffness with the breath held at functional residual capacity (FRC). Spinal stiffness is significantly decreased at higher lung volumes (total lung capacity) (Keaveney et al. 1989) and the lumbar spine is stiffer during tidal breathing than breath holding at FRC (Beaumont et al. 1991). Therefore, the breathing cycle can have an effect on stiffness indicating that instructions about breathing should be kept consistent.

During the breathing cycle there are changes in muscle activity (eg diaphragm and transversus abdominis) and intra-abdominal pressure and these factors may be related to the changes in PA stiffness associated with breathing. The diaphragm is involved in postural stability of the spine during sudden movements of the limbs (Hodges et al. 1997b) and can perform its respiratory and spinal stability functions simultaneously (Hodges and Gandevia 2000a). If the diaphragm has a role in stabilising the spine during active movements of the body it is also possible that it could be activated by unexpected passive perturbations such as an externally applied PA forces. The influence of the diaphragm and intra-abdominal pressure on PA stiffness has not yet been determined.

Patient variations and other factors

Body mass index (BMI) has been shown to influence lumbar stiffness in subjects without LBP (Viner et al. 1997). In subjects without LBP, those with a higher BMI were less stiff than those with a lower BMI (Viner et al. 1997) suggesting that greater proportion of body fat may contribute to the decreased stiffness measured

with a mechanical device. Body fat may be more likely to have an impact on judgements of stiffness response early in range in subjects with a higher proportion of fat as there would be more padding between the therapist's contact and the spinous process of the subject. However, the relationship may be more complex than this as a greater BMI would result in a greater percentage of fat overall. It may be important to note BMI of subjects for research purposes to account for any unexpected variations in measurements.

Other factors that influence PA stiffness include the type of surface the patient is lying on and the position of the patient. Stiffness of patients lying on a padded plinth is less than with the patient on an unpadded surface (Maher et al. 1999), implying that it is important to examine the patient on the same plinth to be able to compare measures. Also, patients are stiffer when they are tested in positions of extension or flexion compared with normal prone lying on the plinth (Edmondston et al. 1998) therefore, it is important to standardise the testing position.

In summary, many factors change the PA stiffness of patients measured with a mechanical device and various factors also have an impact on the perception of stiffness. Therefore, it is important to standardise as much of the measurement procedure as possible so that reliability in the clinic is improved for comparisons between different therapists and for making judgements about improvement. Similarly, standardisation of procedure for research purposes will improve reliability and confidence in interpretation of results.

Mechanical Assessment of PA Stiffness

Mechanical devices have been developed to quantify PA stiffness. The earliest of these devices was used by the chiropractors and was called a tissue compliance meter (TCM) (Fischer 1987). The TCM was used to measure the compliance of soft tissues adjacent to the spine, particularly the muscles (Fischer 1987). Reliability of this device was extremely poor (Intra-class correlation coefficient (ICC)=.005) (Kawchuk and Herzog 1995). There has been some criticism about whether the use of ICC was appropriate for this method (Haas 1996), however the investigators of the study refute this suggestion arguing that ICCs were appropriate. It is perhaps not surprising that reliability was poor as this was a hand held device that was pressed into the tissue and required the examiner to make a judgement of stiffness. A similar instrument that was mechanically operated was developed and was shown to have good accuracy (<0.5% error for expected differences of approximately 2mm) (Kawchuk and Herzog 1996). Whilst the mechanical device for measurement of tissue compliance was shown to be accurate and reliable, all the testing was performed on five foam surfaces so its accuracy on human subjects has not been determined. This device does not appear to have been used to quantify PA stiffness of the spine.

Recently, mechanical devices for measuring PA stiffness of the spine have been developed both in Australia and overseas (Lee and Svensson 1990; Lee and Evans 1992; Latimer et al. 1996b). The first of these devices, called the Spinal Physiotherapy Simulator (SPS), was developed by Lee and Svennson (1990). The

SPS was large and housed in a confined space and this resulted in difficulties testing symptomatic populations. The SPS is highly reliable (ICC= 0.88) for testing PA stiffness on human subjects and has satisfactory accuracy for measuring stiffness (within 1% of true value) of an elastic beam (Lee and Svensson 1990). Many experiments have been performed using the SPS, however, it is best suited to testing asymptomatic subjects as its location and height make it difficult for symptomatic subjects to access for measurement. The need to study the responses of PA pressures on a population with low back pain led to the development of a smaller, portable device that is suitable for use in the physiotherapy clinic (Latimer et al. 1996b). The portable device also has good reliability (ICC=0.96) for measurement of stiffness in a patient population and a high degree of accuracy (maximum error of 2.5%) for testing an elastic beam (Latimer et al. 1996a).

The portable mechanical device developed by Latimer et al. (1996b) consists of a probe, which makes contact with the skin over the spinous process in a similar way to the physiotherapist's hand when manually applying a PA pressure. The stiffness testing device further attempts to simulate force application by a clinician and applies the PA force from a stationary start like the process of manual assessment of PA stiffness in the clinic. The motor that drives the probe up and down in a rhythmical oscillating manner, simulating a physiotherapist performing a mobilisation technique, is housed above the probe in a box. The box also contains a strain gauge that measures the force applied by the probe, and a linear potentiometer that measures the resulting displacement of the skin in a PA direction (Latimer et al. 1996b). Mechanical assessment of PA stiffness is performed by applying a PA force to the spinous process of a lumbar vertebra. The device measures the force applied

and the resulting displacement of the skin surface over the spinous process. Postero-anterior stiffness is usually calculated as the gradient of the force displacement curve between ranges of force between 20 and 100N (Lee and Svensson 1990) or between 30 and 90N (Latimer et al. 1996a).

Other similar devices have been used and developed by researchers in Australia and overseas (Lee and Evans 1992; Edmondston et al. 1998). One device (Edmondston et al. 1998) can only apply one loading cycle at a time and so does not have the scope to study the effect of rhythmical oscillating forces as they are applied in the clinic. The other device (Lee and Evans 1992) measured stiffness with the aid of two linear potentiometers positioned either side of a central force applicator that recorded displacement of the skin when the force was applied. This device was found to be reliable (ICC=0.95 at L4/5) but it was also cumbersome and situated with the testing surface on the floor, which would make it difficult to test symptomatic subjects.

The two variables that have been associated with measurement of PA stiffness are stiffness coefficient K and displacement D30 (Latimer et al. 1996a). These two variables are obtained from a force / displacement curve (Figure 1.1). The curve has a non linear part (the toe region) early in the range and a linear part later in range. Stiffness coefficient K is the slope of a regression line fitted to the linear part of the curve. The linear part of the curve is usually found to be between 30 and 90N of force (Latimer et al. 1996a). The force displacement curve is sufficiently linear in this range of force for it to be well represented by a single stiffness coefficient (Lee et al. 1997). This range of forces corresponds to the range of forces used by

physiotherapists (Harms et al. 1999) during Grade II and III mobilisations in Maitland's grading system (Maitland et al. 2001), although there is a large variation between therapists in the amount of force applied (Harms et al. 1999). Harms et al (1999) reported that forces applied by physiotherapists to asymptomatic subjects varied from approximately 25-75N for Grade II and 90-140N for Grade III mobilisations. The other measure of PA response determined from this curve is known as D30 (Latimer et al. 1996a) and describes the displacement that occurs between 2 and 30N of force. This range of force corresponds to the toe region of the force displacement curve and so D30 describes the amount of displacement early in range. Both stiffness coefficient K and displacement D30 are measures of the mechanical behaviour of the spine during the loading phase of the force displacement curve. They do not give any information about the mechanical properties of the spine during the unloading phase. This thesis will only be concerned with properties of the spine during loading phase.

The mechanical devices (Lee and Svensson 1990; Latimer et al. 1996b) provide reliable and accurate measures of PA stiffness, however, it is not clear how closely the forces applied replicate those used in the manual assessment of PA stiffness. One difference between mechanical and manual measures is that in the clinic, manual assessment is carried out with the patient lying on a padded plinth. The surface of the mechanical device is rigid and unpadded to avoid deformation so that measures can reflect the stiffness of the subject rather than the testing plinth. Stiffness measured with the mechanical device is less when a padded surface is used compared to when a rigid surface is used (Maher et al. 1999). Therefore, it is important for mechanical measurement of spinal stiffness to be carried out on the

same surface and an unpadded surface may give a better indication of spinal stiffness. This would ensure that any measured change is due to real change rather than change in the testing procedure or conditions.

Anecdotal reports from subjects indicate that testing with the device feels different to manual testing. One possible difference may be the contact area of the probe on the skin surface, which is smaller than the contact area of the therapist's hand. People use an average contact area of $16cm^2$ when they apply a PA force to elastic stiffness stimuli (Nicholson et al. 1998) whereas, the area of the probe on the mechanical device is $6cm^2$. In addition, the pisiform area of the hand is soft and fleshy and conforms to the skin surface compared to the probe of the mechanical device, which is firm plastic and does not mould at all to the contact surface. Testing with the mechanical device is likely to be less comfortable to the subject and could impact on the state of relaxation and therefore, the amount of muscle activity that might occur locally in response to discomfort.

Other factors that may alter the comfort of loading to the subject are the timing of the loading cycle and the rhythm of loading. The mechanical device applies the force with equal time in the loading and unloading parts of the cycle. The times spent in the loading and unloading phases may not be equal during manual application of the force and this may impact on comfort to the subject and the lumbar PA response. The mechanical device also applies the force with a constant rhythm, whereas the rhythm may vary during a manually applied force. Further investigation of timing of the loading cycle and rhythm of loading is warranted to determine whether it is possible for the device to more closely replicate manual testing to more

clearly explain practice. The consistent application of force by the mechanical device makes it suitable for evaluating stiffness for research purposes. However, for the results to be useful for clinical practice the device should aim to replicate clinical practice and therefore, duplicate the clinical response.

MUSCLE ACTIVITY

Abnormal muscle function has featured strongly amongst the many theories for the aetiology of pain and dysfunction in patients suffering LBP. Muscle spasm seems to be the most common theory (Cassisi et al. 1993) and has been the subject of many investigations. Most of the investigations have involved patients suffering chronic LBP and only a few mention it as a possible factor in patients with acute LBP.

Increased muscle tension is the term most commonly used to refer to muscle spasm (Travell and Simons 1983; Nouwen and Bush 1984) although these terms are sometimes used interchangeably. In this thesis the term "muscle activity" will be used and abnormal muscle activity in pain subjects will be referred to as increased or decreased.

The concept that increased muscle activity has an important association with LBP is not new. Pioneers of manual therapy believed that increased muscle activity of the lumbar extensor muscles was responsible for the pain and disability that they observed in their patients (Mennell 1960; DePalma and Rothman 1970; Caillet 1995). Current texts of internal medicine and manual therapy also link muscle spasm to LBP and dysfunction (Isselbacher et al. 1994; Maitland et al. 2001). It

seems that these claims are made on the basis of clinical observation or hypotheses about the source of symptoms as no evidence is offered to support this belief.

Reflex muscle activity occurring as a response to pain is the mechanism by which muscle spasm is said to be involved in the production of symptoms in LBP (Mennell 1960; DePalma and Rothman 1970; Farfan 1973; Nouwen and Bush 1984; Caillet 1995). Pain due to increased muscle activity is thought to occur from decreased circulation to the muscle from sustained contraction (Farfan 1973) and although initially resulting secondary to joint dysfunction can become the primary source of symptoms (Mennell 1960; Caillet 1995). This has been described as the pain - spasm cycle (Caillet 1995) which is self perpetuating thus prolonging the symptoms and disability of LBP. It has been described as a reflex mechanism over which the patient has no control (DePalma and Rothman 1970) and which serves to hold the spine in a pain free position (DePalma and Rothman 1970).

Muscle spasm occurring in response to pain and injury has been described as being strong enough to immobilise the joint (Mennell 1960). This statement is an assumption made by Mennell (1960) presumably in an attempt to explain why his LBP patients had lost mobility of their lumbar spine. It has been suggested that muscles can function to increase joint stiffness to minimise the effect of externally applied forces (Neilson and O'Dwyer 1989). Although this suggestion was made in relation to the role of muscle in controlling the position of joints during activities like running, it is possible that muscles can increase joint stiffness to protect the joint from an external painful stimulus such as the application of a PA force to the lumbar spine.

Muscle activity (reflecting a combination of muscle forces and muscle stiffness) has a role in stability of the lumbar spine (Gardener-Morse et al. 1995). This was determined by a series of studies involving the construction of three dimensional models of the lumbar spine which were used to determine the amount of muscle activity necessary to maintain equilibrium of the spine under loading (Stokes and Gardener-Morse 1995). Stokes and Gardner-Morse (1995) suggested that changes in stiffness of the motion segment as a result of injury could alter the mode of transmission of loads through the lumbar spine and its motion segments. Although these studies were based on a model of maximum effort of the lumbar musculature, this suggestion could imply that if a segment of the lumbar spine is injured and there is increased muscle activity present at that segment because of that injury then this would result in increased stiffness of the segment when assessed with PA pressures applied to the lumbar spine.

Increased muscle activity as a source of pain and dysfunction has been investigated to attempt to understand the factors contributing to pain and disability in patients with LBP. A common hypothesis is that patients with LBP have increased muscle activity at rest and during various functional activities. This hypothesis is not completely supported by experimental findings. Back muscle activity (erector spinae mean amplitude) at rest is no different in patients with LBP than normal subjects (Kravitz et al. 1981) and may even be decreased in LBP patients during rest periods (Cassisi et al. 1993). The findings of these studies do not support the theory that increased back muscle activity at rest is associated with LBP but do not rule out the possibility that functional activity is painful due to altered muscle activity.

Altered muscle activity during functional tasks has also been investigated in an attempt to explain the mechanism of pain production in LBP. One study demonstrated a decrease or no change in erector spinae activity (EMG amplitude) during a Valsalva manoeuvre and sit ups and increased erector spinae activity while lifting a load (Soderberg and Barr 1983). No changes were observed in abdominal muscle activity during these functional tasks in subjects with LBP (Soderberg and Barr 1983). Decreased EMG amplitude of the lumbar paraspinal muscles also occurs during activities involving lumbar flexion (Cassisi et al. 1993). Cassisi et al. (1993) proposed a muscle deficiency model for LBP suggesting that pain arises because the muscles are not capable of generating sufficient torque to perform the activity efficiently. This may be true for some activities, however increased erector spinae muscle activity (EMG amplitude) has been reported during some functional activities such as lifting a load (Soderberg and Barr 1983). These findings would support a muscle model in which increased muscle activity would be expected to occur with functional tasks. The results of these studies are variable and inconclusive about the contribution of erector spinae muscle activity to LBP. Perhaps the only possible conclusion is that during functional tasks subjects with pain exhibit different erector spinae muscle activity than pain free controls.

Analysis of the changes in muscle function in people with chronic low back pain indicates that the concepts of a vicious pain spasm cycle and hyperactivity of muscle in repose the pain are not correct (Lund et al. 1991). It seems that there is decreased activity when the back muscles act as agonists and increased activity when they act as antagonists. These changes in activity are thought to occur as reflex adaptations

that may reduce further injury, and as such, have led to the proposal of a pain adaptation model (Lund et al. 1991).

While there has been considerable investigation of the behaviour of muscle activity during functional tasks and at rest there have been no investigations to determine whether increased muscle activity occurs in response to the application of PA forces to the lumbar spine in patients with LBP. Despite this lack of investigation one of the aims of examination with PA pressures is to determine whether increased muscle activity is present (Maitland 1986). An increase in EMG amplitude of the back muscles occurs during application of manipulative procedures (Herzog et al. 1999) and an increase in erector spinae activity (root mean square) occurs in response to slow sustained forces (Kawchuk and Fauvel 2001) applied to the spine in asymptomatic subjects. Erector spinae activity increases during the application of traction to the lumbar spine of asymptomatic subjects and is greatest immediately after the tractive force is applied (Hood et al. 1981). Traction is considered to be a type of passive technique which can be applied to the spine. If these passive techniques can result in increased back muscle activity (most likely erector spinae) then it is possible that other passive techniques such as PA pressures could also result in increased activity in the lumbar musculature. One of the aims of this thesis therefore, is to investigate the response of the lumbar extensor muscles to the application of PA pressures in symptomatic and asymptomatic subjects both at rest and during voluntary activation of trunk muscles.

MEASUREMENT OF MUSCLE ACTIVITY

Surface electromyography (EMG) is a method frequently used to measure muscle activity in subjects with LBP. The value of surface EMG patients with LBP is controversial. It is not considered acceptable as a stand alone clinical measure for diagnosis of LBP (Pullman et al. 2000), however it is considered useful as part of a more comprehensive evaluation of LBP (Ambroz et al. 2000). Although there have been reports that IM EMG is not as reliable as surface EMG (Komi and Buskirk 1970) other reports indicate that surface EMG is a reliable and valuable tool for investigation of muscle activity in patients with LBP (Sihvonen et al. 1991).

Activity of trunk and respiratory muscles that occurred during the application of PA forces to the lumbar vertebrae was measured in the studies described in this thesis. Muscle activity is expressed as a voltage, which represents the potential difference between two electrodes placed on the skin surface (Cromwell et al. 1980). The signal detected at the skin surface is the addition of many single action potentials generated from motor units in the muscle under the skin surface (Cromwell et al. 1980; Gilmore and Meyers 1983; Winter 1990). The units of the EMG signal are μV or mV and the magnitude can range from 1μV to 5mV (Gilmore and Meyers 1983). EMG signals generally occur in the frequency spectrum of 0-500 Hz (De Luca and Knaflitz 1992; Turker 1993).

The EMG signal is affected by the anatomical and physiological factors that are intrinsic to the individual as well as those which are extrinsic such as the filtering

features of the environment and the apparatus. It is possible to exert some control over the extrinsic factors by modifying the methodology used to detect and record the signal, whereas the intrinsic characteristics are determined by the structure and workings of the muscle and subcutaneous tissue (De Luca and Knaflitz 1992). The factors which are intrinsic characteristics can be in part controlled for by selecting subjects that have a similar BMI if comparisons between subjects are to be made.

The subcutaneous tissue acts to filter out the high frequency components of the signal so that they are progressively more attenuated as the tissue distance increases (De Luca and Knaflitz 1992; Barkhaus and Nandedkar 1994). The thickness of the subcutaneous tissue affects the signal detected by the electrodes (Cromwell et al. 1980) and the signal becomes weaker the greater the distance it has to travel (Cromwell et al. 1980; Gilmore and Meyers 1983). Therefore, the signal will be weaker in a person with a greater amount of subcutaneous fat. In addition, further filtering occurs because the electrical characteristics of the metal detection surfaces and the electrolytes of the skin behave as a high pass filter to the signal and further filtering occurs if a bipolar electrode configuration is used (De Luca and Knaflitz 1992).

Electrode characteristics

The EMG surface electrode is the transducer used to convert the muscle activity to an electrical signal. The electrodes consist of a silver/silver chloride disc covered by an electrolyte gel, which forms the conductive path between the skin and the electrode detection surface. The silver/silver chloride electrodes are very stable

chemically (Cromwell et al. 1980) which helps to eliminate noise arising from chemical activity within the electrode. Disposable surface electrodes are commonly used and are examples of floating electrodes, which are designed to eliminate movement artefact between the metal detection surface and the skin (Cromwell et al. 1980). Movement artefact is eliminated because the metal detection surface is separated from the skin by an electrolyte gel thus forming an electrolyte bridge for conducting the signal (Cromwell et al. 1980). The surface electrodes used for the studies in this thesis have the necessary characteristics for minimising noise arising from the electrodes themselves.

Electrode sensitivity

The sensitivity of the surface EMG electrodes as transducers refers to their ability to detect the electrical activity of the particular muscle or muscles being studied. Surface EMG is reported to be useful if the electrodes are placed over large superficial muscles, however, it is also possible that the recordings reflect activity in neighbouring muscles (Gilmore and Meyers 1983). The detection of signals from neighbouring muscles is referred to as cross talk (Turker 1993). The use of a bipolar electrode configuration with a differential amplifier can to some extent control for this problem by negating any signal common to both electrodes (De Luca and Knaflitz 1992; Turker 1993). Cross talk can be removed by the use of double differential recording (De Luca and Knaflitz 1992), although this method has not been used in the studies presented in this thesis.

Electrode placement

Electrode placement is important for the electrodes to be able to detect activity in the muscles being studied. The studies in this thesis record muscle activity that may be contributing to lumbar PA stiffness. For recordings of the lumbar erector spinae muscles, pairs of surface electrodes are positioned bilaterally, adjacent to the L4 spinous process. Each pair of electrodes is placed 4cm lateral to the L4 spinous process with an inter-electrode distance of 2.5cm. A reference electrode is placed over the sacrum. This electrode placement should enable recordings of activity predominantly from the lumbar erector spinae (Bogduk 1997), however, it is possible that they may also detect activity in other muscles eg multifidus in close proximity which might also be affected by the application of PA forces to the lumbar spine. Identifying the specific muscle contributing to the signal was not the major concern as the main aim of this thesis was to determine whether muscle activity is likely to be contributing to lumbar PA stiffness. Identifying whether other muscles were contributing to the signal may not be possible with surface electrodes and could form the basis for further study.

Accuracy of surface electrode recordings

The accuracy of the surface electrode refers to how accurately the EMG recordings reflect the activity of the underlying motor units and not other electrical activity (Turker 1993). Other electrical activity, ie noise, constitutes the major source of error when interpreting surface EMG signals. It may be difficult to identify unwanted activity so extreme care must be taken with the experimental technique to

eliminate unwanted signals whilst not compromising the signal indicating muscle activity (Turker 1993). Another important factor in improving the accuracy of surface EMG is to select appropriate signal conditioning.

Noise in the EMG signal may arise from factors intrinsic or extrinsic to the body. Intrinsic factors may include other electrical activity within the body such as heart or brain activity or cross talk from other muscles (De Luca and Knaflitz 1992; Turker 1993). Extrinsic factors may be due to mechanical artefacts produced by movement of the electrodes or the electrode leads (Turker 1993), electrical noise from the mains supply, and noise from the amplifier and the recording system (Turker 1993).

Elimination of noise from the signal is essential to ensure that the recorded EMG signal reflects activity from the muscle to be studied. Elimination of extrinsic factors can be achieved by careful preparation of the subject and set up of equipment. Noise can be reduced by thorough skin preparation to lower the skin resistance to below 5000Ω (Gilmore and Meyers 1983) and some authors suggest even below 1000Ω (Winter 1990). The use of chemically stable electrodes such as the silver/silver chloride floating type (Cromwell et al. 1980) minimises noise occurring from chemical activity and movement within the electrode. In addition, noise resulting from mechanical artefacts can be reduced by securing the electrodes and the leads to the subject with tape. The use of short leads reduces mechanical artefact from movement of the leads (Turker 1993). The use of a reference electrode will also help to eliminate external interference (Gilmore and Meyers 1983) as well as help to protect the system against a too large common mode signal.

The use of a bipolar electrode configuration and differential amplifier will tend to cancel common mode signals such as cross talk from other muscles (Gilmore and Meyers 1983; De Luca and Knaflitz 1992; Turker 1993). There are four important specifications of an EMG amplifier; amplifier gain and dynamic range, input impedance, frequency response and common-mode rejection (Winter 1990).

The amplifier gain is the ratio of the output voltage to the input voltage (Winter 1990). All EMG signals must be amplified equally to avoid signal distortion (Winter 1990).

The input impedance of the amplifier must be high enough to prevent attenuation of the EMG signal (Winter 1990). Input impedance greater than 1 MΩ is recommended (Winter 1990; Turker 1993). The impedance between the electrode and the skin is influenced by factors such as the thickness of the skin, skin preparation prior to the application of the electrodes, the area of the detection surface and the temperature of the electrode paste (Winter 1990). It is recommended that the skin be prepared to reduce the impedance to be less than 1000Ω (Winter 1990). This can be done by shaving, abrading and thoroughly cleansing the skin before the electrodes are applied (Gilmore and Meyers 1983) and measuring the resistance with a multi-meter. The above procedure of skin preparation has been adopted for this thesis and values of skin resistance less than 5000 Ω were considered acceptable. This value is higher than the one recommended by Winter (1990), however, this value is often used in research involving surface EMG (Gilmore and Meyers 1983).

The frequency bandwidth of the amplifier should amplify all frequencies in the EMG signal (Winter 1990). Recommended values for frequency bandwidth range from 10-100 Hz (Winter 1990) to 0-500 Hz (De Luca and Knaflitz 1992; Turker 1993) which is more common.

Common-mode rejection is where the differential amplifier rejects signals which are common to both active terminals when electrodes are applied in a bipolar configuration (Winter 1990). A perfect subtraction of the common-mode signals never occurs so the common mode rejection ratio (CMRR) is a measure of how well the subtraction occurs. It is recommended that the CMRR be higher than 1000:1 (Turker 1993), although good quality amplifiers have a CMRR of 10000:1 (Winter 1990).

The area of the detection surface of the electrode affects the impedance and the detection volume of the electrode. The larger the detection surface the lower the electrode impedance and the greater the detection volume (De Luca and Knaflitz 1992). An inter-electrode distance of 10mm is recommended which should allow signals from a significant portion of the muscle to be collected while minimising unwanted signals (De Luca and Knaflitz 1992). Greater inter-electrode distances are still acceptable (De Luca and Knaflitz 1992) and may be appropriate when there is a large volume of muscle eg erector spinae to detect a representative sample of activity occurring in the muscle.

In addition, subjects need to be electrically isolated so that low voltage hazards are eliminated and possible current paths through the patient are eliminated. Isolation can be achieved by the use of isolated circuits or power isolation transformers (Du Bovy 1978).

Data processing and analysis procedures

Data processing and analysis procedures must be undertaken with consideration of potential sources of noise in the recording system (Turker 1993). The EMG signal can only be interpreted with confidence if the investigator is sure that it reflects activity of the motor units being studied. In Chapter Three the use of dual recording (chart recorder and computer) enabled identification of any discrepancies in the data due to noise by allowing examination of raw data made with the chart recorder. In the later chapters the use of equipment with a real time display of EMG and data acquisition software that can perform a fast fourier transform (FFT) allowed the immediate identification of noise in the data.

When comparing EMG activity between individuals a normalisation procedure is recommended to reduce inter-subject variability (Turker 1993). Usually the recorded EMG signals are expressed as a percentage of a maximal voluntary contraction (Sihvonen et al. 1991). However, in some instances maximal voluntary contraction may not be possible eg with subjects with LBP, and a submaximal contraction may be used. Submaximal contractions are an accepted form of normalisation and are considered by some to be more reliable then maximal contractions (Yang and Winter 1983).

Controversy exists over the most appropriate method for normalising EMG data to obtain the values that best reflect the behaviour of the EMG signal for statistical comparison. Most of the literature focussing on activity of the lumbar extensors measured the amplitude of activity (Nouwen and Bush 1984; Sihvonen et al. 1991). In other studies the researchers asked the subjects to perform an activity and measured an index of fatigue (De Luca 1993). Neither of these approaches is appropriate for identifying an involuntary or reflex response of the lumbar muscles as a result of a stimulus applied to the lumbar spine. Mean and peak ensemble averaging have also been used, however, these methods have been criticised as they tend to eliminate the characteristics of the EMG signal that are of interest (Yang and Winter 1984). It is important to select a method of processing that will not remove the characteristics that most accurately reflect the pattern of activity that were observed in the raw data.

In summary, surface EMG is an appropriate tool for non invasively recording muscle activity from large superficial muscle groups in symptomatic and asymptomatic subjects. Good experimental technique is essential to eliminate noise and artefacts from the signal. It is important to be aware of the signal conditioning process so that any distortions present in the signal can be recognised. Normalisation of the EMG signal allows comparison of mean amplitude data between different subjects although there is some difference of opinion about the best method. Normalisation using either maximal or submaximal contractions is considered acceptable.

RELATIONSHIP OF LBP, MUSCLE ACTIVITY AND STIFFNESS

Physiotherapists rely heavily on the assessment of PA stiffness and muscle activity in the clinical examination to make treatment decisions in patients with LBP. While there is some evidence that increased PA stiffness is associated with LBP (Latimer et al. 1996c) and increased muscle activity occurs during the application of manual techniques to the spine in asymptomatic subjects (Herzog et al. 1999), there is little scientific evidence to support a relationship between LBP muscle activity and PA stiffness.

Lumbar PA stiffness was increased during an episode of LBP compared to when the pain resolved (Latimer et al. 1996c). Increased muscle activity is one of the proposed mechanisms of increased stiffness in response to applied PA forces. Maximal voluntary contraction of the spinal extensors increases lumbar PA stiffness in healthy subjects (Lee et al. 1993). These reports led to speculation that the small amounts of spontaneous muscle activity that were observed in response to PA forces applied to the spine in subjects with LBP may also increase PA stiffness (Shirley and Lee 1993). It is not known whether spontaneous involuntary muscle activity can alter PA stiffness so consideration of the possible mechanisms involved is warranted to determine whether it is likely.

Increases in muscle activity have been documented in response to the manual application of forces to the spine via therapeutic techniques. Chiropractic manipulation applied to the spine resulted in activation of muscles adjacent to the

spine and also muscles distant from the spine (Herzog et al. 1999; Kawchuk and Fauvel 2001). It was suggested that the activity was due to the activation of a stretch reflex (Herzog et al. 1999). In contrast, other studies have reported a decrease in excitability of the H reflex of the soleus muscle (Dishman and Bulbulian 2000) and a decrease in the amplitude of activity of spinal extensors during a sustained load to the spine (Kawchuk and Fauvel 2001) following the application of manual treatments to the spine. It is difficult to interpret the differences between these conflicting results. However, the increases in activity (Herzog et al. 1999; Kawchuk and Fauvel 2001) were recorded during application of the force and the decreases in activity were recorded after application of the force (Dishman and Bulbulian 2000; Kawchuk and Fauvel 2001). Therefore, it is possible that two different mechanisms are involved particularly as both of these responses have been observed in the same group of subjects (Kawchuk and Fauvel 2001). The increased activity reported during application of the force may be due to an initial increased excitability of the motor neurones. The decreased activity post application of forces may be due a latent decreased excitability caused by the manually applied force activating the descending inhibition system (Wright, 1995).

If increased muscle activity is involved in the mechanism of increased PA stiffness during assessment with PA forces then increased excitability at the time of force application is the most likely mechanism. Previous work suggests that the muscle response is related to the speed of the force application (Herzog et al. 1995). Although the application of PA forces during physiotherapy assessment of the spine is slower than a chiropractic manipulation, it is faster than the slow sustained loading employed by Kawchuk and Fauvel (2001). It is not known whether PA forces

applied during physiotherapy assessment are fast enough to provoke muscle activity. The behaviour of muscle activity and PA stiffness in response to applied PA forces has not yet been investigated in both symptomatic and asymptomatic subjects.

Pain mechanism

A review of the neurophysiological processes involved in the perception of pain is necessary to explore the mechanism whereby muscle activity could result in increased lumbar PA stiffness in people with LBP. Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage" (Merskey and Bogduk 1994) p 210. Therefore, pain is a complex experience which may involve the interaction of one or a number of factors such as nociception and neuropathy, and will also be influenced by psychologic and environmental factors (Siddall and Cousins 1997).

Pain can be experienced when a noxious stimulus is applied to the lumbar spine. Such a stimulus might involve tissue damage, inflammation or in some cases even movement of innervated structures (Siddall and Cousins 1997). These structures include the posterior annulus of the intervertebral disc, capsule of the zygapophyseal joints, synovium, ligaments, muscle and associated tissue eg nerves (Bogduk 1997).

During the process of nociception action potentials ascend from the site of the stimulus up to the cerebral cortex. This requires free nerve endings of nociceptors to be present at the stimulus site (Cavanaugh 1995; Siddall and Cousins 1997). The

axons of these free nerve endings then transmit the action potentials to the dorsal horn of the spinal cord where they make their first synapse (Cavanaugh 1995; Siddall and Cousins 1997). The signal is then continued by a second order neuron in the white matter of the contralateral side of the spinal cord where a second synapse is made (Cavanaugh 1995). The signal is then relayed to the somatosensory cortex of the brain by a third order neuron (Cavanaugh 1995). The nociceptive signal can be modulated by descending inhibitory circuits in the spinal cord and excitatory and inhibitory circuits in the dorsal horn (Cavanaugh 1995).

The above mechanism describes the process involved in the transmission of a noxious stimulus. In many cases of LBP the pain sensation persists long after the noxious stimulus has been removed. The mechanisms involved in persisting pain are known as peripheral and central sensitisation (Siddall and Cousins 1997). Peripheral sensitisation and primary hyperalgesia occur when the peripheral nerve endings become sensitised by chemicals released during tissue damage or inflammation (Cavanaugh 1995). This results in a lowering of the threshold of some of the receptors e.g. mechanoreceptors (Cavanaugh 1995) so that movement of the spine that would not normally provoke pain becomes a noxious stimulus (McMahon and Koltzenberg 1990). Initially this process might serve as a protective mechanism to alert the body to potential further damage, however, since pain persists after the expected healing time its purpose is unclear.

Central sensitisation and secondary hyperalgesia result from processes that occur in the dorsal horn of the spinal cord that leads to a "wind up" of neuronal activity in the spinal cord (Siddall and Cousins 1997). This increase in neuronal activity results in

expansion of the size of the receptive field, an increase in the magnitude and duration of stimuli that are higher than the threshold, a reduction in threshold so that innocuous stimuli activate receptors that are involved in nociception (Siddall and Cousins 1997). In other words the receptors become hypersensitive to all stimuli even if they are usually innocuous (such as mechanical stimuli) resulting in a pain response from stimuli such as touch and movement. Therefore, both central and peripheral sensitisation result in lowering the threshold of receptors in the area related to the site of injury and even tissues remote from the site of injury.

Reflex muscle activity

When the threshold of receptors is lowered due to noxious input, excitability of motor neurons is increased (Vujnovich 1995) and could lead to reflex activity. Muscle spasm is sometimes initiated by reflex muscle activity (Vujnovich 1995) and can be defined as a sustained involuntary contraction which is usually painful and cannot be relieved by voluntary effort (Fisher and Chang 1985). The exact neurophysiological mechanism involved in the production of muscle spasm is not clear but it may be reflex activity associated with one or more of the reflex types that have been described in relation to musculoskeletal pain. These reflex types could include a stretch or somatic reflex (Denslow and Clough 1941), polysynaptic pathways (Nade et al. 1978) and the flexion withdrawal reflex (Wall and Woolf 1984).

Stretch Reflex

Reflex muscle activity occurring in response to stimulation of tissues in the lumbar spine is well documented in humans (Denslow and Clough 1941; Elliott 1944; Denslow et al. 1947) and animals (Nade et al. 1978). Reflex activity in the spinal extensor muscles occurs in response to manual palpation of areas of abnormal tenderness within the muscle (Denslow and Clough 1941). It was proposed that abnormal pressures developed in dysfunctional joints caused reflex muscle activity, similar to stretch or postural reflexes (Denslow and Clough 1941). Although, it has not been determined whether this is the mechanism of reflex muscle activity, activation of the stretch reflex arc is one possibility.

The stretch reflex arc is monosynaptic whereby the afferent neuron enters the dorsal horn of the spinal cord and synapses with the efferent neuron in the ventral horn. The efferent neuron then exits to the target tissue in the periphery (Schmidt 1985). Normally the stretch reflex is activated by stretch to muscle which results in contraction of the muscle (Willis and Grossman 1977). This is the type of response seen in deep tendon jerks to assess integrity of the neural system. The nervous system is capable of increasing the stiffness of joints by muscle activation to keep movement caused by external forces to a minimum during dynamic activity (Neilson and O'Dwyer 1989). It was proposed that joint stiffness could be regulated by control of the stretch reflex mechanism, which involves self-generated activation of the muscle causing it to produce a reflex force to oppose the applied stretch (Neilson and O'Dwyer 1989). Further, it is suggested that in disease states the reflex may be hyperactive causing the muscle to oppose stretch when there is passive movement of

a joint (Willis and Grossman 1977). Therefore, muscle activity may occur in the lumbar spine as a response to an externally applied PA force to stiffen the spine and limit movement in a PA direction.

The passive extension of the spine that occurs during application of a PA force (Lee and Moseley 1991) would involve movement of the erector spinae muscles. It is possible that passive extension of the spine could activate the stretch reflex in the erector spinae and deeper multifidus and muscles. In addition, if the neural system is in a state of increased sensitivity or "windup" the manual application of a PA force to the lumbar spine could also stimulate mechanoreceptors and provoke a pain response, which triggers the stretch reflex. Activation of these muscles may restrict movement in a PA direction (physiological extension) and result in an increase in PA stiffness.

Opposing this theory is the proposal that manual therapy reduces pain and increases movement by stimulating discharge from joint afferents resulting in inhibition of muscle contraction (Zusman 1986). This proposal is supported by the recent reports that muscle activity is decreased following manual techniques in asymptomatic subjects (Dishman and Bulbulian 2000; Kawchuk and Fauvel 2001). The distinction between applying PA forces which cause muscle activity, and forces which inhibit muscle activity may rest with the magnitude of PA force applied. It seems possible that applying a force that provokes pain might lead to increased muscle activity whereas applying a force that does not provoke pain has an inhibitory effect on muscle activity. This is consistent with the clinical practice of avoiding pain

provocation when applying a PA mobilisation when the aim of treatment is to reduce pain (Maitland et al. 2001).

When pressure loads are applied to the spinous processes of the lumbar spine low threshold segments of the spine demonstrate reflex hyperactivity (Denslow et al. 1947). Low threshold segments have a large proportion of motoneurons in a state of facilitation to bombardment of impulses from an unknown source but that probably arise from structures related to that segment (Denslow et al. 1947). It is possible that spasm occurs as a result of either peripheral or central sensitisation, which could involve either the stretch reflex or a more complex centrally mediated polysynaptic reflex.

Polysynaptic pathway

Evidence for the increased muscle activity occurring as a result of central sensitisation (Siddall and Cousins 1997) and possibly involving a reflex mediated by a complex polysynaptic pathway (Nade et al. 1978) is suggested by the response of muscles to the insertion of needle electrodes (Elliott 1944). Muscle activity occurred when needle electrodes were inserted into tender muscles and also in response to deep palpation of the surrounding muscle (Elliott 1944). Furthermore, it was suggested that the increased activity resulted from a disturbance of central origin rather than a response to the local inflammatory process (Elliott 1944). It is possible that a state of excitability may be induced in the anterior horn cells of the spinal cord that results in motor activity (Elliott 1944). The excitability of anterior horn cells

could occur as a result of central sensitisation and if involved in a reflex, a complex polysynaptic pathway seems most likely.

Flexion withdrawal reflex

The flexion withdrawal reflex that occurs in response to pain is another possible mechanism to explain muscle spasm occurring in the lumbar spine. The flexion withdrawal reflex results in the part of the body being rapidly pulled away from a painful stimulus (Willis and Grossman 1977). Animal studies report that the threshold of the flexion withdrawal reflex can be lowered in response to noxious stimulation (Qing-Ping and Woolff 1995). Activation of the flexion withdrawal reflex during PA force application to the lumbar spine could cause the spine to move away from the source of force application by moving into extension, thereby increasing PA stiffness.

Summary

Review of literature on reflex muscle activity suggests that increased muscle activity in the lumbar spine that occurs in response to a manually applied mechanical load is most likely to be due to initiation of the stretch reflex. The stretch reflex arc is initiated by loading tissues of the lumbar spine that have been sensitised by tissue damage or the associated inflammation and, even though the load would not normally be noxious it is perceived as painful. The mechanical load activates the afferent neurons of the stretch reflex arc resulting in activation of the motor units in the muscle in an attempt to control movement of the spine. Thus, by controlling

movement of the spine in a PA direction when PA force is applied the muscles could increase PA stiffness.

Muscle activity that occurs as a response to PA loading in people with LBP could be a reflex response to an unexpected perturbation in an attempt to protect the spine from further pain/damage or the normal function of the muscles to modulate stiffness of the spine (Hodges 2000). The pain adaptation model proposed by Lund et al. 1991 might explain the activation of muscles to limit movement of the spine when PA forces are applied and therefore potentially prevent further injury or pain. Modulation of stiffness may be more likely than reflexes which are latent responses and possibly too slow to respond to immediate demands of joint protection (Johansson et al. 1991). In contrast, EMG responses of the back muscles observed during chiropractic manipulations are described as reflexogenic in nature (Herzog et al. 1999). Therefore, EMG responses of the back muscles have been observed in response to rapidly applied forces in asymptomatic subjects, however the exact mechanism remains speculative. In some people there may be activation of a stretch reflex and in others there may be an increase in the overall amplitude of activity. While erector spinae muscles contribute to stiffness of the spine during maximal voluntary activation (Lee et al. 1993), it is possible that erector spinae and other muscles that stabilise the spine could also be activated during applied PA forces and also contribute to PA stiffness.

Back muscles

Back muscles likely to be involved in increased activity when PA force is applied to the spine are the superficial erector spinae and the deeper multifidus. The superficial erector spinae produce extension and contribute to unilateral movements of the spine (Bogduk 1997). More specifically, activation of these muscles causes posterior sagittal rotation of the vertebrae (Bogduk 1997) and increased resistance to anterior shear (Potvin et al. 1991) and consequently, probably increases PA stiffness of the vertebral column. Multifidus is a small segmental muscle and produces posterior sagittal rotation of the vertebrae (Bogduk 1997). In addition, the fibres of deep multifidus may be responsible for control of intervertebral motion (Moseley et al. 2000) and are likely to increase intervertebral stiffness. Therefore, the superficial muscles are primarily responsible for orientation of the spine and the deeper muscles act to control intervertebral movement. Contraction of erector spinae and multifidus muscles could increase PA stiffness by their action at individual vertebral levels. Thus, when a PA force is applied during muscle activation it is likely that the resistance to anterior vertebral movement will result in increased lumbar PA stiffness.

Other trunk muscles

Other trunk muscles also play a role in controlling stability of the spine. The muscles that have been studied to date include the abdominal muscles and the diaphragm. The superficial abdominal muscles external oblique (EO), internal

oblique (IO) and rectus abdominis (RA) are responsible for global movements of the spine and transversus abdominis (TrA) controls intervertebral motion (Hodges and Richardson 1999b). Activity of TrA occurs prior to limb movements that result in perturbations of the spine and is thought to control spinal movement by producing intra-abdominal pressure (IAP) or by tensioning the thoraco-lumbar fascia (Hodges and Richardson 1997b). Changes in IAP may contribute to increasing stiffness of the intervertebral joints of the spine (Cresswell and Thorstensson 1994). It is also thought that lateral tension of the thoraco-lumbar fascia may influence translations of the intervertebral joints by applying tension to the transverse processes of the lumbar vertebrae, or by making the trunk a rigid cylinder by stiffening the spine (McGill and Norman 1993). Therefore, in a similar situation to the back muscles, the superficial abdominal muscles produce movements to orient the spine and TrA is responsible for controlling stability of the spine.

The diaphragm also contributes to stability of the spine. The diaphragm is primarily a respiratory muscle, which is active during inspiration to increase lung volume and also increases IAP (De Troyer and Estenne 1988). The diaphragm consists of two components; the costal diaphragm and the crural diaphragm which directly attaches to the first three lumbar vertebrae (De Troyer and Estenne 1988). It is proposed that the diaphragm could contribute to stability of the spine either indirectly by its role in increasing IAP or by directly contributing to stiffness of the spine and controlling intervertebral movement (Hodges and Gandevia 2000b). The contribution of the diaphragm to stability of the spine has been demonstrated during repetitive movements of the upper limb where the diaphragm is observed to be active

throughout the duration of limb movement and not just during the respiratory cycle (Hodges and Gandevia 2000b).

The diaphragm is able to be active during respiration and simultaneously carry out its role of stability. This is also true for TrA, which can perform both respiratory and stability function at the same time. It is postulated that these muscles may have different fibres responsible for each of their functions (Hodges and Gandevia 2000a).

The diaphragm and TrA are both thought to stiffen the spine. Studies that have examined the role of these muscles in spinal stability have done so while subjects perform a functional task that results in perturbations of the spine. The role of these muscles in stability of the spine when passive unexpected perturbations such as PA forces are applied has not been determined. However, as both muscles play a role in stabilising the spine and are thought to do this by increasing spinal stiffness, it is possible that these muscles could be recruited to stabilise the spine during application of passive PA forces. Whether this would be different in subjects with LBP is open to debate. Transversus abdominis does not perform its stabilising role as well in subjects with LBP (Hodges and Richardson 1999a) and some people with acute LBP have a decreased diameter of multifidus (Hides et al. 1994). Finally, the role of the diaphragm in spinal stability of people with LBP is unknown. Therefore, it is necessary to determine the response of these muscles to PA forces in asymptomatic subjects to obtain a baseline before investigating the response in people with LBP.

SUMMARY

Low back pain is a common problem in the world today and the associated costs and disability are escalating. Manual therapy has been used throughout time in the assessment and treatment of LBP but the mechanisms of effect are not yet well understood. Postero-anterior forces are used to assess lumbar PA stiffness. People with LBP have increased lumbar PA stiffness and increased muscle activity is proposed as one of the factors that influence increased stiffness. A number of muscles are considered to increase stiffness of the lumbar spine. Understanding the relationship between PA stiffness and muscle activation in response to PA forces in symptomatic and asymptomatic people will advance the knowledge of the mechanisms underlying increases in lumbar PA stiffness. This may enable the identification of those patients most suited to treatment with manual therapy.

The review of the literature in this chapter has highlighted some deficiencies in knowledge concerning the contribution of trunk muscle activity to lumbar PA stiffness. The purpose of this thesis was to investigate this contribution and add to the body of knowledge relating to the relationship between trunk muscle activity and lumbar PA stiffness. Many of the previous studies of muscle activity in response to applied forces to the spine have considered the response of the erector spinae muscle. There have been no reports of how other muscles such as the abdominal muscles and respiratory muscles (including the diaphragm) contribute to PA stiffness. To date the role of these muscles can only be suggested from investigations of their role in stabilising the spine during functional activities.

The study in Chapter 2 was necessary in order to ensure reliability of the custom made stiffness testing device. While reliability of similar devices has been ascertained previously there has not been a report of the reliability of the stiffness

response (Stiffness coefficient K and D30) from cycle to cycle within a test. It was essential to understand the cyclic behaviour of the stiffness response so that the later studies evaluating muscle activity could be correctly interpreted.

The study in Chapter 3 investigated the effect of maximal and submaximal activity of the lumbar extensor muscles in PA stiffness. Particular emphasis was placed on muscle activity equivalent to small percentages of MVC. This was undertaken to gain an understanding of whether small amounts of lumbar extensor muscle activity could increase stiffness so that the results of Chapter 4 could be evaluated. Chapter 4 was conducted to determine to response of the lumbar muscles to applied PA forces in subjects with LBP compared to subject without LBP. This has not been determined in a symptomatic population before.

Many muscles have been proposed to contribute to stability of the spine and thus could also be involved in increasing stiffness of the spine. Some of these muscles include the lumbar erector spinae, abdominal muscles and respiratory muscles. The studies in Chapters 3 and 4 only measured activity of lumbar erector spine and thus were not able fully explain the contribution of trunk muscle activity to PA stiffness in subjects with LBP. In order to get a more complete picture of which trunk muscles contribute to lumbar PA stiffness the studies in Chapters 5 and 6 were conducted to provide information about the respiratory and abdominal muscles as well as the erector spinae muscles. In addition, as IAP has also been shown to contribute to stability of the spine and to possible stiffness the spine its contribution to PA stiffness was also evaluated in Chapter 6. It was appropriate to determine the contribution of respiratory and abdominal muscle and IAP to AP stiffness in people without pain before determining what happens in people with LBP.

CHAPTER TWO

RELIABILITY: THE RESPONSE OF POSTERO-ANTERIOR LUMBAR STIFFNESS TO REPEATED LOADING

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ABSTRACT

Lumbar postero-anterior (PA) responses are determined by manual examination and are used to guide treatment decisions and interpret changes in symptoms within and between treatments. Mechanical devices that simulate manual assessment have been developed to measure lumbar PA responses. Two variables used to describe lumbar PA responses to mechanical loading are stiffness coefficient K and displacement D30. The purpose of this study was to investigate the behaviour of lumbar PA responses with repeated loading over time.

Lumbar PA responses at L4 were measured in 18 pain free subjects using a mechanical device. Measurements were made for five consecutive loading cycles on three test occasions. The responses were compared between the five cycles within a single test occasion and between three test occasions. An identical procedure was also used to test a set of elastic springs for comparison.

There was a significant increase in both stiffness coefficient K and displacement D30 between the first cycle and subsequent cycles of a single test occasion on human subjects. This response which demonstrates an increase in stiffness and displacement between the first and subsequent cycles can be considered a normal response to PA loading. Lumbar PA responses (stiffness coefficient K and displacement D30) remain constant over several tests both within one day and between days.

INTRODUCTION

Assessment with postero-anterior (PA) forces is performed to identify segments of the spine with limited mobility (clinically, sometimes called increased stiffness) and pain. Mechanical devices that simulate manual assessment (Latimer et al. 1996b) reliably measure PA stiffness on subjects with pain within a treatment session, suggesting that stiffness remains consistent over a short period of time. Stiffness has also been reported to be stable in subjects without pain over two sessions, separated by around one day (Lee and Svensson 1990) or several days (Latimer et al. 1996c). However, there have been no reports on whether the behaviour of PA stiffness varies between individual cycles of a stiffness test. Also, the behaviour of stiffness within a day and between days has not been reported for the same group of subjects.

Cyclic variations in behaviour of tissues in the lumbar region have been determined in a group of subjects without low back pain (Lee and Evans 1992). Lee and Evans (1992) applied three PA loading cycles to L4 spinous processes using a mechanical device. The response was described by non linear curves with a progressive increase in displacement during loading. The increase in displacement was greatest between the first two loading cycles. A similar pattern of cyclic variations of lumbar spine tissues has also been reported in other studies using cadaveric specimens (Yahia et al. 1991; Asano et al. 1992).

While the purpose of this study is primarily to determine the reliability of the stiffness testing device, it also explores some previously unexplained findings

related to stiffness testing. Previous studies have excluded the first cycle loading cycle from analysis due to an observed different behavior (Latimer et al. 1996a; Latimer et al. 1996c) This study will determine the cycle to cycle behaviour as well as the reliability from cycle to cycle which has not been previously reported. It is important to understand the PA stiffness response before the device can be used to evaluate other responses, eg muscle activity, in relation to stiffness.

The aim of this study was to determine the behaviour of the lumbar PA response (both K and D30) during repeated loading with a PA force similar to PA mobilisation within one test (which might correspond to one assessment dose), between two test occasions within the same day and between two testing days. This response was compared with elastic (steel) springs in order to differentiate the human characteristics of the response.

METHODS

Experimental Design

This study used a repeated measures design where measurements of spinal PA stiffness were taken on two separate occasions. On the first occasion spinal stiffness was measured twice with a five minute interval between tests. Subjects then returned on a second occasion between two and eight days later for a third test.

Subjects

Eighteen healthy subjects without low back pain volunteered for the study. The sample consisted of 12 female and 6 male subjects with a mean age of 28 (SD 7) years. Subjects were excluded if they reported the presence of any spinal disease or if they reported experiencing low back pain for which they had sought treatment within the previous six months. Subjects were recruited from the staff and students at a university campus. The study had ethical approval from the University's Human Ethics Committee.

Measurement of PA responses

Lumbar PA responses were measured using a portable mechanical device (Nebula Electronics) (Figure 2.1) that was custom made to apply PA forces to the lumbar spine in a manner similar to the way a physiotherapist manually performs a PA mobilisation. The device has been described in detail elsewhere (Latimer et al. 1996b) however a brief description is also provided here. The force is applied by a mechanically driven probe which makes contact with the skin over the subject's L4 spinous process at an angle of 4.5° in a caudal direction (Stagnara et al. 1982). The L4 spinous process was chosen because the L4/5 intervertebral level is often symptomatic in people with LBP (Maitland et al. 2001). The force applied by the probe is measured by a strain gauge and the resulting displacement of the skin in a PA direction is measured by a linear potentiometer. The device has been shown to have high reliability (ICC=0.96) for test-retest measurements of stiffness within one testing session (Latimer et al. 1996a) and a high level of accuracy (maximum error of 2.5%) for testing elastic beams (Latimer et al. 1996a).

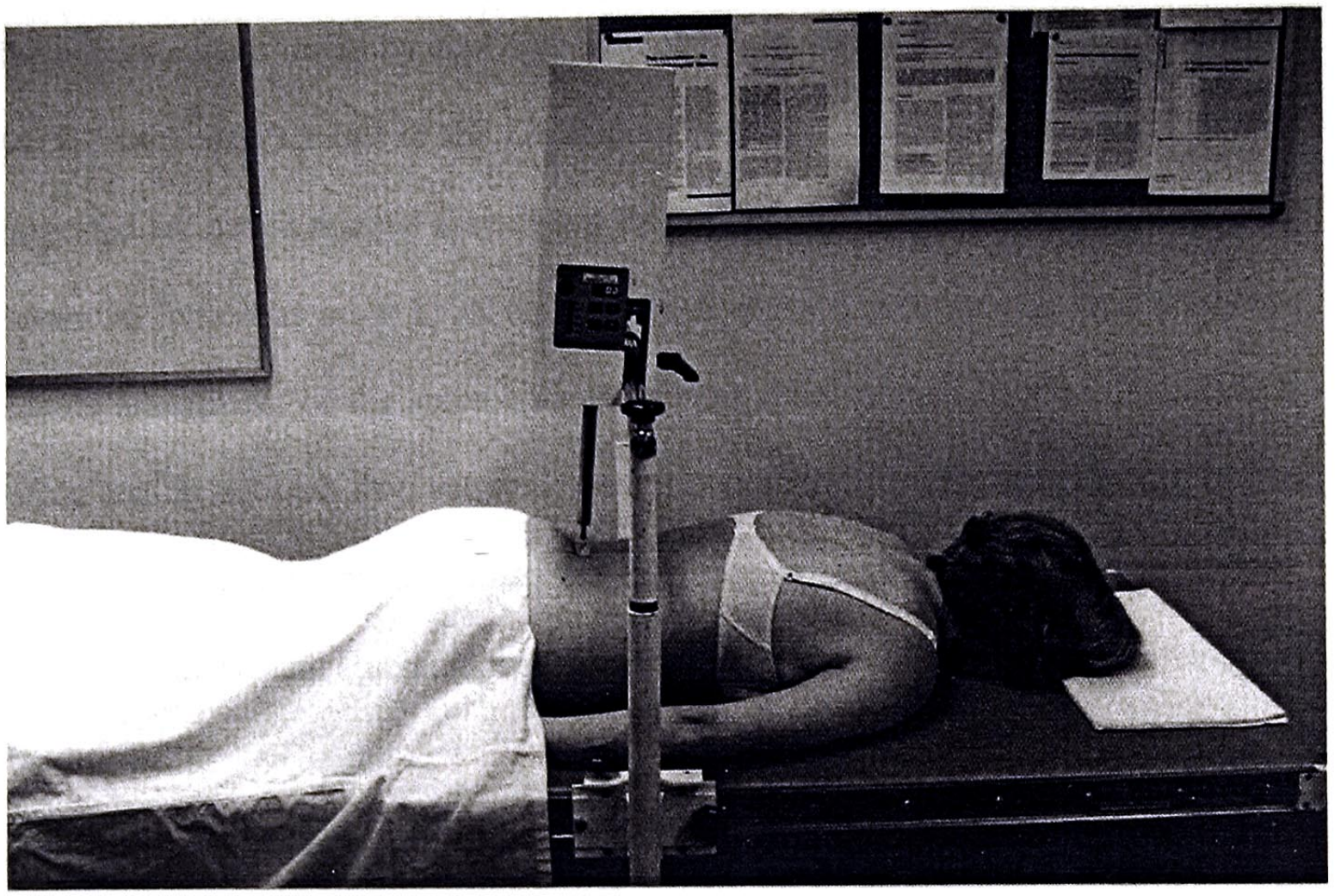

Figure 2.1. Mechanical Device for testing lumbar PA responses

Procedure

Each subject's lumbar spine was examined to locate and mark the L4 spinous process. The L4 spinous process was identified by manual palpation, using the procedure described by (Grieve 1984) to identify the L5 spinous process and the L4/5 interspace, and marked. The subjects were asked to lie prone on the testing plinth with the head resting over a breathing hole and their arms by their sides. A pillow was placed under the subject's shins. During each stiffness test the subject was asked to hold their breath at the end of a normal expiration so that the breathing cycle was standardised during testing as PA stiffness changes during tidal breathing (Beaumont et al. 1991).

The subjects were tested by applying five loading cycles at a frequency of one cycle every 2 seconds (0.5 Hz) to the L4 spinous process. This rate is within the range suggested for use in manual assessment by mobilisation (Maitland et al. 2001). The maximum force was set at 150N and this level of force is commonly reached during manual assessment of the spine with PA forces (Harms et al. 1999). Immediately before testing on each day, subjects were pre-conditioned with four loading cycles. The stiffness testing device applied the PA force from a stationary start to simulate the process of manual assessment of PA stiffness in the clinic.

Subjects were tested on three occasions. Test 1 and Test 2 were conducted on the same day, with a five minute interval between the tests during which the subjects rested in the testing position on the plinth. Test 3 was undertaken on a different day that was greater than 24 hours and less than 8 days from Test 1. The mean time between Test1/Test2 and Test 3 was 5.2 days with a range of 2 - 8 days. Each stiffness test consisted of five loading cycles, therefore the total period of mechanical loading for each stiffness test lasted for 10 seconds.

In addition, steel (elastic) springs were tested to provide a comparison with the behaviour of the responses of the human subjects. The spring responses would be expected to show minimal variation with time and so would allow evaluation of the upper limit of variation due to the measurement device itself. Three elastic springs were used to provide 20 different test stiffnesses with stiffness coefficient K values ranging from 3.9 - 22.2 N/mm. The 20 different stiffnesses were produced by mounting three elastic springs in a device purpose built for providing test stiffnesses of known values (Maher and Adams 1995). The test stiffness produced by each

spring was altered by changing its position in the device. These elastic springs were tested with the same test – retest sequence as the human subjects. It is assumed that the stiffness of the springs did not change throughout the experiment.

Data Analysis

Stiffness coefficient K and displacement D30 were calculated from the force displacement curve as described in Chapter 1 (Figure 1.1) using custom written analysis software. Stiffness coefficient K was calculated as the gradient of a regression line fitted to the force displacement curve between 30 and 90N of force. This range of force was chosen because it was the linear part of the curve. Displacement D30 is the displacement in millimeters between 2 and 30N of force (the non linear part of the force displacement curve early in the loading range). For the within-test measures, stiffness coefficient K and displacement D30 were calculated for each cycle of each test. For the between test comparisons, the force displacement curves of cycles 2-5 were averaged to produce mean values for stiffness coefficient K and D30 for each test on the human subjects and the springs. Cycles two to five were chosen for this analysis to be consistent with previous studies that excluded the first cycle from analysis due to observations of different behaviour (Latimer et al. 1996a; Latimer et al. 1996c).

A repeated measures analysis of variance with contrasts (Winer 1971) was used to determine differences between cycles within a test and between the three tests. In addition, Intraclass Correlation Coefficients (ICC 2,1) were performed for tests 1, 2

and 3 to determine the reliability of the values between test occasions, 95% Confidence Intervals to indicate the variability of the measured values, % agreement to indicate the value where there is 90% agreement between tests (Shrout and Fleiss 1979; Fleiss 1986; Portney and Watkins 1993), and standard error of measurement to give an estimate of the precision of the measurement (SEM) (Domholdt 1993). ICC's were also used to examine the correlation between the stiffness coefficient K for the first cycle in a test compared with the average of the stiffness values of the subsequent four cycles.

RESULTS

Behaviour during five successive cycles

Stiffness coefficient K in human subjects was less stiff in the first loading cycle than successive cycles in a stiffness test of 5 cycles (ANOVA; $P<0.05$). There was no difference amongst the means of the remaining four cycles. Stiffness coefficient K for the elastic springs was also less in the first cycle than in successive cycles (ANOVA; $P<0.05$). The means (standard errors) for the five successive cycles are presented in Table 2.1 and shown graphically in Figure 2.2. The stiffness coefficient K for the first cycle in a test was highly correlated with the average of the subsequent four cycles for each of the test occasions (ICC= 0.85, 0.89 and 0.93 for tests 1, 2 and 3 respectively). When comparing ICC values for humans and elastic springs consideration should be given to the fact that there is a greater range of stiffness in the springs than the humans.

The D30 value was significantly less in the first cycle than the remaining four cycles in human subjects (ANOVA; $P<0.05$). The same behaviour was also observed for the D30 values of the elastic springs with the first cycle showing less displacement than the subsequent cycles (ANOVA; $P<0.05$). The means (standard errors) for the five successive cycles are detailed in Table 2.1 and shown graphically in Figure 2.3.

Table 2.1. Mean values (SE) for stiffness coefficient K and displacement value D30 for Test 1 during five successive loading cycles.

	Stiffness Coefficient K		***Displacement Value D30***	
Loading Cycle	People	Springs	People	Springs
Cycle 1	12.04 (0.67)	11.90 (1.25)	4.72 (0.37)	2.53 (0.34)
Cycle 2	13.15 (0.71)	11.97 (1.25)	5.83 (0.37)	2.61 (0.34)
Cycle 3	13.33 (0.78)	12.01 (1.26)	5.96 (0.38)	2.65 (0.34)
Cycle 4	13.24 (0.63)	11.98 (1.25)	6.12 (0.37)	2.63 (0.33)
Cycle 5	13.22 (0.80)	12.01 (1.26)	5.96 (0.38)	2.66 (0.34)

Table 2.2. Mean values (SE) for stiffness coefficient K and displacement D30 for the three tests.

	Stiffness Coefficient K (N/mm)		**Displacement D30 (mm)**	
Test	People	Springs	People	Springs
Test 1	13.18 (0.71)	12.01 (1.26)	5.97 (0.37)	2.64 (0.34)
Test 2	13.14 (0.63)	11.94 (1.23)	6.21 (0.44)	2.69 (0.31)
Test 3	13.21 (0.77)	11.99 (1.24)	5.60 (0.42)	2.67 (0.30)

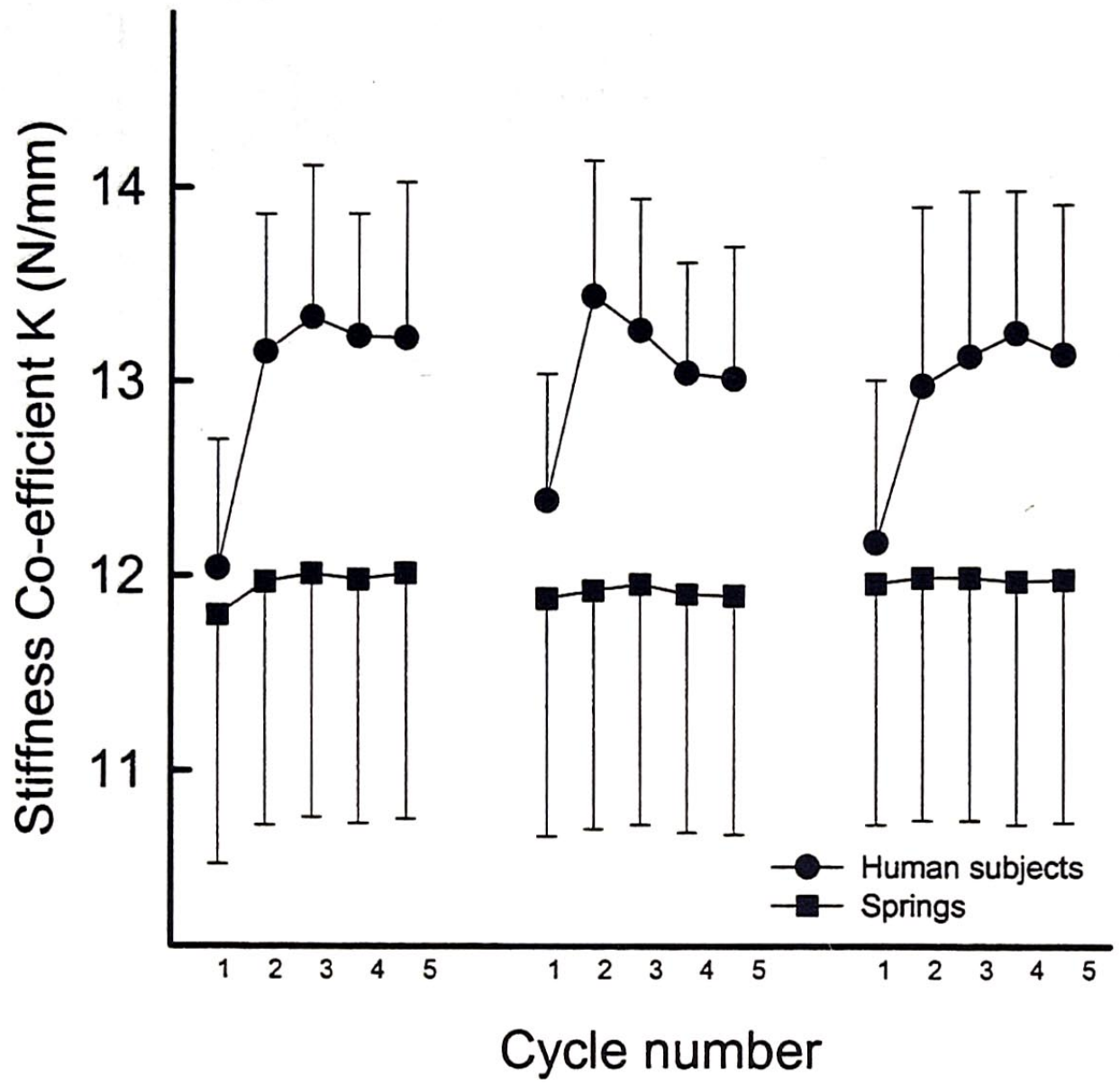

Figure 2.2. Means (SE) for human L4 and springs for Coefficient K during 5 consecutive loading cycles.

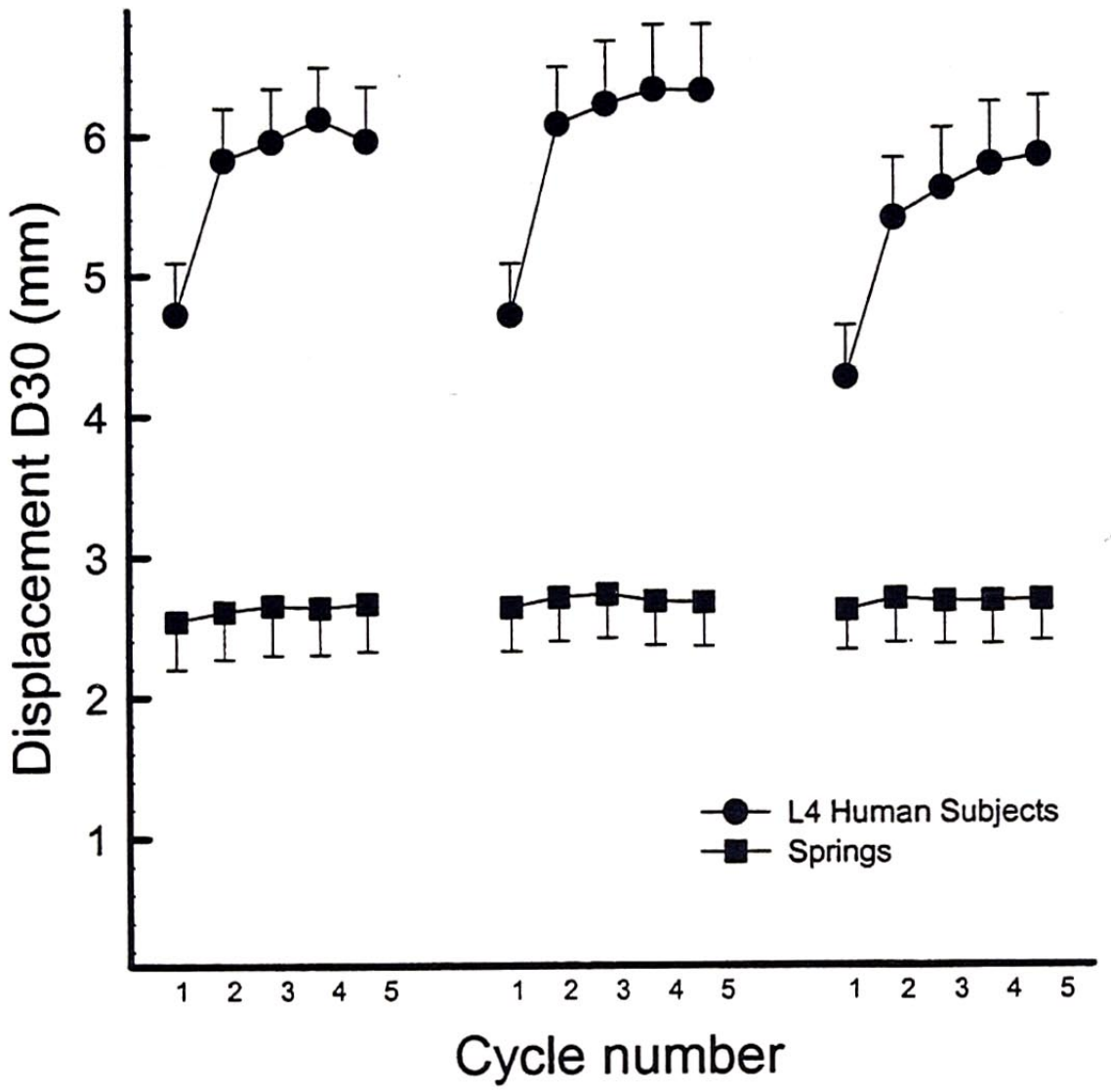

Figure 2.3. Means (SE) for human L4 and springs for Displacement D30 during 5 consecutive loading cycles for displacement caused by 30N of PA force.

Behaviour over three test occasions

There was no significant change in the stiffness coefficient K for either human subjects or springs between tests 1, 2 and 3 (Table 2.2). A comparison of the three test occasions for the human subjects resulted in an ICC (2,1) of 0.88, the standard error of the measurement of 1.03 N/mm and on 90% of occasions the test-retest difference was less than 2.0 N/mm. A comparison of the three test occasions for elastic spring stiffness resulted in an ICC (2,1) of 0.99, SEM of 0.55 N/mm and on 90 % of occasions the difference was less than 0.5 N/mm (Table 2.3).

The displacement value D30 for the human subjects demonstrated no change between test 1 and test 2 or test 1 and test 3 but there was a significant difference (ANOVA; $P<0.05$) between test 2 and test 3 (Table 2.2). For the human subjects, on the three test occasions the ICC was 0.85, the standard error of the measurement was 0.69mm and on 90% of occasions the test -retest difference was less than 1.5 mm. There was no difference in the D30 value between the three tests for the springs. The ICC was 0.99, SEM was 0.13 mm and on 90 % of occasions the difference was less than 0.5 mm (Table 2.4).

Table 2.3. Comparison between tests for Stiffness Coefficient K.

Inter-test Comparison	Standard Error of the Measurement		ICC		95% Confidence Interval		90% Agreement	
	People	*Springs*	*People*	*Springs*	*People*	*Springs*	*People*	*Springs*
Tests 1 and 2	0.57	0.54	0.96	0.99	0.90 - 0.99	0.99-0.99	1.1 N/mm	0.5 N/mm
Tests 1 and 3	1.2	0.54	0.85	0.99	0.64 - 0.94	0.99-0.99	4.0 N/mm	0.5 N/mm
Tests 2 and 3	1.2	0.54	0.83	0.99	0.59 - 0.93	0.99-0.99	3.0 N/mm	0.5 N/mm
Tests 1, 2 and 3	1.03	0.55	0.88	0.99	0.75-0.95	0.99-0.99	2.0 N/mm	0.5N/mm

Table 2.4. Comparison between tests for Displacement D30.

Inter-test Comparison	Standard Error of the Measurement		ICC		95% Confidence Interval		90% Agreement	
	People	*Springs*	*People*	*Springs*	*People*	*Springs*	*People*	*Springs*
Tests 1 and 2	0.59	0.15	0.88	0.99	0.72-0.95	0.99-0.99	1 mm	0.5 mm
Tests 1 and 3	0.64	0.15	0.86	0.99	0.64-0.95	0.89-0.99	1 mm	0.5 mm
Tests 2 and 3	0.81	0.15	0.81	0.99	0.44-0.93	0.90-0.99	1.5 mm	0.5 mm
Tests 1, 2 and 3	0.69	0.13	0.0.85	0.99	0.68-0.94	0.96-0.99	1.5 mm	0.5mm

DISCUSSION

The results of this study indicate that the linear portion of the force displacement curve, described by stiffness coefficient K, and non linear toe region, described by displacement D30, behave differently during the first loading cycle than in subsequent loading cycles of a stiffness test in normal subjects. In addition, stiffness coefficient K was constant over three test occasions, whereas displacement D30 was constant for the two tests on day one and between Test 1 on day 1 and Test 3 on day 2. However, there was a difference between the value for displacement D30 for Test 2 on day 1 and Test 3 on day 2.

Within five consecutive loading cycles

The stiffness coefficient K was significantly lower in the first cycle than subsequent cycles in a test involving five loading cycles, indicating that PA stiffness in the lumbar spine was less during this cycle and that subsequent cycles demonstrate increased stiffness. The displacement value D30 was significantly lower in the first cycle than subsequent cycles indicating that PA displacement increased with repeated loading suggesting increased mobility in a PA direction. These results suggest that there is increasing stiffness after the first PA force is applied and the linear portion is occurring later in range.

Our findings, which demonstrated a pattern of increasing stiffness and increasing displacement over five loading cycles, are consistent with other work that has examined loading of tissues in the lumbar region (Yahia et al. 1991; Lee and Evans 1992). A study using cadaveric specimens of the lumbar spine sectioned to isolate the interspinous / supraspinous ligament complex has indicated that there is increasing stiffness and increasing displacement with repeated loading (Yahia et al. 1991). Other work on living subjects using three cycles of loading in the lumbar region has also demonstrated similar behaviour (Lee and Evans 1992). In both studies there was a greater change in response between the first and second loading cycles than with subsequent cycles. A number of spinal soft connective tissues have shown this pattern of behaviour eg skin, interspinous and supraspinous ligaments (Twomey and Taylor 1982; Yahia et al. 1991; Lee and Evans 1992), intervertebral (IV) discs (Nachemson et al. 1979) and whole IV joints (Panjabi et al. 1977). A study examining the role of various spinal soft tissue structures in resisting PA loads applied to functional spinal units demonstrated that the IV disc was the structure that contributed the most resistance (Lee 1995), although the role of the skin and subcutaneous tissue was not examined. There is evidence that subcutaneous soft tissue may play a role in lumbar PA stiffness as it has been demonstrated that overweight and obese individuals are less stiff than normal or underweight individuals (Viner et al. 1997). It is most likely that all of the structures mentioned above, including the skin and subcutaneous soft tissue and abdominal tissues, contributed to our results. Our results suggest that increases in stiffness with repeated loading reflect the normal mechanical behaviour of lumbar soft tissues.

A set of elastic springs tested in the same way as the human subjects demonstrated that K and D30 were significantly less in the first cycle compared to the other four cycles. The difference between the first and subsequent cycles for both stiffness coefficient K and D30 of the springs was very small. The mean increase in K from cycle 1 to cycle 2 was 0.08 N/mm in springs compared with 1.11 N/mm in human subjects. Displacement D30 increased by 0.04 mm in springs and 1.09 mm in human subjects. These small differences between tests of springs indicate that the effects observed in the human subjects can be largely attributed to the mechanisms within the human subject rather than to the testing system itself.

Previous studies that have used similar methodologies for quantifying lumbar PA stiffness have elected to discard the results of the first cycle interpreting it as transient behaviour on commencement of testing (Latimer et al. 1996a; Latimer et al. 1996c). Our results suggest that the difference in behaviour of the first loading cycle appears to be a normal response to PA loading. Therefore, the procedure of ignoring the first cycle in analysis of stiffness for research purposes is justified. However, the clinical implications of the different behaviour of the first loading cycle for making judgements of PA stiffness are not clear. Our results demonstrate that the first loading cycle is different from, but highly correlated with the average of the four subsequent cycles (ICC=0.85, 0.89 and 0.96 for tests 1, 2 and 3 respectively). It has also been shown that the use of three loading cycles is optimum when making stiffness judgements of a movement with constant stiffness (Macfadyen et al. 1998). The different response in cycle 1 may make it more difficult for clinicians to evaluate stiffness. In addition, some researchers working in this area only apply a single loading force to their subjects (Edmondston et al. 1998). As the first cycle

behaves differently to subsequent cycles, comparison of stiffness values between studies with differing numbers of loading cycles should be avoided. It would be expected that studies using only one loading cycle would report lower stiffness values than studies discarding the first loading cycle.

Behaviour over three test occasions

Our results demonstrate that stiffness coefficient K was consistent over three different test occasions with the same group of subjects. In a previous study stiffness coefficient K was consistent within a single test session in subjects with pain (for K, ICC= 0.96, 90% agreement 1.8 N/mm; for D30, ICC =0.89, 90% agreement 1.1mm) (Latimer et al. 1996c). In our study, stiffness coefficient K and displacement D30 were consistent over the three test occasions (for K ICC=0.96, 90% agreement of 1.1 N/mm and for D30 ICC=0.88, 90% agreement of 1mm). In another study, there was no significant difference between tests on different days in subjects without pain (no ICC data included) (Latimer et al. 1996c). Also, testing on similar equipment to that used in this study demonstrated that stiffness was consistent over 2 different days ICC (2,1) was 0.88 (Lee and Svensson 1990) which is consistent with our finding of ICC (2,1) of 0.85.

As stiffness coefficient K was consistent over three test occasions (including a time interval of 8 days) in asymptomatic subjects, PA stiffness in subjects without LBP would not be expected to change over several days. However, changes in PA

stiffness do occur in people with LBP (Latimer et al. 1996c). The standard error of the measurement for coefficient K was 1.03 N/mm which suggests that changes in stiffness would need to be greater than this to be attributed to factors other than chance variations over time. For the subjects in this study the mean stiffness was 13.2 N/mm. The SEM (1.03 N/mm) is equal to 8% of the mean stiffness. Most physiotherapists are able to detect changes in stiffness of elastic springs in the order of 11% and some even lower (Maher and Adams 1995; Nicholson et al. 1997), however it is likely that this value would be greater for human subjects. The changes in PA stiffness that we observed are at the lower end of the detectable range for elastic springs. The tissues of the spine have viscoelastic rather than purely elastic properties. Therefore, the ability to detect stiffness of humans is likely to be less accurate, and a change of stiffness of 1.03 N/mm may not be detectable in the clinical setting.

Displacement early in range (D30) was consistent between Test 1 and Test 2 during the first test session and also between Test 1 in the first session and Test 3 in second session. There was, however, a difference between Test 2 in the first session and Test 3 in the second occasion. This difference could be due to a trend for the mean to increase slightly between Test 1 and Test 2 (Figure 2.3). Previous studies of isolated tissues suggest that small changes will tend to keep occurring even after many cycles (Yahia et al. 1991; Asano et al. 1992). Between Tests 1 and 2 the displacement returned to the pre test value, however, in our study the value on the second day (Test 3) was less than for Test 1 in the first session. Although there was no significant difference between Test 1 and Test 2 or Test 1 and Test 3 there was a significant difference between Test 2 and Test 3.

Stiffness coefficient K and displacement D30 are the two measures used in this study to describe the lumbar responses to PA loading. These two measures are derived from the force displacement relationship when PA forces are applied to the spine and are calculated from the loading portion of the curve below 90N. This means that K and D30 are not able to describe the behaviour of the total response to PA loading which includes both the loading and unloading curves. Further research is being undertaken to investigate all components of the loading and unloading curves (i.e. the linear and non linear, elastic and viscous properties of the force displacement relationship) (Nicholson et al. 2001).

Clinical Implications

Although the measurable changes that we observed in the response of lumbar PA stiffness to repeated loading have relevance to testing with mechanical devices, they are small compared with the stiffness differences that are likely to be detectable with manual examination. Therefore, clinical observations of marked changes in stiffness are probably not due to the effects of repeated loading on normal soft tissue. It is possible that the behaviour of soft tissues in the lumbar spine is different in the presence of pain or damage. Further studies using subjects with LBP could clarify the possible source of stiffness changes observed in the clinic.

CONCLUSION

The lumbar PA responses to repeated PA loading (ie of 5 loading cycles) demonstrates a time dependent behaviour. The findings of this study are consistent with those of other studies investigating the biomechanical properties of lumbar spine soft tissues. The lumbar PA response demonstrating an increase in stiffness to 90 N and an increase in displacement to the linear portion of the force displacement curve between the first and subsequent cycles can be considered a normal response to PA loading. The significance of the behaviour of the first cycle is not clear and is possibly best ignored when making judgements about stiffness. However, when comparing results of PA responses to other studies it is important to ensure consistency between the cycles used for analysis. The responses observed in this study are short lived and the tissues return to their pre testing state in less than five minutes.

CHAPTER THREE

THE RELATIONSHIP BETWEEN MAXIMAL AND SUBMAXIMAL ACTIVATION OF THE LUMBAR EXTENSOR MUSCLES AND LUMBAR POSTERO-ANTERIOR STIFFNESS

This study was supported in part by a University of Sydney Mechanical Equipment Grant.

ABSTRACT

Patients with low back pain have increased lumbar PA stiffness. It is believed that increased activation of the lumbar extensors could contribute to this stiffness. Increased muscle activation which may occur in response to an applied PA force is thought to equate to only a small percentage of maximal voluntary contraction. Maximal voluntary contraction of the lumbar extensors increases lumbar PA stiffness, however the effect of small amounts of voluntary contraction on lumbar PA stiffness is not known. This study investigated the effect of varying amounts of voluntary isometric muscle activity of the lumbar extensors on lumbar postero-anterior (PA) stiffness.

Twenty subjects aged between 24 and 45, without low back pain, participated in the study. Subjects were asked to perform a maximal voluntary isometric contraction (MVC) of their lumbar extensor muscles by exerting a force against a steel plate located over their T4 spinous process with their pelvis fixed. They were then asked to perform contractions equivalent to 0%, 10%, 30%, 50%, and 100% of MVC. PA stiffness at L4 was measured during these contractions.

The results of this study indicated a significant difference in PA stiffness between all levels of muscle activation. Voluntary contraction of the lumbar extensor muscles will result in an increase in lumbar PA stiffness even at low levels of activation.

INTRODUCTION

The relationship between LBP, lumbar muscle activity and lumbar PA stiffness is not well understood. The assumption that these variables are related underpins the clinical reasoning process, which is used by physiotherapists to make treatment decisions. This relationship was investigated in a pilot study undertaken by Shirley and Lee (1993) designed to examine whether people with LBP had different lumbar muscle activity and PA stiffness when a PA force was applied to their lumbar spine than people without pain. It was observed that some subjects with low back pain showed relatively high PA stiffness and demonstrated different patterns of lumbar muscle activity in response to the application of a PA force than similar subjects without low back pain, thus indicating a possible link between muscle activity and PA stiffness.

The aim of manual stiffness assessment of the lumbar spine in a person with LBP is to reproduce pain (Maitland et al. 2001). It has been suggested that reflex muscle activity in the low back muscles occurs in response to pain (Mennell 1960; Travell and Simons 1983). Therefore stiffness assessment that provokes a pain response could result in the reflex activation of lumbar extensor muscles. Patients with LBP have greater PA stiffness during an episode of LBP (Latimer et al. 1996c). It is proposed that muscle activity could be responsible for the clinically observed increases in lumbar PA stiffness in subjects with LBP. The relationship between muscle activity that occurs in response to the application of PA forces and lumbar PA stiffness has not yet been fully explored in either patients with LBP or people without LBP.

Maximal voluntary contraction (MVC) of the back extensors can substantially alter PA stiffness at L3 in normal subjects (Lee et al. 1993). Maximal back extensor activation has been found to produce a mean increase in PA stiffness of 350% (Lee et al. 1993). In the study conducted by Shirley and Lee (1993) using subjects with LBP, it was estimated that the amount of muscle activity produced in subjects with LBP during the application of a PA force would be equivalent to around 5%-10% of MVC. Although it has been established that a maximal voluntary contraction of the back extensors will substantially increase PA stiffness (Lee et al. 1993), it is not known whether small amounts of muscle activity could be responsible for the increased PA stiffness perceived by physiotherapists when they examine patients with LBP. Our hypothesis is that small amounts of voluntary muscle activity will result in an increase in lumbar PA stiffness. The aim of our study, therefore, was to determine the effect of different levels of voluntary back muscle activity on lumbar postero-anterior (PA) stiffness in normal subjects.

METHODS

Experimental Design

The design of the study involved measurement of lumbar PA stiffness at L4 under five conditions of muscle activation. The conditions were 0%, 10%, 30% 50% and 100% of the force produced during a maximal voluntary contraction (MVC) of the subjects' back muscles. The focus on lower percentages was chosen because we were particularly interested in whether small amounts of muscle activity were capable of altering PA stiffness as the amounts of spontaneous muscle activity observed in subjects with low back pain in response to PA forces are likely to equate

to small percentages of MVC. The subjects performed these levels of activation in a random order. For each level of activation the surface electromyograms (EMG) of the lumbar extensors were recorded, in addition to the force generated by the subject, while the lumbar PA stiffness was measured. The L4 level was chosen as it is one which is commonly found to be stiff and painful during assessment with PA pressures (Maitland 1986).

Subjects

Twenty asymptomatic subjects were included in this study. Subjects without low back pain were selected so that a maximal voluntary contraction could be tested. Subjects with low back may not be able to perform a maximal voluntary contraction without exacerbation of their symptoms. Therefore, it would be difficult to ascertain whether a maximal contraction was achieved and quantification of smaller contractions would be difficult.

The subjects were recruited from the population of staff and students at the Faculty of Health Sciences of the University of Sydney. Subjects were included if they were not currently experiencing low back pain, had no history of low back pain in the last six months and were not suffering from high blood pressure (diastolic blood pressure below 90 mmHg). The study was approved by the Human Ethics Committee of the University of Sydney. Informed consent was obtained before subjects were admitted to the study. There were sixteen females and four males who were aged between 26 and 45 with a mean age of 34.

Equipment

PA stiffness in the lumbar spine was measured using the Spinal Physiotherapy Simulator (SPS) (Lee and Svensson 1990). For the purpose of this study PA stiffness is defined as the gradient of the force displacement curve between 20 and 100 Newtons. Stiffness was calculated using analysis software specifically written for this purpose. The SPS is a device, which has been designed to assess PA stiffness by applying a predetermined maximum force to the spinous process of a lumbar vertebra. The SPS measures the force applied and the resulting displacement of the skin surface over the spinous process. The force applied and the resulting displacement can then be used by a custom designed computer analysis program to calculate stiffness. The SPS has demonstrated good test-retest reliability (ICC=0.88) for measuring PA stiffness on human subjects at L3 (Lee and Svensson 1990). The SPS has demonstrated satisfactory accuracy for measuring stiffness (within 1% of true value) of an elastic beam (Lee and Svensson 1990). In order to measure PA stiffness the indentor of the SPS was positioned over the L4 spinous process (Figure 3.1). The indentor is the part of the device that makes contact with the skin over the spinous process in a similar way to the physiotherapists hand when manually applying a PA pressure. Above the indentor is a load cell (XTRAN S1W 250N) that measures the applied force and there are two linear potentiometers (Tsushin Kogyo Co. Ltd and Penny and Giles) that measure the displacement of the skin surface. A motor drives the indentor up and down in a rhythmical oscillating manner similar to the way a physiotherapist applies a mobilisation technique. The indentor was angled at 4.5° to vertical in a caudal direction (Stagnara et al. 1982). Data were collected via a personal computer using an A/D converter (Data Translation DT2801A) with a

sampling rate of 100 samples-per second for 30 seconds. To ensure that activation of the lumbar extensor muscles was isometric subjects were prevented from extending their lumbar spine. Extension of the lumbar spine was prevented by restraining the subject at the level of T4 and the pelvis. The subject was restrained at T4 by a using padded steel plate positioned over T4 and connected via a steel frame to the table. The pelvis was restrained by a belt that was placed around the pelvis and the table. A study with a different protocol involving measurement of isometric lumbar extension strength by restraining the pelvis and the upper thoracic spine has demonstrated good reliability (r=0.8) for testing isometric strength when the subject has zero degrees of extension (Graves et al. 1990).

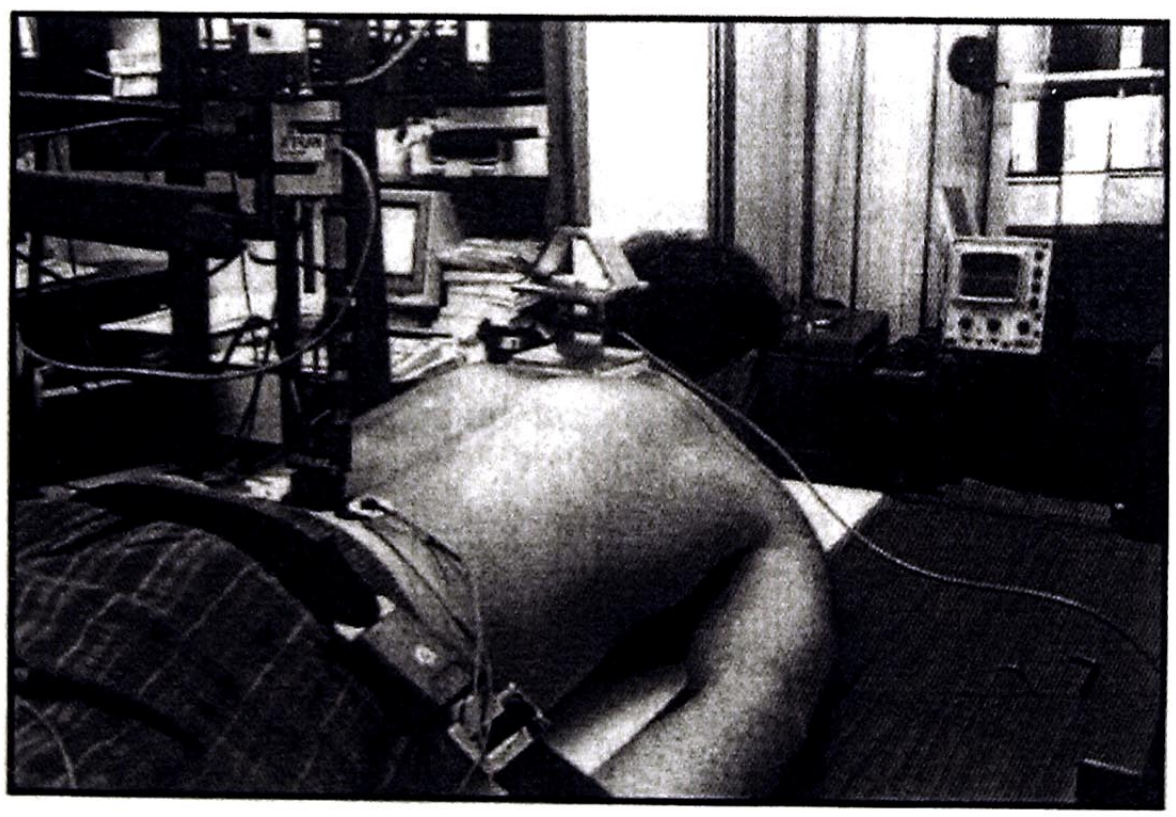

Figure 3.1. Testing apparatus. The subject is lying on the testing table with the padded steel plate positioned over T4, the indenter of the SPS positioned over L4 and the SEMG electrodes in place. The oscilloscope used to provide visual feedback is visible at the right side of the picture.

Procedure

Following the initial screening subjects lay prone on the testing apparatus and the L4 and T4 spinous processes were palpated and marked. The padded steel plate was positioned in contact with the skin over the T4 spinous process. The padding deformed only slightly under load, thus ensuring a near- isometric activation. A load cell (XTRAN S1W 250N) attached above the steel plate recorded the force generated by activation of the back extensors. The output of the load cell was displayed on an oscilloscope and recorded on computer. In the prone position subjects were first asked to produce a maximal isometric voluntary contraction of their back extensors and the force generated was recorded. Values of 10%, 30%, 50% and 100% of MVC were then calculated from this force. The subjects were then asked to push against the plate with forces that were equal to these percentages of their maximal voluntary contraction. An oscilloscope was provided to give feedback of the required level of force while the subject was attempting to maintain this level of force. A line was marked on the oscilloscope and the subject was asked to push so that the force signal just reached the target line. Subjects were also required to lie at rest without any activation of their back extensors for a period of data collection and this was regarded as a 0% maximal voluntary contraction. Subjects were asked to hold each contraction for a period of ten seconds and were given a practice attempt for the MVC at each required level of force.

During testing with the SPS subjects lay prone on the table of the testing apparatus with their arms by their sides and their head resting on a forehead support. A belt was placed around their pelvis and strapped to the table to provide stability.

Immediately before testing subjects were pre-conditioned with four loading cycles. Subjects were asked hold their breath at the end of a normal expiration while maintaining an isometric contraction for the duration of five cycles of force application, which was for a period of 10 seconds. The five loading cycles were applied at a frequency of 0.5 Hz, which was used to maintain reliability and to allow direct comparison with previous studies using similar methodology (Shirley and Lee 1993; Latimer et al. 1996a; Latimer et al. 1996c). Also, it is within the hypothesised optimum number of cycles used by physiotherapists in manual assessment of PA stiffness (Maitland 1986). The force was applied to a maximum of 150N which is within the range of forces used clinically by physiotherapists during manual assessment of the spine (Harms et al. 1999).

For each target % MVC the mean force displacement curve was calculated by averaging the middle three loading cycles. These cycles were used because this period of data collection was where the T4 force was most stable. A regression line was fitted to the linear portion of the mean curve between 20 and 100N. Force/displacement curves are usually linear within this range (Lee and Svensson 1990). The PA stiffness value (coefficient K) is the slope of the regression line fitted to the force/displacement curve between 20 and 100N and was calculated for each level of % MVC.

Surface EMG of the muscles adjacent to the lumbar spine were measured during stiffness testing at all levels of muscle activation. Two channels of EMG were used, recording from the left and right sides. Two self adhesive Medi-Trace pellet electrodes (Graphic Controls Corporation) were attached 4cm lateral to the L4

spinous process on each side with an inter-electrode distance of 2.5cm. The muscles underlying this region of the spine are the lumbar erector spinae (Bogduk 1997). A reference electrode was placed over the sacrum. Similar methods of electrode placement have been used to record activity of the lumbar paraspinal muscles (Sihvonen et al. 1991; Lavender et al. 1994). The surface EMG signals initially underwent analogue processing in which the raw surface EMG was filtered (bandwidth 8 Hz to 500 Hz), then amplified (Medelec AA6 Mk II, Common Mode Rejection Ratio 10000:1, input impedance 5000Ω) then the mean value was obtained (Medelec I6) using a 50 ms time constant. The processing described above all occurred with analogue signals before they were converted from analogue to digital (Data Translator DT2801A, Data Translation, Inc) and stored in the computer. After analogue to digital conversion the signal was termed the initial digital surface EMG.

Digital processing of the initial digital surface EMG signal was subsequently carried out using Sigma Plot (Jandel Corporation) software. After importing the initial digital surface EMG into Sigma Plot (Jandel Corporation) it was first processed by performing a running average with a time window of 2 seconds. The running average of the initial digital signal was then used to calculate the activation surface EMG. The activation surface EMG is a mean value for the time interval that corresponded to the subject's activation of their back extensors during the measurement of PA stiffness (i.e. the running average surface EMG was averaged over the time that the spinous process was being loaded from 20 - 100N). The activation surface EMG was calculated for each target %MVC. In order to compare muscle activity between subjects, the activation surface EMG was normalised by expressing the level of activity achieved during each target level as a percentage of

the MVC. To normalise the data the lowest surface EMG value recorded from the running average of the initial digital surface EMG signal for each test was first identified and subtracted from the activation surface EMG at each target % MVC. This procedure was performed so that the surface EMG value used in data analysis reflected the amount of muscle activity resulting from voluntary activation of the muscle. This value was termed the corrected activation surface EMG. Each corrected activation surface EMG was then expressed as a percentage of the surface EMG recorded during the maximal voluntary contraction and was called the normalized surface EMG. A similar method for normalization of surface EMG signals has been used in other work for analysis of surface EMG of trunk muscle activation (Lavender et al. 1994). A normalized surface EMG was calculated from the data collected from both the right and left sides. The final figure used in data analysis was an average of the normalized surface EMG recorded from right and left lumbar extensors. This value is described as % Maximum surface EMG (Table 3.1).

The forces measured by the load cell positioned over T4 were expressed as percentages of the force achieved during the maximal voluntary contraction. These forces at the target % MVC were calculated by averaging the force over the time corresponding to the calculation of stiffness and surface EMG and subtracting the minimal value for the period of data collection. A minimal value was calculated because positioning the padded steel plate over the T4 spinous process caused the load cell to register a force in some cases. The force due to positioning of the plate was termed the minimal value. The minimal value was subtracted from the average T4 force so the resulting value, which was used to describe the force at target %MVC, would reflect the actual force exerted against the plate due to muscle

activity. The minimal value was obtained by averaging data collected over 0.5 seconds in a part of the test where the lowest force was recorded which, ideally, was before activation of the back extensors occurred. If this was not possible, data averaged over 0.5 seconds from lowest force recorded during the period of data collection was used.

Data Analysis

The stiffness data for each target level of %MVC were analysed using a Friedman's one way analysis of variance for a repeated measures design (Sigma Stat, Jandel Corporation) to examine for differences in stiffness among the different levels of muscle activation. A non parametric test was used as the data were not normally distributed. A post hoc (Student Newman-Keuls) (Sigma Stat, Jandel Corporation) analysis was performed to isolate the levels that differed from the others.

RESULTS

When PA stiffness of the spine was measured while subjects performed activation of their lumbar extensors there was a significant increase in mean PA stiffness values at each target % MVC (Table 3.1) ($P<0.0001$). In addition, there was a significant increase in median stiffness values between all levels of activation ($P<0.05$) indicated by a Student Neuman Keuls post hoc analysis.

A close linear relationship ($r^2=.99$) was observed between the mean % maximum T4 force (Table 3.1) produced by the back extensors and mean % maximum surface

EMG values. The regression equation for these data was (% Max T4 Force) = 0.95 (% Max surface EMG) + 10.38.

The mean values of PA stiffness, % maximum surface EMG and % maximum T4 force at the different levels of back muscle activation are given in Table 3.1. The mean values and standard deviations for PA stiffness are plotted against mean % maximal surface EMG and are illustrated in Figure 3.2. A linear regression was performed to fit a line of best fit to the mean values. The regression showed a linear relationship between % maximal surface EMG and PA stiffness (r^2=0.92). The equation of the regression line for these data was Lumbar PA Stiffness = 0.15 (Target % surface EMG) + 14.7.

Table 3.1. Mean (SD) of PA stiffness, T4 force and surface electromyogram (SEMG) values at the different levels of maximal voluntary contraction (MVC).

Target %MVC	PA Stiffness N/mm	% Increase in mean stiffness from resting mean value	% Maximum T4 Force	% Maximum surface EMG
Rest	14.8. (5.2)		0.48 (1.6)	6.9 (8.9)
10% MVC	17.5 (4.0)	11.8	11.8 (4.3)	25.4 (14.3)
30% MVC	21.9 (5.4)	41.2	31.7 (5.4)	44.2 (16.3)
50% MVC	26.3 (8.8)	75.2	50.3 (5.8)	59.6 (22.9)
100% MVC	29.6 (7.3)	91.5	92.3 (11.8)	110.1 (45.8)

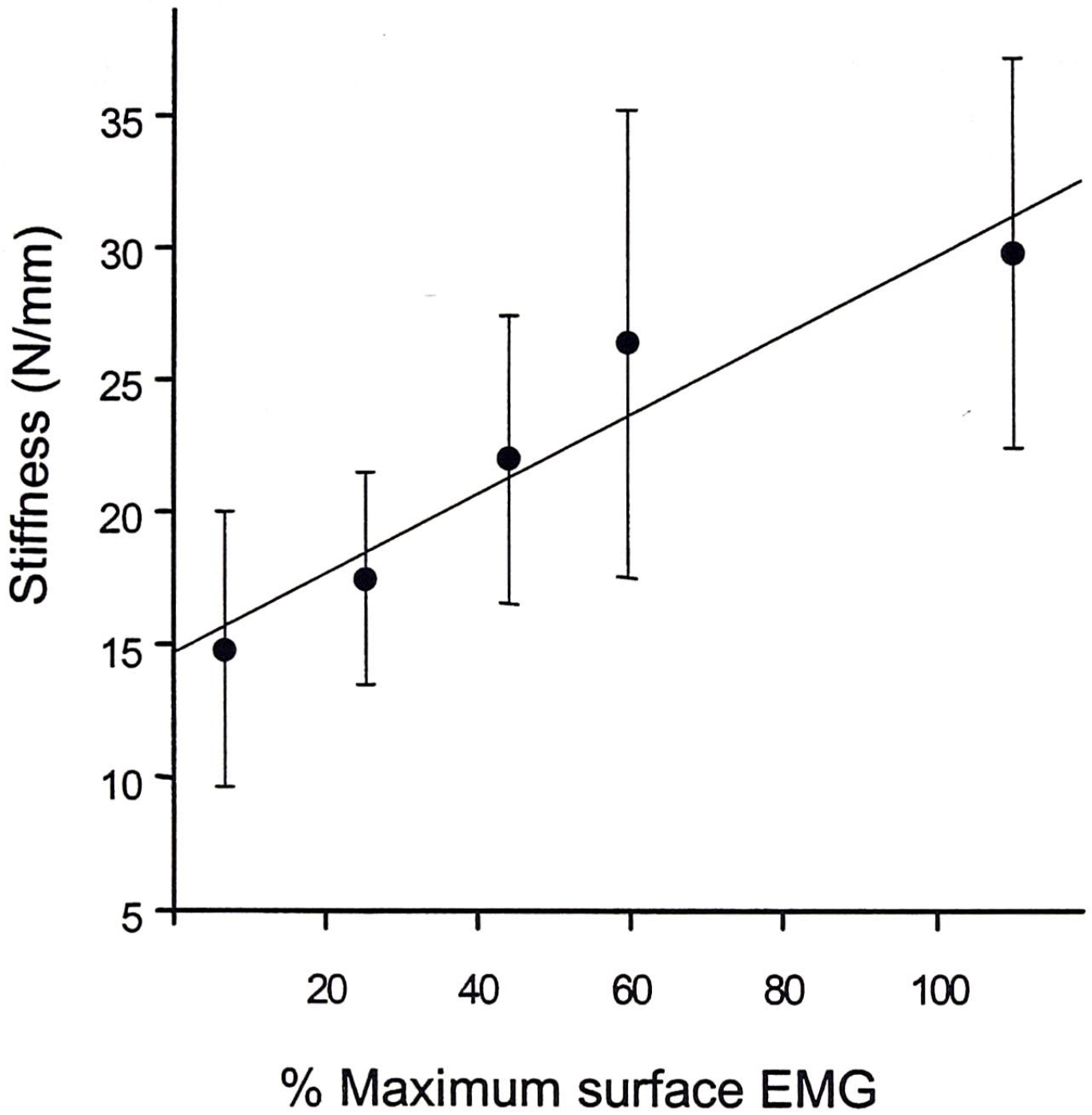

Figure 3.2. Postero anterior (PA) stiffness versus % maximum surface EMG. This graph shows the mean PA stiffness and standard deviations at each level of mean percentage of maximum surface EMG activity. A least-squares regression line fitted to the mean data is shown.

DISCUSSION

The results of this study indicate that even small amounts of back muscle activity can increase lumbar PA stiffness. The finding that voluntary submaximal isometric activation of the trunk extensor muscles increases lumbar PA stiffness is of clinical interest because clinicians frequently report increased activity in this muscle group during their manual examination of a patient (Grieve 1984; Maitland 1986). It has been previously demonstrated that a MVC of the back extensors can significantly alter lumbar PA stiffness (Lee et al. 1993), and the results of our study support that finding.

In a previous study by Lee et al. (1993) the mean increase in PA stiffness under MVC was 350%, whereas the mean increase in stiffness in the current study at MVC was 92%. There are a number of reasons why this difference may have occurred. In our study stiffness at L4 was measured whereas Lee et al. (1993) measured L3. Spinal PA stiffness varies depending on the vertebral level being tested (Lee and Liversidge 1994). The effect of muscle activation may also be dependent on vertebral level due to the ability of muscles such as multifidus to exert control very precisely on a specific vertebral level (Aspden 1992). Strength of subjects is another factor that may have contributed to the difference in results between our study and that of Lee et al. (1993). In the study by Lee et al. (1993) there was a higher ratio of males to females than in our study, where the majority of subjects were female. It is possible that the subjects used by Lee et al. (1993) were stronger and produced higher absolute T4 forces. The mean PA stiffness value in the study by Lee et al. (1993) at MVC was 50.9 N/mm which was boosted by three subjects who had

particularly high stiffness values, whereas the mean stiffness at MVC in our study was 29.6 N/mm.

The T4 force was recorded to provide visual feedback to the subjects while activating their back muscles. The close linear relationship between mean % maximum T4 force and mean % maximum surface EMG indicates that analysis of surface EMG is appropriate to reflect the target forces the subject was asked to match with feedback from the oscilloscope. Surface EMG was not used to give visual feedback because it is difficult to determine the various percentages of MVC from a raw signal on an oscilloscope, whereas, % maximum force was easy to determine. Surface EMG was used in the analysis of muscle activation at the various target % MVC because the aim of this study was to determine the effect of activation of the lumbar extensor muscles on lumbar PA stiffness.

We believe that the surface EMG signal primarily reflects activity of the muscles immediately underlying the electrodes (ie erector spinae) (Bogduk 1997). It is possible that the electrodes recorded activity of other muscles that could be recruited while the subject was attempting to activate their back extensors eg deeper or more medial extensors (multifidus) at that level or erector spinae at other levels. The erector spinae are likely to be the main extensors at this level and even if activity of other muscles is recorded, their activity is likely to be closely related to the lumbar erector spinae (Bogduk 1997).

During a voluntary contraction of the back extensors it is possible that a physiological extension of the lumbar spine could occur. Physiological extension

resulting from raising the head of a plinth 24° results in a 12.4% increase in lumbar PA stiffness (Edmondston et al. 1998). In the current study increases in PA stiffness ranged from 12% at 10%MVC to 92% at 100%MVC. Therefore, increases in PA stiffness due to the spine moving into a small amount of extension would not explain increases that we observed. The subjects were positioned with their arms by their sides with their head in a neutral position. The thoracic spine was restricted by a plate at T4 and a belt was also placed around their pelvis, strapping the pelvis to the table for stability. Although some degree of spinal extension may have occurred in this position, it is our opinion that the effect of the increased extension would have been minimal compared to the effect of muscle activity.

Contraction of spinal extensor muscles could increase PA stiffness by their action at individual vertebral levels. Activation of these muscles causes the vertebrae to rotate posteriorly (that is, in the direction of extension) in the sagittal plane (Bogduk 1997) and increased resistance to anterior shear (Potvin et al. 1991) and consequently, probably increased PA stiffness of the vertebral column. Thus, when a PA force is applied during muscle activation it is likely that the resistance to anterior vertebral movement will result in increased PA stiffness, as shown in our study.

A degree of variance between subjects in the PA stiffness values was observed at each target % MVC, however, all subjects in this study demonstrated an increase in stiffness with increasing muscle activity. In the resting state the mean stiffness at L4 was 14.8 N/mm $\pm$ 5.2 (mean $\pm$ SD). These values are similar to those reported in other studies that have used this method of testing for PA stiffness of the lumbar spine. Lee et al. (1993) reported stiffness at L4 to be 17.5 N/mm and Latimer et al.

(1996a) reported values of 14.84 N/mm ± 3.46 in pain free subjects, however, the vertebral level was not specified. Therefore, the PA stiffness values of this study for the resting state are consistent with other studies. The PA stiffness at MVC in our study was 29.6 N/mm ± 7.3 compared with 50.9 N/mm ± 23.4 observed by Lee et al. (1993). Therefore the variance at MVC in our study was less than that reported by Lee et al. (1993). In our study the standard deviation gradually increased as stiffness increased, so the larger variance observed in the study by Lee et al. (1993) is possibly a function of the larger stiffness values obtained and the fact that three subjects' stiffness values were substantially greater than the rest of the sample.

It is important to consider whether the magnitude of increases in PA stiffness observed in our study is clinically relevant. That is, could the changes in PA stiffness resulting from levels of muscle activation that might be observed in subjects with low back pain be detectable by manual palpation procedures? Our results indicate that a 10% increase in muscle activity will lead to a mean 11.8 % increase in lumbar PA stiffness (Table 3.1). The mean threshold for stiffness discrimination when assessing linear elastic springs is 11% for physiotherapists (Maher and Adams 1995), although this value may be higher for more complex force displacement relations (Latimer et al. 1998). In other words, physiotherapists can detect changes in stiffness as low as 11%. The increases in stiffness resulting from large increases in muscle activity eg. 30% MVC, 50% MVC and 100% MVC, should be easy to detect manually as they are well above the stiffness discrimination threshold for most physiotherapists. Some physiotherapists have an extremely low stiffness discrimination threshold with elastic springs (Maher and Adams 1995) and may be able to manually detect the increases in stiffness resulting from muscle activity as

low as 10% MVC. It is also possible that the increases in muscle activity (~5% - 10% MVC) observed in some patients with low back pain by Shirley and Lee (1993) could result in increases in lumbar PA stiffness which some physiotherapists would be able to detect during manual stiffness assessment. Further investigation is needed to better establish the level of lumbar extensor muscle activity that occurs in patients with low back pain in response to applied PA forces, and whether the pattern and extent of muscle activation in a voluntary contraction is similar to the activation in patients with low back pain.

This study investigated the ability of a voluntary isometric contraction of the lumbar extensor muscles to alter PA stiffness and has determined that there is a linear relationship between voluntary activation of these muscles and lumbar PA stiffness. It has also been reported that subjects with low back pain may have increased activation of their lumbar muscles in response to the application of a PA force (Shirley and Lee 1993). There is not necessarily any parallel in the pattern and extent of response between the voluntary activity produced in the present study and any muscle activity that may occur in response to an applied PA force in a patient population. Therefore, the degree to which our results can be applied to patients with low back pain has not yet been established. One possible difference is that the activation occurring in response to an applied PA force in the patient with low back pain will be more localised than during voluntary activation of the trunk extensors.

Conclusions

Voluntary activity of the back extensor muscles resulted in an increase in lumbar PA stiffness. This increase in lumbar PA stiffness was observed at all levels of activation that we examined. We think it is possible that the levels of muscle activity achieved with 10% to 30% MVC are similar to the amount of muscle activity that is thought to occur in response to the application of a PA force to the lumbar spine in patients with low back pain. I suggest, therefore, that a relationship may exist between lumbar muscle activity and increased PA stiffness as measured by the manual assessment of PA forces. Of potential clinical importance is the fact that even low levels of activity produce an increase in PA stiffness. That is, clinicians should be aware that increased PA stiffness may not be solely due to passive properties of the spine, but rather may be due to increases in muscle activity.

CHAPTER FOUR

LUMBAR STIFFNESS, LUMBAR EXTENSOR MUSCLE ACTIVITY AND LOW BACK PAIN

This study was supported in part by a George Burniston – Cumberland Foundation fellowship and a Cumberland Grant.

ABSTRACT

Physiotherapists who practice manual therapy commonly use spinal mobilisation in the management of low back pain. One common mobilisation technique is the application of a force to the lumbar spine in a postero-anterior (PA) direction. It is widely believed by physiotherapists that people with low back pain may have increased lumbar spine stiffness and muscle activity related to their LBP. This muscle activity may be present as a result of the low back pain or may be a response to the application of a PA force to the back. As the relationships between pain, stiffness and muscle activity have not been confirmed, the aim of this study was to determine whether such a relationship exists.

In this study a group of subjects with pain had their stiffness measured with a mechanical device while they had acute LBP and again, once their pain had resolved. A group of subjects without LBP pain matched for age and gender were tested at similar time intervals. Surface EMG was used to record activity of the muscles adjacent to the spine at L4 during stiffness testing.

The results of this study indicate that people with higher pain have higher PA stiffness suggesting a relationship between low back pain and PA stiffness. There is also an association between acute low back pain, activity of the lumbar erector spinae, and PA stiffness in response to the application of PA forces at rest.

INTRODUCTION

Manual examination with PA pressure is performed to determine which intervertebral level is related to the patient's symptoms and whether there is any abnormality in the quality or quantity of movement present (Magarey 1985; Jull 1986). Physiotherapists commonly describe movement abnormalities in vertebral motion in terms of the movement stiffness, which they perceive when applying the PA pressure. Increased muscle activity, which may or may not be related to variations in stiffness, is also noted during the assessment of the spine.

It is commonly assumed that a relationship exists between pain, stiffness and increased muscle activity and that treatment leading to a change in one will affect the others. PA stiffness is increased during an episode of LBP and it is proposed that increased muscle activity could be responsible for the increase in PA stiffness (Latimer 1995). Recently, erector spinae activity has been demonstrated in response to applied PA force in asymptomatic individuals (Kawchuk and Fauvel 2001) however stiffness was not measured. In addition, it has been noted that asymptomatic subjects can have a muscle response, described as a reflex response, to the application of a manipulative force to the spine (Herzog et al. 1999). In these studies the frequency of force application was different to that used clinically during manual examination with PA forces which is 0.5–2 Hz (Maitland 1986; Harms et al. 1999). Therefore, the results do not indicate if the muscle response would be the same during manual examination on symptomatic subjects. To date, the relationship between lumbar PA stiffness, increased muscle activity and LBP has not been

adequately investigated, yet, despite this fact, treatment decisions are made on the basis of an assumed relationship.

The purpose of this study was to determine whether there is a relationship between lumbar PA stiffness, muscle activity and LBP. The specific aims were to determine whether 1) a group of people with acute LBP have higher stiffness coefficient K values and lower D30 values during an episode of LBP than when their pain has resolved; 2) people with acute LBP have increased erector spinae activity (mean amplitude, coherence and gain) during the application of PA forces; 3) people with acute LBP are stiffer (higher stiffness coefficient K and lower displacement D30) and have greater erector spinae activity (mean amplitude, coherence and gain) than a group of people without LBP matched for age and gender and 4) whether there is a relationship between intensity of pain, stiffness (coefficient K) and muscle activity (mean amplitude, coherence and gain) in people with LBP.

METHODS

Experimental Design

The study used a repeated measures design to test two groups of subjects; a LBP group and a control group (Figure 4.1). The LBP group was comprised of people experiencing an acute episode of low back pain, which could be classified as non specific mechanical low back pain and the control group was comprised of people

not currently experiencing low back pain matched by age and gender to the experimental group. While the subjects were not matched for BMI, information about height and weight was collected so that BMI could be calculated to determine whether it could account for any variability in the data between the groups. There was no significant difference in BMI between the pain group and the control group (Independent Samples t test, $p=0.84$). The control group was included to observe the behaviour of PA stiffness and muscle activity over time.

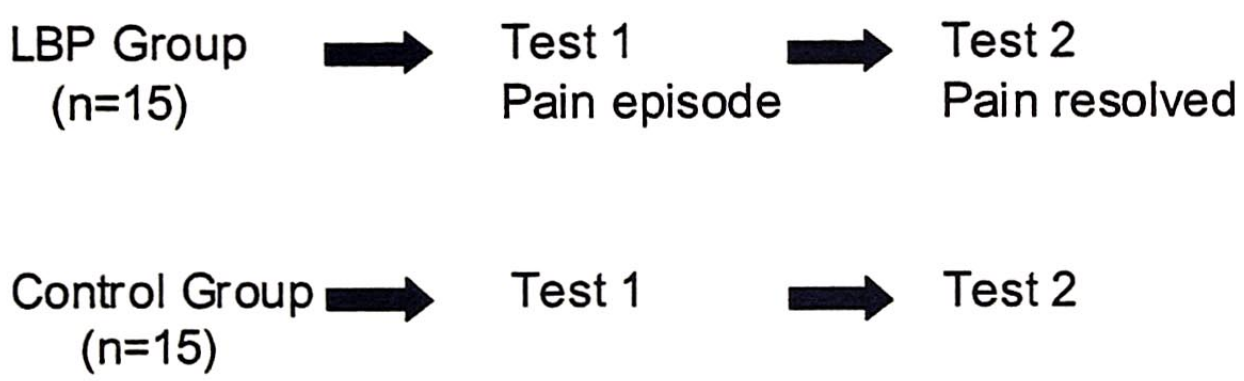

Figure 4.1. Diagrammatic representation of the Experimental Design

The subjects in the LBP group were tested initially during their episode of LBP and again when their pain had resolved. The control group was tested on two occasions at similar time intervals to their matched pain subjects.

Subjects

Each group consisted of 15 subjects matched for age and gender. The mean age of subjects in the pain group was 27.9 (± 7.3) years and in the control group was 28 (± 5) years. Both groups consisted of 13 males and 2 females. Subjects were included in the pain group if they were in the age range 18-50; they were suffering from a current episode of acute LBP which had been present for less than four weeks; the LBP was assessed by the researcher to be non specific mechanical low back pain and their symptoms could be reproduced by the physiotherapist's manually applied PA pressure. Subjects were included in the control group if they were not currently experiencing LBP, they had not felt LBP in the previous six months requiring treatment, and there were no known contraindications to the application of PA forces.

Subjects were excluded from the pain group if they were experiencing any altered sensation in their lower limbs or genital region; there was any condition present that would contraindicate the use of PA pressures eg. intervertebral disc prolapse, spinal canal stenosis, fracture, spondylolisthesis, other significant spinal disease or tumour or if their symptoms were significantly exacerbated by the application of a PA pressure. Subjects were excluded from the control group if they reported symptoms during screening or if there was any known spinal disease present.

The relatively small sample size in this study was due to the difficulties of recruiting larger numbers of subjects and being able to access them for follow up. Most of the

subjects for this study were recruited from the Australian defence forces (Army and Navy personnel). These subjects were tested during the first four weeks of their episode of acute LBP and it was planned to retest them when that episode had resolved and their pain score on the McGill Pain Questionnaire had decreased by 80%. For some subjects there was a lengthy time interval between tests as they were moved to different postings either within Australia or overseas. In some cases this resulted in periods of up to twelve months before retesting. Another factor resulting in long delays between testing was that once the episode of pain had settled many subjects returned to their normal duties involving heavy work and rigorous physical training routines which caused them to feel stiff and sore in their low back. Therefore, even though the initial pain had resolved often subjects did not return a low enough score on their McGill pain questionnaire for retesting. In many cases it was only possible for retesting to occur after a period of recreational leave or change of duty for attending training courses.

Subjects were recruited from the population of patients presenting with LBP to the Physiotherapy Department of Balmoral Naval Hospital or the Physiotherapy Department at Holsworthy Army Barracks and the Accident and Emergency Department at Royal North Shore Hospital. The experimental procedure was explained to all subjects before written consent was gained. Ethical Approval was obtained from the Human Ethics Committee of the University of Sydney, the Australian Medical Defence Ethics Committee and The Human Ethics Committee of Royal North Shore Hospital.

Procedure

Subjects were screened by the investigator to ensure that they were suitable for inclusion in the study. In addition, subjects were asked to complete a McGill Pain Questionnaire (Melzak 1983) to obtain an initial measure of their pain. The pain measure used was the pain rating index based on rank values of pain descriptors (Melzak 1975). The McGill Pain Questionnaire is reported to have acceptable reliability and validity for pain assessment (Reading 1983). Following the initial questioning the investigator examined the lumbar spine of subjects in the experimental group by manually applying a PA force in order to determine the painful vertebral level. The skin over the painful vertebral level was then marked and this level was recorded for retesting. The lumbar PA force was applied to this level during stiffness testing.

Activity of the low back muscles was recorded with surface electromyography (EMG). To minimise skin resistance, the skin adjacent to the L4 spinous process was cleaned, shaved and lightly abraded. Skin resistance of less than 5000Ω was deemed to be acceptable. Five self-adhesive surface electrodes were then applied to the skin surface. Two electrodes were placed either side of the L4 spinous process at a distance of 4cm lateral to the spinous process with an inter-electrode distance of 2.5cm. A reference electrode was also placed over the sacrum. These electrodes remained in place throughout the entire testing procedure.

Once the electrodes were in place the subject was asked to lie face down on the testing apparatus. To obtain a measure of muscle activity for normalisation of EMG, subjects were required to produce a submaximal isometric contraction of their trunk extensors by having their arms outstretched and lifting them 2.5cm off the treatment couch and holding the position for 5 seconds. Surface EMG was recorded during this activity. It was not considered appropriate to ask subjects with LBP to attempt a MVC due to the risk of exacerbation of their symptoms. It would also be difficult to determine whether the attempt was maximal as pain can inhibit muscle activity. Therefore, the submaximal procedure was a method of obtaining a standard measure of EMG activity for normalisation of EMG signals. Controls were asked to perform the same submaximal contraction so that comparison between the groups could be made. The control subjects were also asked to perform a maximal isometric contraction in the same position as the submaximal contraction but with resistance applied over the upper thoracic spine. The submaximal contraction produced muscle activity equivalent to 19.9% MVC in the control subjects.

Surface EMG recordings were also made while the subject was tested with a portable mechanical testing device (a description of the device can be found in Chapters 1 and 2). EMG was also measured with the subject lying relaxed without any force being applied.

The mechanical device was used to apply a PA force at an angle of 16° in a caudal direction at L5 and 4.5° in a caudal direction at L4. These angles were chosen as they are perpendicular to the average angulation of the vertebral bodies in the

standing posture (Stagnara et al. 1982). While applying the force the device measured the force applied and the displacement at the skin surface. During mechanical loading a maximum force of 150N was applied. The 150N level of force is commonly reached during this type of assessment by physiotherapists (Harms et al. 1999) and has been used extensively in studies of PA stiffness without causing adverse effects. Prior to stiffness testing on each of the two test occasions, all subjects were pre-conditioned with four loading cycles.

Mechanical loading of the spine involved two tests of force application for five cycles each. The PA forces were applied at two different loading frequencies i.e. 0.5 Hz (2 seconds per cycle) and 1 Hz (1 second per cycle). The two frequencies that were chosen are included in the range of frequency used in clinical assessment (Maitland 2001; Harms et al. 1999) and because different muscle responses have previously been observed at different frequencies of force application (Herzog et al. 1999; Kawchuk and Fauvel 2001). Control subjects were tested at the same vertebral level as their matched experimental group subject with the same experimental procedure.

All subjects were tested on two occasions. The mean time between tests for the pain subjects was 7.1 ($\pm$ 3.4) months and for the subjects without pain was 5 ($\pm$ 2.7) months. There was no significant difference in time between test occasions for the two groups ($P<0.05$). Subjects in the experimental group were retested when their pain has reduced by at least 80% as determined by reduction in their McGill Pain Questionnaire score.

Data Analysis

PA Stiffness

Two parameters describing the lumbar PA response were obtained from the data; stiffness coefficient K and displacement D30, using a custom designed computer analysis program. The method for calculation of these values is the same as that previously described in Chapter Two. The mean values for stiffness coefficient K and displacement D 30 were obtained for each test by calculating the average of cycles 2-5 of the stiffness test. These cycles were chosen for the analysis as the first cycle has been shown to behave differently than subsequent cycles in a stiffness test (Chapter Two). Stiffness and displacement values were determined for each subject for both testing occasions. Subsequently, the means for each group during both stiffness tests were calculated.

EMG Analysis

After amplification of the EMG and force, the EMG, displacement and force signals were sampled by a 16-bit A-D converter at 2000 Hz and stored on computer. The raw EMG was imported into Matlab software (The MathWorks, Inc Version 4.2 1994) for analysis. In order to remove any 50 Hz line frequency interference or low-frequency movement artefact, the EMG was high-pass filtered (digital 8th-order Butterworth) at 80 Hz. Subsequently, the EMG signals were full-wave rectified and

low-pass filtered (digital 8th-order Butterworth) at 5 Hz and the absolute value was calculated to obtain a DC voltage (integrated EMG: IEMG) proportional to the contraction level of the muscles. This cut-off frequency was chosen because the frequencies of interest were less than 5 Hz.

The EMG data were then analysed to determine whether a muscle response to applied PA forces was characterised by activation of a stretch reflex or by an change in the mean level. The force and IEMG signals were resampled at 20 Hz and subjected to cross correlational and spectral analysis (Bendat and Piersol 1971; Neilson 1972) to quantify any stretch reflexes evoked by the application of PA force (Figure 4.2). This analysis allows stretch-evoked muscle activity at the stretching frequencies of 0.5 and 1 Hz to be distinguished from other activity unrelated to the force application. This provided a measure of the coherence square between the signals, ie the proportion of the variance of the IEMG that is correlated with the force at each frequency. This measure for each frequency is analogous to r^2 in regression analysis. Therefore, a coherence square of 1 indicates perfectly cross-correlated signals, at that frequency, while a value of 0 indicates no cross-correlation. Coherence square differs from r^2 in that it is not dependent on the phase between the force and IEMG signals. In fact, the analysis also provides a measure of this phase relationship between force and IEMG response as a function of frequency. The gain of the reflex response as a function of frequency was also obtained. The gain provides a measure of the magnitude of any reflex response evoked by PA force, being defined as the IEMG amplitude divided by the amplitude of the force at that frequency.

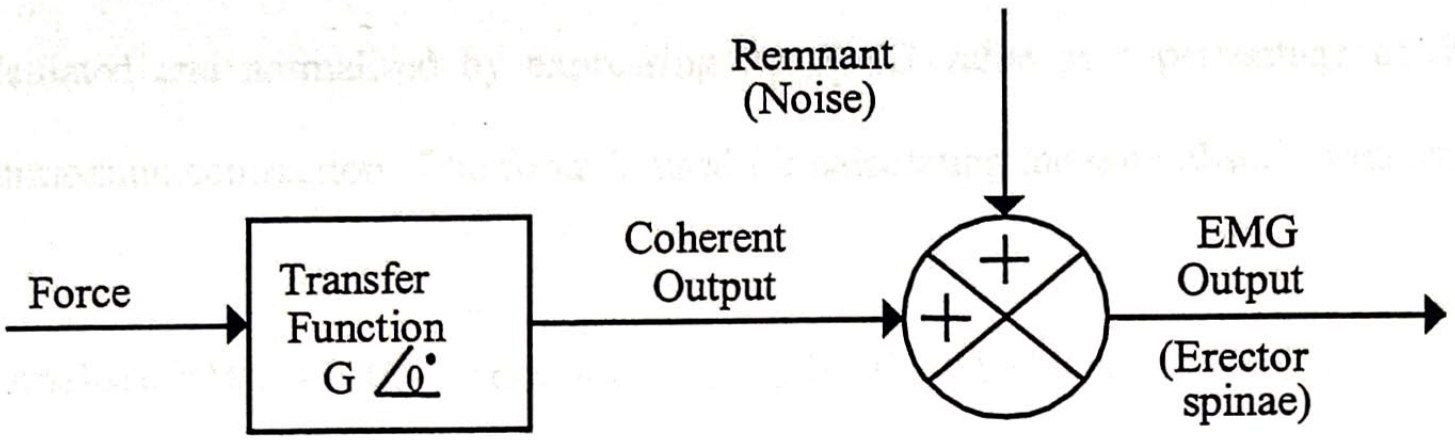

Figure 4.2. Schematic diagram for the process of calculating the coherence, gain and phase between the surface electromyographic signal (EMG) and the applied PA force. In this diagram the gain of the signal (G) is analogous to the slope of the line in a regression equation. The analysis computes a linear relationship between the applied force and EMG, where the proportion of EMG that is related to force is separated out from the signal. The remnant or noise is analogous to the residuals in a regression analysis.

If stretch reflexes of any reasonable magnitude were present, the mean level of IEMG activity would also be expected to increase during the stretching procedure. Therefore, in addition to the gain of the reflex, the mean amplitude of IEMG activity during stretch was measured and compared with that during rest. The displacement and force signals were also subjected to cross correlational and spectral analysis and the resistance to passive movement was quantified by the gain of the torque-angle relation, ie, the magnitude of the stretch-evoked force divided by the magnitude of the stretch.

To determine the mean level of EMG corresponding to each testing frequency, the mean amplitude of IEMG signal during the period of force application was calculated and normalised by expressing the EMG value as a percentage of the submaximal contraction. The formula used for calculating the normalised value was

$$\text{Normalised EMG} = 100\% \times [EMG_{\text{force application}} - EMG_{min}]/[EMG_{submax} - EMG_{min}].$$

Where $EMG_{\text{force application}}$ was the mean IEMG value calculated during the period of force application. This range was selected by identifying where force application commenced and finished and selecting the EMG data in between for analysis. EMG_{submax} was the mean value of IEMG calculated during the submaximal test from a period of 2 seconds of data where the signal was most consistent. EMG_{min} was the lowest value of IEMG recorded in all tests.

Mean normalised EMG for right and left erector spinae was obtained for both groups of subjects at both testing frequencies. The right and left mean normalised EMG values were averaged for use in statistical analysis

Statistics

The means of PA stiffness and displacement D30 for both subject groups and at each loading frequency were extracted and compared using *t* tests for within groups comparisons. Paired *t* tests were used so that the results of the current study could

be compared with those of the study by Latimer et al. (1996c). Latimer et al. (1996c) used a similar test retest design to investigate the behaviour of PA stiffness in subjects with LBP, however, their data was only analysed using paired *t* tests and there were no between groups comparison for their data. In the current study the means of all variables (PA stiffness, displacement D30, EMG mean amplitude, coherence and gain), for both subject groups and at each loading frequency were also compared using an analysis of variance (ANOVA) for between groups comparisons. Pearson's correlations between the variables of muscle activity (ie coherence, gain and normalised mean amplitude) and PA stiffness were examined to test for a linear relationship between variables. Linear and multiple regressions were also performed to test whether there was an association between pain, PA stiffness and muscle activity. In order to explore the relationship between PA stiffness, pain and muscle activity, the PA stiffness values for each testing frequency were categorised as "high" or "low" stiffness using a median split of data. For multiple regression analyses variables were entered using backwards deletion, with non-significant variables (p >0.1) removed according to criteria described by Hair (1998). The problems of relying on one approach have been noted by Hair, so a combinatorial approach has been used. Where non-significant results were interpretable and meaningful in the context of previous research the variables were retained. The statistical analyses were carried out using SPSS for Windows, release 10.0.5 (SPSS Inc, Chicago).

RESULTS

Stiffness and Low Back Pain

When PA stiffness and displacement D30 of subjects during an episode of low back pain were compared to the values when pain had resolved there was a significant decrease in PA stiffness (P=0.028) for loading at 0.5 Hz but there was no difference in D30 (P>0.05). No difference was observed in the group without pain between the two tests (Fig 4.4 and 4.5). When the comparison was carried out with data collected at 1 Hz there was no difference between tests for either group (Fig 4.6 and 4.7). In contrast, a between groups analysis (ANOVA) did not detect a difference between groups or test occasions for stiffness coefficient K ($F(1,28)=0.642$, P=0.43) and displacement D30 ($F(1,28)=0.65$, P=0.43) when tested at loading frequencies of 0.5 and 1 Hz. However, the ANOVA did detect a significant interaction for loading frequency for D30 ($F(1,28)=5.045$, P=0.033). The mean values for stiffness coefficient K and displacement D30 are presented in Table 4.1.

Stiffness coefficient K decreased between tests for the pain group but there was no significant change in D30 so it was decided to further evaluate stiffness coefficient K. There was a higher degree of variance of PA stiffness in the pain group than for control group. The stiffness data of the pain group were then classified as high or low using a median split of data and mean stiffness was compared using a two samples *t*-test. On average the people with higher stiffness had significantly higher pain (0.5 Hz P=0.038 and 1 Hz P<0.001). Therefore the level of pain varies significantly by high and low stiffness. This effect was seen at both loading frequencies (Figure 4.3)

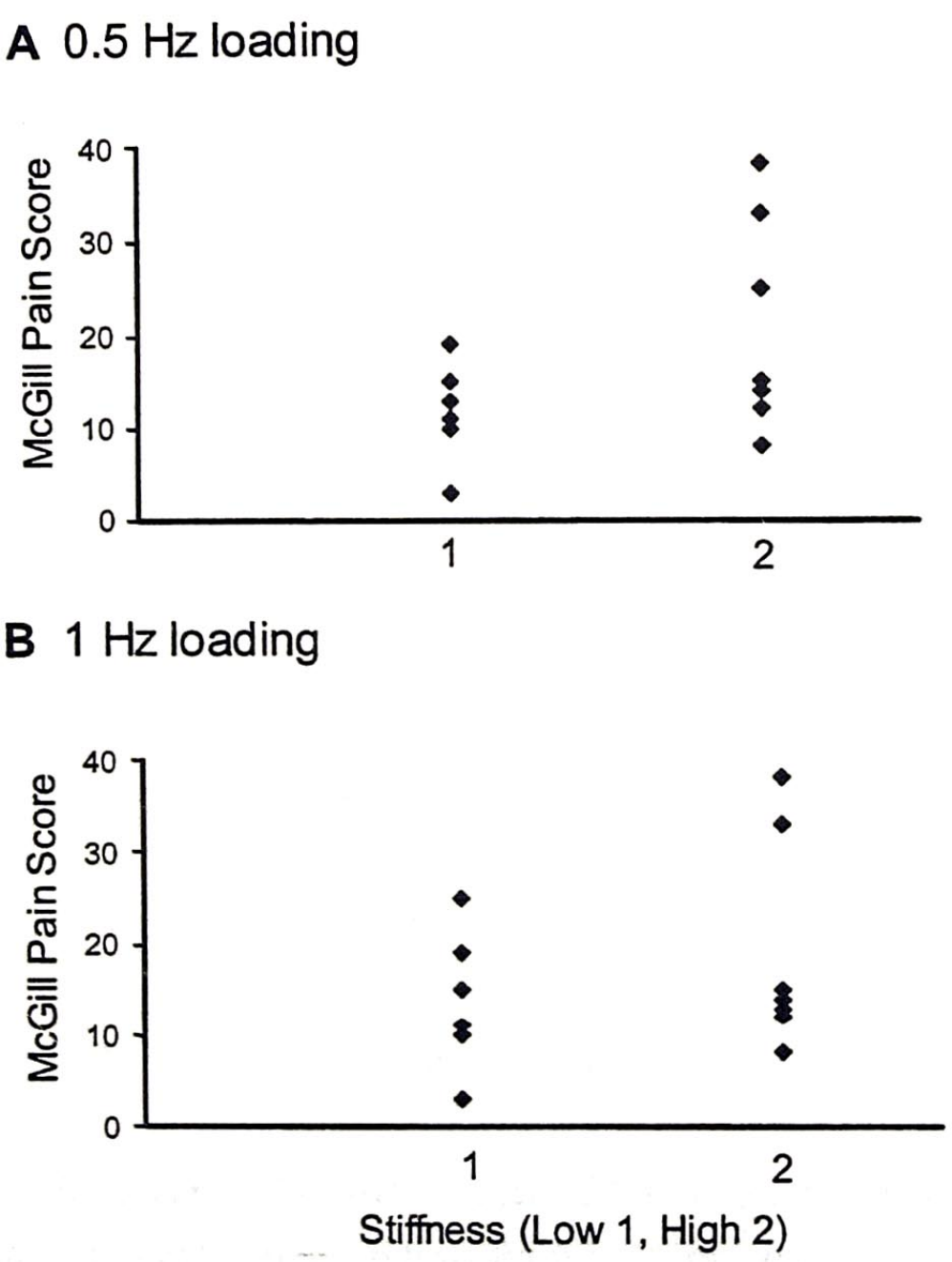

Figure 4.3. Pain scores for the subjects with LBP compared to stiffness categorized as low (1) or high (2) at 0.5Hz loading (A) and 1Hz loading (B).

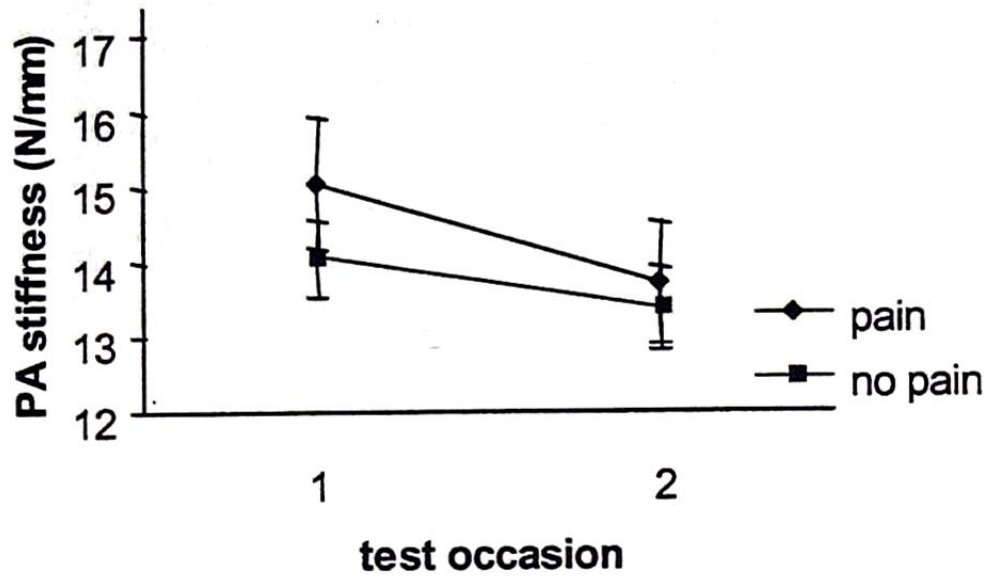

Figure 4.4. Mean (SE) PA Stiffness values for both the pain and no pain groups at initial and follow up tests for 0.5 Hz loading.

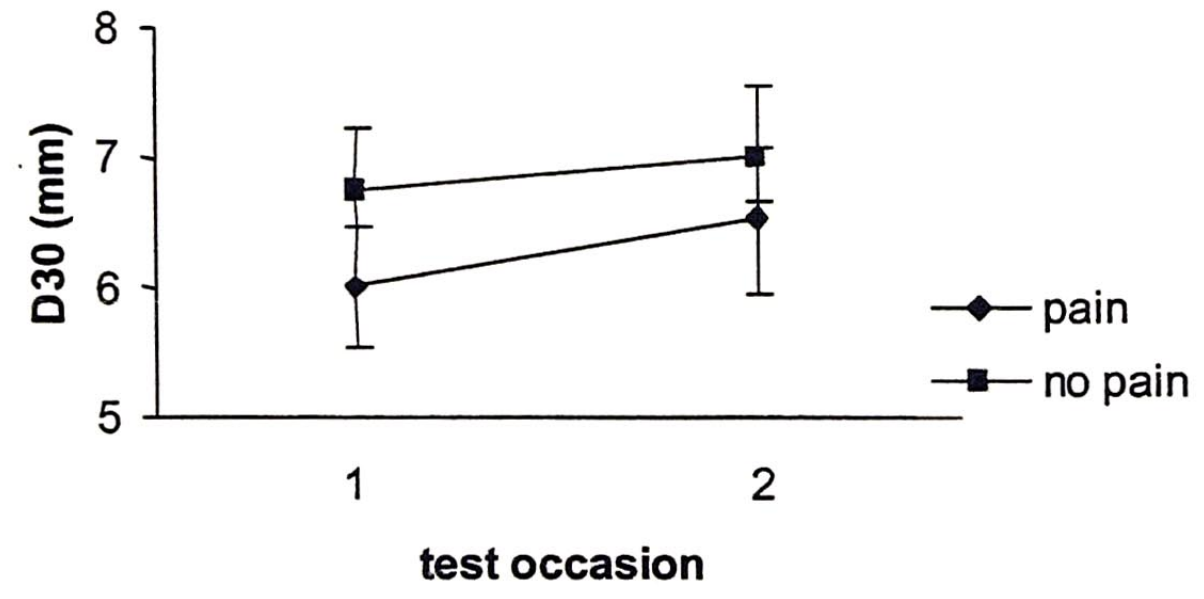

Figure 4.5. Mean (SE) Displacement D30 values for both the pain and no pain groups at initial and follow up tests for 0.5 Hz loading.

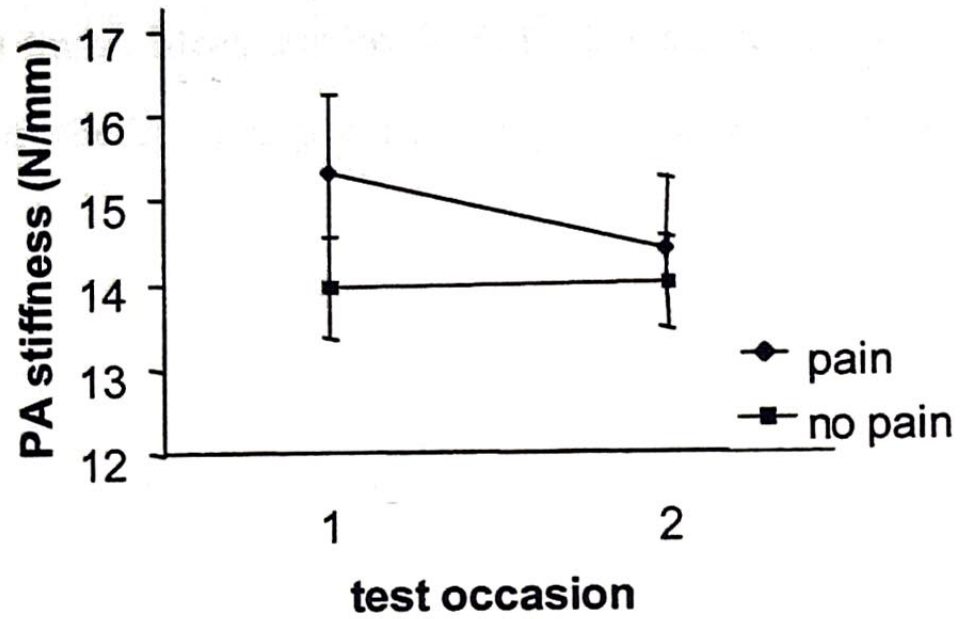

Figure 4.6. Mean (SE) PA Stiffness values for both the pain and no pain groups at initial and follow up tests for 1 Hz loading.

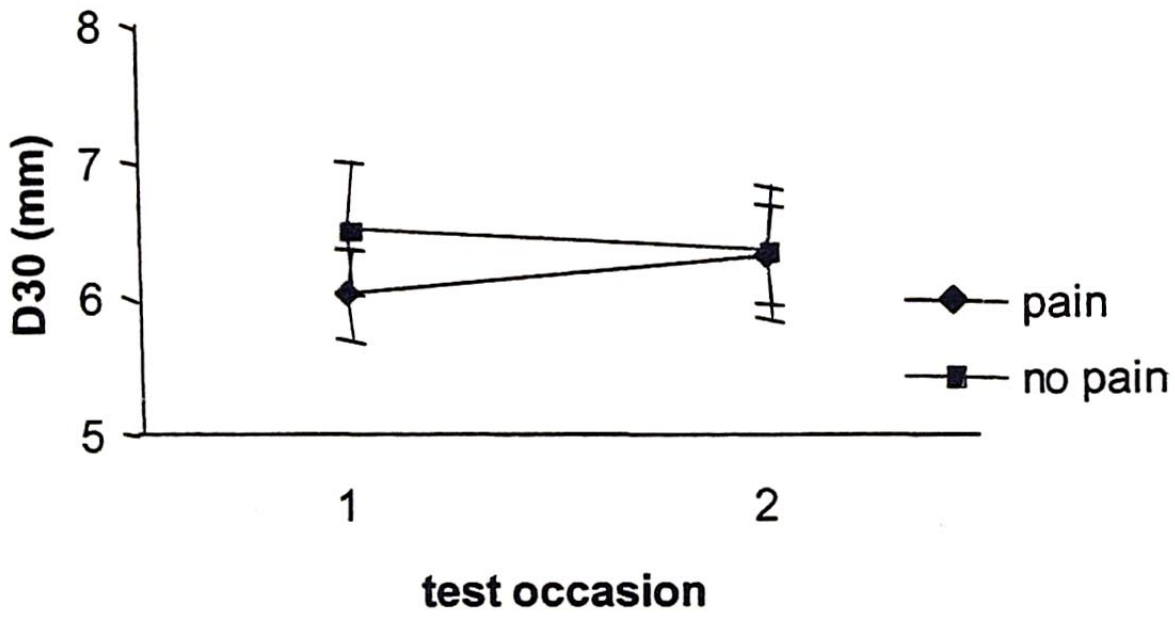

Figure 4.7. Mean (SE) Displacement D30 values for both the pain and no pain groups at initial and follow up tests for 1 Hz loading.

Table 4.1. Mean values (SE) for Stiffness coefficient K (K) (N/mm), Displacement D30 (D30) (mm), Mean amplitude EMG (Amplitude), EMG Coherence and EMG Gain for both the LBP group and control group on both test occasions.

	K	D30	Amplitude	Coherence	Gain
Test 1 - 0.5 Hz					
LBP	15.06 (0.87)	5.99 (0.47)	0.16 (0.05)	0.39 (0.07)	0.01 (0.003)
Control	14.05 (0.5)	6.75 (0.28)	0.68 (0.3)	0.24 (0.04)	0.003 (0.001)
Test 1 - 1 Hz					
LBP	15.28 (0.95)	6.02 (0.48)	0.14 (0.04)	0.52 (0.06)	0.007 (0.002)
Control	13.94 (0.61)	6.51 (0.33)	0.06 (0.02)	0.44 (0.06)	0.005 (0.002)
Test 2 - 0.5 Hz					
LBP	13.7 (0.8)	6.52 (0.57)	0.10 (0.03)	0.35 (0.07)	0.006 (0.002)
Control	13.38 (0.55)	6.99 (0.32)	0.12 (0.05)	0.26 (0.04)	0.003 (0.001)
Test 2 - 1 Hz					
LBP	14.36 (0.85)	6.31 (0.48)	0.09 (0.03)	0.56 (0.06)	0.005 (0.002)
Control	13.99 (0.54)	6.32 (0.36)	0.09 (0.03)	0.49 (0.06)	0.006 (0.002)

Stiffness and Muscle Activity

The variables for muscle activity that were extracted were the normalised mean amplitude, coherence (measure of reflex activity) and gain (measure of magnitude of reflex) (Table 4.1). A significant increase in normalised mean amplitude during stiffness testing was observed in the pain group for 0.5 Hz loading frequency (P=0.039), however there was no difference in any of the other muscle activity variables for either group of subjects between test occasions or at either of the loading frequencies. The normalised mean amplitude of surface EMG for pain subjects during their first test was equal to 22% of the submaximal contraction for 0.5 Hz and 17.9% for 1 Hz. In contrast for the subjects without pain the submaximal contraction was about 9.2% and 8.1% of a submaximal contraction respectively. In the pain subjects the normalised EMG decreased to 13.7% and 13.5% when their pain episode had resolved whereas for the control subjects the values were 11% and 10.8% respectively. These data indicate that the amplitude of erector spinae activity was greater when subjects had pain was to similar the values for the group without pain when their pain had resolved.

A trend was noted for the pain group to have higher coherence during the first test (pain episode) than the second test. In addition, obvious patterns of reflex activity occurring in response to PA loading were evident in some subjects in both groups. An ANOVA detected an interaction between the loading frequency and coherence (P<0.001), indicating that there is increased coherence with higher loading

frequency. There was also a significant interaction between loading frequency and group for gain (P=0.008) but there was no effect for group or test occasion.

When variables of muscle activity of the pain group were tested to determine whether they vary with "high" or "low" stiffness no significant variation was detected at either loading frequency. In addition, a multiple regression analysis did not find an association between PA stiffness and any of the variables for muscle activity at either loading frequency (P>0.05).

McGill pain scores

The mean McGill pain score for the LBP subjects at the time of their first test was 15.87 (±9.4) and this reduced to 1.14 (±2.2) at the second occasion of testing. This difference indicates a significant reduction in pain level (P<0.05). Nine out of the fourteen subjects retested had a score of zero on their second test. It was not possible to retest one subject, as the McGill pain score did not reduce to 80% of the initial test score during the timeframe of the study.

Although subjects in the control group did not fill in a McGill pain questionnaire they were only included if they had no back pain on screening for inclusion of the study. None of the control subjects reported any LBP on either testing occasion.

Relationship between LBP, muscle activity and stiffness

Pearson's correlations were assessed to examine whether there was a linear relationship between pain, PA stiffness and muscle activity. No linear relationships were significant (P>0.10). However, multivariate regression analyses detected a significant association between pain, PA stiffness and muscle activity variables for loading at both 0.5 Hz (p=0.034) and for 1 Hz (p=0.031).

A significant association between pain during the acute episode and four variables measured at the initial test was also found for loading at 0.5 Hz (P=0.034, Adjusted R^2= 49.7%) with multiple regression analysis. Therefore, 49.7% of the variance in pain scores during an episode of LBP can be explained by four measures taken during the acute episode. After allowing for the level of EMG amplitude, coherence and gain, PA stiffness was significantly associated with the level of pain during the acute episode. Subjects with higher stiffness values reported significantly more pain (P=0.043) (Table 4.2).

Table 4.2. Predicting Pain from PA stiffness and muscle activity at 0.5 Hz: multivariate analysis.

Independent Variable	*β*	*SE Coeff*	*T*	*P*
Constant	14.21	7.95	1.79	0.108
High Low stiffness	9.06	3.85	2.35	0.043
EMG Amplitude	-17.37	10.72	-1.62	0.139
EMG Coherence	-13.01	8.20	-1.59	0.147
EMG Gain	-613.50	349.10	-1.76	0.113

R^2= 0.652: Adjusted R^2 = 0.497

A significant association between pain during the acute episode and five variables measured at the initial test was also found for the 1 Hz data (P=0.031, Adjusted R^2= 53.2%). Therefore, 53.2% of the variance in pain scores during the acute episode can be explained by measures of stiffness and muscle activity taken during the same episode. After allowing for the level of EMG amplitude, coherence and gain, PA stiffness was significantly associated with the initial level of pain. Subjects with higher stiffness values again reported higher levels of pain (P=0.017) (Table 4.3).

Table 4.3. Predicting Pain from PA stiffness and muscle activity at 1 Hz: multivariate analysis.

Independent Variable	***β***	***SE Coeff***	***T***	***P***
Constant	18.65	10.22	1.82	0.101
PA stiffness 1 Hz	-1.88	0.88	-2.15	0.060
EMG Amplitude	-29.30	13.32	-2.20	0.055
EMG Coherence	16.80	10.04	1.67	0.129
EMG Gain	-725.50	298.30	-2.43	0.038
High Low stiffness	17.45	6.00	2.91	0.017

R^2= 0.699: Adjusted R^2 = 0.532

At seven month follow up some subjects had no pain, with the remaining subjects recording pain scores above zero on the McGill Pain Questionnaire. Presence of pain seven months after an episode of low back pain was marginally significantly associated with four variables measured during the during the acute episode of pain (P=0.062 Adjusted R^2 =38.5%) with force application of 0.5 Hz. That is 38.5% of the variance in pain scores seven months after the acute episode can be explained by

four measures taken during the episode of pain. After allowing for the level of EMG amplitude and gain, stiffness was significantly associated with the level of pain at 7 month follow up. The effect of stiffness was different for high and low stiffness patients (P=0.014) (Table 4.4). There was an association between pain at follow up and EMG amplitude and high low stiffness during the acute episode. This indicates that low stiffness and EMG amplitude in the acute episode was associated with higher pain seven months later. This is in contrast to the results of testing during the acute episode where subjects with high stiffness reported high pain and subjects with low stiffness reported lower pain levels. This association was not evident for the 1 Hz data. Since nine subjects recorded no pain on follow up the data was not normally distributed on the dependent variable, suggesting that the marginally significant result could be due to influential outliers. However, data subsetting for lack of fit showed no evidence of lack of fit (P=0.1), suggesting that the result is not due to outliers.

Table 4.4. Predicting Pain at follow up from PA stiffness and muscle activity at 0.5 Hz during the acute episode: multivariate analysis.

Independent Variable	***β***	***SE Coeff***	***T***	***P***
Constant	2.63	2.04	1.29	0.226
PA stiffness 1 Hz	0.47	0.22	2.10	0.062
EMG Amplitude	-6.78	2.63	-2.58	0.027
EMG Gain	-48.66	35.56	-1.37	0.201
High Low stiffness	-4.30	1.45	-2.97	0.014

R^2= 0.561, Adjusted R^2 = 0.385

DISCUSSION

Approximately 50% of the variance in pain during an episode was predicted by a combination of stiffness and muscle activity (see Tables 4.2 and 4.3). In particular, PA stiffness at two loading frequencies was related to McGill pain scores indicating that a high pain score corresponds to higher PA stiffness. In addition, the amplitude of erector spinae EMG at 0.5 Hz loading was greater during an episode of acute low back pain.

For many years, practitioners of manual therapy have made the assumption that muscle activity is a contributing factor to the increased lumbar PA stiffness clinically observed in patients with LBP (Mennell 1960; Maitland 1986). Increased PA stiffness has been previously demonstrated in people with LBP (Latimer 1995). Latimer et al (1996) proposed that increases in muscle activity could be contributing to increased stiffness during the episode of pain. The current study does not demonstrate a relationship between activity of the erector spinae and PA stiffness in subjects with LBP as no association was demonstrated between any of the variables of muscle activity and PA stiffness. There was a significant interaction between coherence and loading frequency suggesting there was higher coherence with faster loading. Coherence is affected by loading frequency but the lack of interaction for pain state suggests this interactions is independent of LBP in a relaxed subject.

In our study, muscle activity was quantified by the overall mean normalised amplitude, coherence and gain during the period of PA loading. The absence of a

relationship between the variables of muscle activity we measured and stiffness suggests that activity in the superficial extensor muscles may not be contributing to PA stiffness in the relaxed subject. However, we did observe a trend of increased coherence (stretch reflex) in subjects with LBP at 0.5 Hz. It is also possible that the magnitude of change in EMG amplitude we measured was not great enough to result in clinically relevant increases in PA stiffness. Submaximal voluntary contraction of the lumbar erector spinae as low as 10% of maximal voluntary contraction results in significant increases in PA stiffness (Chapter Three). In our study the mean amplitude at 0.5 Hz at the initial test was 22 % of the submaximal contraction value. This equates to only approximately 4.4% of the mean amplitude of the maximal contraction for the subjects without pain. Therefore, the amplitude of muscle activity occurring in response to PA loading is relatively small and may not be great enough to result in significant increases in PA stiffness.

Various muscles (e.g. erector spinae, multifidus, transversus abdominis and the diaphragm) have been proposed to contribute to stiffness of the spine either directly or indirectly (Hodges and Richardson 1997b; Chapter Three; Hodges and Gandevia 2000a). In this study the electrodes were placed on the skin over the erector spinae adjacent to the lumbar spine at L4. Therefore, activity of the erector spinae is likely to be the major contributor to the signal we detected. It is possible that activity of other muscles that were not measured were contributing to the increases in lumbar PA stiffness that we observed in this study. Multifidus and transverus abdominis (TrA) are deep muscles and surface electrodes are unlikely to detect activity in these muscles. The diaphragm only attaches to the spine as far as L2-L3 (De Troyer and Estenne 1988) and any activity during PA loading would not have been detected

with our placement of surface electrodes. Therefore, this study does not rule out the possibility that other muscles may contribute to, or influence spinal PA stiffness.

Our results demonstrated an interaction between the loading frequency and coherence of the EMG signal but there was no interaction for group. Herzog et al. (1999) described a reflex responses associated with the application of manipulation which is a high speed technique. The loading frequencies used in this study are slower than the application of force during a manipulation. However, consistent with Herzog et al. (1999) findings, the interaction between coherence and loading frequency indicates that a modulation of activity is more likely to accompany a faster loading frequency even with the relatively slower frequencies of cyclic loading. Our findings indicate that there was no difference between the groups which suggests that this response is likely to be a normal response to externally applied forces.

Lumbar PA stiffness measured during loading at 0.5 Hz decreased with resolution of pain in subjects with acute low back pain. However, there was no change in PA stiffness or displacement D30 at 1 Hz loading. These results are in accordance with those of Latimer et al. (1996c) who also demonstrated a decrease in lumbar stiffness as pain settled, when measured during PA loading at a frequency of 0.5 Hz. The study by Latimer et al. (1996c) indicated that there was a small - medium effect size of 0.51 standard deviations (Welkowitz et al. 1976) for stiffness coefficient K. In our study the effect size was 0.41 standard deviations which is of a similar magnitude to that of the one found by Latimer et al. (1996c). While Latimer et al. (1996c) report paired *t*-tests to analyse their data there is no report of a comparison between groups. The results of the current study indicate no effect with comparison

between groups, therefore, the effect of decreasing PA stiffness in subjects with resolution of low back pain must be considered weak.

The relatively small number of subjects may have contributed to the lack of significant results detected with between groups comparisons for PA stiffness, displacement D30 and variables of muscle activity (except amplitude at 0.5 Hz). Greater numbers of subjects would give the analyses more power, however, some significant results were detected. The lack of significance in comparisons of PA stiffness may simply be reflecting the wide variability in the general population (Chapter Three). The increased stiffness during the episode of pain that we observed should be viewed with caution since, while a significant decrease in stiffness was observed for the subjects with pain (P=0.028) the change in stiffness was not significant when compared in an overall ANOVA allowing for comparison of stiffness between pain and control groups (P>0.05). This difference may be due to the lack of power due to the limited number of subjects.

The aim of this study was to determine whether there is a relationship between PA stiffness, lumbar muscle activity and low back pain. The association between pain scores during an episode of low back pain and a combination of stiffness and muscle activity variables does suggest the existence of a relationship between these variables. The relationship, however, is likely to be a simple one. The results indicate that high pain scores are related to increased stiffness possibly suggesting that increased PA stiffness may be an important response in subjects with acute LBP. It is interesting to note the strong trend that pain at follow up several months after the acute episode is associated with low initial stiffness and low muscle activity.

There is increasing clinical and research interest in the proposal that ongoing LBP is related to a lack of spinal stability and muscle dysfunction, particularly in the muscles described as stabilisers of the spine (Hides et al. 1994; Hodges et al. 1997a; Hodges and Richardson 1997a; Hodges and Richardson 1999b; Hodges and Gandevia 2000a). The results of this study may be an indication that low stiffness during acute LBP is due to failure of the muscles to adequately stabilise the spine when an external force is applied. One of the mechanisms by which spinal stabilising muscles are thought to act is by directly or indirectly stiffening the spine (Hodges and Richardson 1997b; Hodges and Gandevia 2000a). Many of the muscles involved in spinal stability were not measured in this study.

Clinical Implications

The association between pain with PA stiffness and variables of muscle activity is suggestive of a relationship between these factors in people with acute low back pain (Table 4.2 and 4.3). It is interesting to note, that although relationships between pain and stiffness and pain and muscle activity were demonstrated there was no evidence of a direct relationship between muscle activity and stiffness during testing which simulates the clinical testing of PA stiffness. There were however significant interactions between loading frequency and displacement D30 as well as loading frequency and muscle activity which indicated that displacement was decreased with faster loading frequency and there was greater modulation of the EMG signal with faster loading. Modulation of EMG activity may be considered a normal reaction to PA loading in some subjects but appears to be independent of the pain state.

Therefore, if muscle activity is perceived during manual assessment of a patient it does not necessarily mean it is related to the stiffness.

The response of EMG coherence and gain that we observed is not consistent with a stretch reflex response in all subjects. The trend of coherence to be increased in subjects with LBP could be demonstrating a voluntary reaction to the PA forces. However, in a few subjects the characteristics of the observed EMG signal was suggestive of a stretch reflex response ie there was high coherence and gain associated with an appropriate phase relationship to the application of force. Therefore, it is possible that our group of subjects with acute non specific mechanical LBP did not represent a homogeneous group of subjects. There could be different categories within the classification of non specific LBP of pain and some may in fact demonstrate reflex response to externally applied forces. This highlights an area for further investigation.

Conclusions

This is the first study to compare the effect of the application of PA forces on muscle activity in subjects with LBP with the effect on subjects without LBP. The association between pain, stiffness and muscle activity that we observed is important as it confirms that a relationship exits between theses variables however it does not suggest a mechanism for PA stiffness in the resting patient. Amplitude of muscle activity is greater in subjects with LBP but modulation of muscle activity is more likely to occur with faster loading frequencies and should be considered a normal

response. The lack of association between muscle activity and PA stiffness suggests that increases in muscle activity in response to PA loading are not great enough to influence PA stiffness at rest. Other muscles that were not measured in this study and have been proposed to contribute to lumbar stiffness, particularly during voluntary tasks, could be investigated in future studies.

SMILE ! YOUR HAVING A GREAT DAY

CHAPTER FIVE

TRUNK MUSCLE ACTIVITY DURING DIFFERENT BREATHING TASKS INFLUENCES LUMBAR PA STIFFNESS

This study was supported in part by a George Burniston-Cumberland Foundation Fellowship.

ABSTRACT

The diaphragm may contribute to lumbar postero-anterior (PA) stiffness as it attaches directly to the upper lumbar vertebrae. The diaphragm contributes to stability of the spine and is thought to do this by increasing stiffness and intra-abdominal pressure. If diaphragm activity contributes to PA stiffness it would be expected that PA stiffness would be greater during phases of respiration where the diaphragm is active. The aim of this study was to determine whether low lumbar PA stiffness is affected by respiration.

Ten subjects without low back pain were recruited for this study. L4 PA stiffness was measured with a mechanical device during a number of static and dynamic breathing tasks. Activity of the chest wall and abdominal wall was recorded with surface EMG and chest movement was recorded with an impedance coil (Respitrace). In order to accurately determine PA stiffness under a variety of conditions it was necessary to devise a new method to calculate lumbar stiffness. The reliability of the new method of stiffness calculation was determined.

Lumbar PA stiffness at L4 did not increase during the static breathing conditions tested despite increases in lung volume. During dynamic breathing conditions stiffness was greater during inspiration than expiration. Although lung volume during breath holding does not change L4 PA stiffness, stiffness is increased during dynamic inspiration and is probably due to inspiratory muscle activity. This study also demonstrates that the new method of stiffness calculation has excellent reliability for both static and dynamic breathing conditions.

INTRODUCTION

Many factors are thought to influence PA stiffness, however all the contributing factors have not yet been identified. Activity of the trunk extensor muscles is one factor that has been shown to increase PA stiffness (Chapter Three). Activity of the diaphragm is another factor which may influence PA stiffness because the diaphragm is anchored to the lumbar spine down as far as L3 (De Troyer and Estenne 1988). The diaphragm is thought to play a role in stability of the spine either by influencing spinal stiffness directly by restricting intervertebral motion or indirectly by increasing intra-abdominal pressure (Hodges et al. 1997a). Therefore, it is possible that activity of the diaphragm might contribute to lumbar PA stiffness. The contribution of the diaphragm to spinal PA stiffness may be inferred by determining the effects of the breathing cycle on PA stiffness. An early study investigating the effect of breathing and breath holding on lumbar PA stiffness indicated that stiffness was greater when measured during tidal breathing than at functional residual capacity (FRC) (Beaumont et al. 1991). Activity of the diaphragm is one possible explanation for these results as the diaphragm is active during the inspiratory phase of tidal breathing but should be relaxed while breath holding at FRC. However, PA stiffness is less with breath holding at maximal inspiration than at FRC (Keaveney et al. 1989).

Manual assessment of lumbar PA stiffness in the clinic does not control for breathing. However as stiffness appears to be influenced by different states of breathing (eg normal tidal breathing and breath holding), it may be appropriate to

standardise breathing during testing. Stiffness testing protocols that require subjects to hold their breath at FRC, have been used extensively in a number of laboratories (Latimer et al. 1996c; Allison et al. 1998; Edmondston et al. 1998; Chapter Three). This procedure was adopted for the studies already described in this thesis due to observations made in previous studies (Beaumont et al. 1991), which indicated that breathing may influence spinal PA stiffness and to avoid the confounding effect of displacement due to breathing. Determining the effect of different phases of breathing on stiffness should increase understanding of factors contributing to PA stiffness and may provide guidelines for standardising clinical practice. Therefore, the aim of this study is to investigate more fully the effect of both static and dynamic breathing patterns on lumbar PA stiffness.

METHOD

Experimental Design

This study was conducted using a repeated measures design that involved measuring lumbar PA stiffness during five different breathing tasks. Each subject was tested performing all tasks during the one testing session.

Subjects

Two male and eight female subjects (mean age 22.9 ±6.8 years) without low back pain were recruited for this study. Subjects were included in the study if they were not currently experiencing low back pain. Subjects were excluded from the study if they had experienced back pain in the previous six months, had any known spinal disease, were currently taking oral corticosteroid medication, had diagnosed osteoporosis or if they reported symptoms during screening. No volunteers had to be excluded from the study. The institution's Human Ethics Committee approved the study and subjects were required to read an information sheet and sign a consent form.

Measurements

Lumbar PA Stiffness

Postero-anterior (PA) stiffness of the lumbar spine was measured with a mechanical device that simulates the clinical application of PA forces by physiotherapists. This device has been described in Chapter One. The device was used to apply a PA force over the L4 spinous process at an angle of 4.5° directed caudally. This angle was chosen as it simulates manual force application in the clinic (Viner and Lee 1995). For this study the force was applied in a rhythmical oscillating manner at a frequency of 0.5 Hz or one loading cycle each 2 seconds. A loading cycle is the time from starting to apply the force to the complete release of the force with equal parts of the cycle for loading and unloading. The maximum level of force was 110N,

which is within the range of force commonly used by physiotherapists for the assessment and treatment of low back pain (Harms et al. 1999). The level of force was kept low so that the force application during the dynamic tidal breathing did not interfere with the breathing pattern of the subject.

During stiffness testing surface electromyography (EMG) recordings were made from the chest wall and the abdominal wall to give an indication of the level of activity in the underlying muscles. Silver/Silver Chloride (Ag/AgCl) surface electrodes were placed in pairs over the right antero lateral abdominal wall midway between the rib cage and iliac crest and the 7-8th intercostal space in the mid clavicular line, a reference electrode was also placed over the sacrum. Prior to electrode placement the skin impedance was reduced to less than 5000Ω by shaving, lightly abrading and rubbing with alcohol. EMG recordings were sampled at a rate of 2000 samples per second. Data was collected using a BIOPAC system with Acknowledge software (BIOPAC Systems, Inc). Recordings of the chest wall electrodes were most likely to be from the intercostal muscles and diaphragm and these recordings will be referred to as Respiratory EMG. The anatomy suggests that there are no external intercostal muscles in the region of the electrodes so the activity recorded should be mainly that of diaphragm and internal intercostal muscles (Osmond 1985). Although there is some controversy surrounding the action of the intercostal muscles is it generally accepted that the internal intercostal muscles are predominantly expiratory (Osmond 1985). Therefore, activity recorded during inspiratory tasks is most likely to indicate activity of the diaphragm. The abdominal wall electrodes recorded activity of the abdominal muscles including internal

oblique, external oblique and Transversus abdominis and will be referred to as abdominal EMG.

A lightweight, elasticised band with an impedance coil (respitrace) was placed around the lower chest wall. This is designed to detect changes in chest wall shape and was used to monitor phases of the respiratory cycle during testing.

Procedure

After obtaining informed consent subjects were asked to undress to their underwear to expose the lumbar spine and then to lie face down on a treatment couch with a hole for the face. The surface EMG electrodes were applied and the elasticised band (Respitrace, Ambulatory monitoring, NY, USA) was placed around the lower chest. Subjects placed their arms by their head as this position was needed so that the subject could insert the mouthpiece for assisted mechanical ventilation. The L4 spinous process was then palpated and marked using the method employed in previous studies of this thesis and described by Grieve (1984).

Subjects were initially asked to perform a maximal inspiration during which Respiratory EMG was recorded and the volume was measured with a Wrights Respirometer (BOC, England). EMG during an inspiration of 50% of the maximum recorded with the spirometer was also measured. The EMG recordings were made to provide a standardised value to use for normalising the EMG data that were

recorded during the breathing tasks. The volumes were measured to give values for "calibrating" the respitrace recordings.

The PA stiffness at L4 was then measured by the mechanical device under static and dynamic conditions, in random order. The static conditions were breath holding at the end of normal expiration (functional residual capacity - FRC), breath holding at end of normal tidal inspiration (TI) and breath holding at end of maximal inspiration (MI). The dynamic conditions were prolonged inspiratory effort (PI), normal tidal breathing (V_TI for inspiration and V_TE for expiration) and relaxed breathing with assisted mechanical ventilation (V_MI for inspiration and V_ME for expiration). Before commencing any stiffness testing subjects were preconditioned with 4 loading cycles.

To perform the static breath holding tasks, subjects were first asked to breathe out to FRC and then to achieve the required volume based on the instructions given. On reaching the required volume they were instructed to hold their breath and maintain breath holding for the duration of 5 loading cycles (approximately 10 seconds). Lumbar PA stiffness measurement was carried out once during each static condition.

When performing the test involving prolonged inspiratory effort, subjects were first asked to breathe out to FRC and then inhale slowly and steadily to a volume of 1000ml indicated by a marker on an incentive spirometer (Voldyne 5000, Sherwood Medical, St Louis, MO 63103, USA). They were asked to maintain this volume using the incentive spirometer while stiffness was testing during the application of

five loading cycles. The absolute value of 1000 ml would represent different relative proportions of maximum inspiratory capacity for different subjects.

For stiffness testing during tidal breathing subjects were first asked to establish a relaxed pattern of breathing. When a regular pattern of breathing was observed on the recording from the respitrace, mechanical loading commenced. For testing during assisted ventilation the subject was asked to relax and establish a pattern of comfortable assisted ventilation before mechanical loading commenced. Ventilation was assisted by a portable volume cycled ventilator (Drager EV800) intended for home use. Subjects wore a nose clip during the assisted ventilation and the mouthpiece of the ventilator was passed through the face hole in the couch so that the subject could remain in the same testing position as for the rest of the study. The subject could easily remove the mouthpiece that was used to deliver the assisted ventilation if assisted ventilation caused any discomfort. The ventilator was set to each subject's rate and depth of breathing. Before stiffness testing, subjects were given practice with assisted ventilation for as long as it took them to achieve maximal relaxation with the machine triggering breathing.

To measure PA stiffness during both the tidal breathing and mechanical ventilation tasks loading was carried out for 9 cycles (maximum possible in one test with the device) each with a maximum force of 110N. The 9 cycles ensured that a suitable number of inspiratory and expiratory cycles were collected for analysis. In addition, to assist in achieving enough cycles for analysis, the tidal breathing and mechanical ventilation tests were performed 3 times. All other tests were performed once. Respiratory and abdominal EMGs were recorded during all stiffness testing.

Data Analysis

A common method of calculating PA stiffness involves taking the slope of a regression line fitted to the force displacement curve between 30 and 90N of force (Latimer et al. 1996b). However, for this study a new method of calculating PA stiffness was devised (instantaneous PA stiffness). It was necessary to devise a new method, as a consistent level of force could not be achieved in all subjects during the dynamic breathing conditions due to movement of the spine. Instantaneous PA stiffness was calculated at 50N of force, as this was the highest level of force that was achieved in all test conditions for all subjects. To calculate instantaneous PA stiffness, the force displacement curves were plotted for each stiffness test. A second order polynomial was fitted to the force displacement curve and the quadratic roots were determined for 50 N. The quadratic roots for 50N were then used in a linear regression to determine the slope of the curve at 50N. The slope of the force displacement curve at 50N was the stiffness value. This calculation was carried with Matlab software (The MathWorks, Inc Version 4.2 1994) using an analysis program specifically written for this purpose.

The raw EMG signals were processed by the same method described in Chapter Four to obtain an integrated EMG. The integrated EMG was then expressed as a proportion of the value obtained in the maximum test (i.e. the procedure used or normalising the EMG signals). Mean values for each condition were calculated and used for analysis.

Reliability

Reliability of the procedure for the calculation of instantaneous PA stiffness was examined by calculating the ICC (2,1) to compare multiple stiffness values obtained within each of the breathing tasks performed. Reliability for the static conditions was determined by comparing the stiffness at 50N of force between two successive cycles excluding the first cycle (the first cycle is shown to behave different to the subsequent cycles) (Chapter 2) in each stiffness test. In the dynamic conditions (tidal breathing and assisted ventilation) reliability was determined by calculating the average stiffness at 50N of force for all cycles in one test that could be identified as wholly inspiratory and expiratory. The consistency of the average values in the three different tests for the same breathing task was then determined. A different method was used for the dynamic conditions as three tests were undertaken for all subjects and sometimes there was not one wholly inspiratory or expiratory cycle within a test. Therefore, testing 2 cycles within the same test was not possible in all cases.

In addition, PA stiffness was calculated for the static tasks using the method of taking the slope of a regression line fitted to a force displacement curve between 30 and 90N (Latimer et al. 1996b). The range of force used at FRC was 30 – 90N, however for TI and MI 30 – 70N was used as the maximum force was not greater than 70N in some tests. These calculations were carried out to compare whether the instantaneous and 30-90N methods of stiffness calculation given the same result.

Statistics

An ANOVA (Winer 1991) with planned contrasts was used to compare the mean values of lumbar PA stiffness at L4 for all subjects across all conditions. As the experiment involved both static and dynamic tasks, the planned contrasts compared stiffness values of the static tasks with each other and stiffness values of the dynamic tasks with each other as well as performing a comparison of the stiffness values of static and dynamic tasks together.

Pearson's r was calculated to examine the correlation between the new and old methods and ICC (2,1) was used to look at the reliability of the old method over these tests. In addition, the difference between stiffness values at FRC for the normal and new methods of calculation was compared with a paired *t* test.

RESULTS

Reliability of instantaneous PA stiffness measure

The instantaneous PA stiffness at specific force values demonstrated excellent reliability for both static and dynamic conditions (Table 5.1). The lowest ICC (2,1) value (0.88) was for prolonged inspiration (although still high reliability), which may be explained by the continued respiratory effort required for this task. This

means there is likely to be increasing stiffness throughout the test. In contrast, once the desired lung volume was achieved during the static tasks it was maintained by breath holding which requires less effort.

The instantaneous PA stiffness values were highly correlated with those using 30-90N method for calculating stiffness (FRC r^2=0.94, TI r^2=0.98, MI r^2=0.94). An ICC (2,1) was also calculated to determine the reliability between the two methods. The reliability of PA stiffness at FRC calculated between 30 and 90N was 0.61 compared to 0.91 and 0.96 for TI and MI respectively. The stiffness values for MI and TI were calculated between 30 and 70N, as 70N was the maximum force achieved in both tests for all subjects. Lumbar PA stiffness calculated with the 30-90N method (13.52 N/mm) was significantly greater than the value obtained for instantaneous PA stiffness (11.00 N/mm) (P<0.0001). When PA stiffness at FRC was calculated between 30 and 70N the reliability increased to 0.89. The 30-90N method of stiffness calculation also demonstrated excellent reliability for the data from the static conditions in this study (Table 5.1). Only the static conditions were analysed in this way so they could be compared with reliability of previous studies using that method of analysis.

Table 5.1. ICC (2,1) values for calculation of stiffness

Test	Condition	ICC (2,1) instantaneous	ICC (2,1) 30-90N	ICC (2,1) instantaneous vs 30-90N (2-5 cycles)	
Static	**Within test reliability**				
	Functional Residual Capacity	0.96	0.98	0.61	(30-90N)
	(FRC)			0.89	(30-70N)
	End of tidal inspiration (TI)	0.98	0.97	0.91	
	Maximal Inspiration (MI)	0.90	0.91	0.96	
Dynamic	**Between test reliability**				
	Prolonged inspiration (PI)	0.88			
	Inspiratory cycles (V_TI)	0.99			
	Expiratory cycles (V_TE)	0.95			
	Inspiratory cycles (V_MI)	0.96			
	Expiratory cycles (V_ME)	0.92			

Comparison of instantaneous stiffness between different breathing conditions

When subjects were asked to perform breath holding during a number of static inspiratory breathing tasks there was no change in stiffness between the tasks despite obvious changes in lung volume ie there was no difference between stiffness with breath held at FRC and at tidal inspiration (P=0.59) or between FRC and maximal inspiration (P=0.52). However, there was a significant increase in PA stiffness when PA stiffness during these static tasks was compared to prolonged inspiration (P<0.05) (Table 5.2, Figure 5.1)

PA stiffness testing during relaxed tidal breathing was greater during inspiratory than expiratory cycles (P=0.003). A similar pattern was also observed with the assisted ventilation (P<0.001). The stiffness during inspiration with assisted ventilation was greater than for tidal breathing (P=0.007), however, there was no significant difference in the values for the expiratory cycles (P=0.3). Stiffness during prolonged inspiration was greater than stiffness during expiration with both tidal breathing and mechanical ventilation (P<0.005) but not different from inspiration during tidal breathing or mechanical ventilation (P>0.1).

When PA stiffness during static tasks was compared with that during dynamic tasks there was no difference between FRC and tidal breathing (P=0.2), however PA stiffness was greater during mechanical ventilation than at FRC (P<0.05). Stiffness during breath holding at tidal inspiratory volume was also greater than expiration during tidal breathing (P=0.016) but no different from inspiration during tidal

breathing (P=0.16). Stiffness during breath holding with maximal inspiration was no different from both types of dynamic expiration (P=0.43 V_TE and P=0.29 V_ME). Although stiffness with breath held at maximal inspiration was less than stiffness during the inspiratory cycles of mechanical ventilation (P=0.0008), there was no difference in stiffness between breath held at maximal inspiration and stiffness during inspiratory cycles of dynamic tidal breathing (P=0.06).

Table 5.2. Mean instantaneous stiffness (standard error) during different breathing tasks (functional residual capacity (FRC), tidal inspiration (TI), maximal inspiration (MI), prolonged inspiration (PI), dynamic tidal inspiration (V_TI), dynamic tidal expiration (V_TE), mechanical inspiration (V_MI) and mechanical expiration (V_ME)) for the new method of calculation and for the normal method of calculation for the static tasks. Mean values (standard error) for normalised respiratory EMG and Abdominal EMG.

	FRC	**TI**	**MI**	**PI**	**V_TE**	**V_TI**	**V_ME**	**V_MI**
Instantaneous	11.00	11.3	10.60	12.74	10.24	11.73	9.95	12.78
PA Stiffness	(0.64)	(1.04)	(0.94)	(1.08)	(0.93)	(1.05)	(0.85)	(0.87)
(K N/mm)								
PA Stiffness	13.52	12.57	10.45					
30-90N	(0.95)	(1.39)	(0.6)					
(K N/mm)								
Respiratory EMG	0.166	0.103	0.384	0.481	0.067	0.079	0.096	0.137
	(0.09)	(0.02)	(0.07)	(0.06)	(0.02)	(0.02)	(0.03)	(0.03)
Abdominal EMG	0.515	0.491	0.860	0.679	0.528	0.489	0.509	0.469
	(0.27)	(0.26)	(0.41)	(0.37)	(0.26)	(0.26)	(0.29)	(0.28)

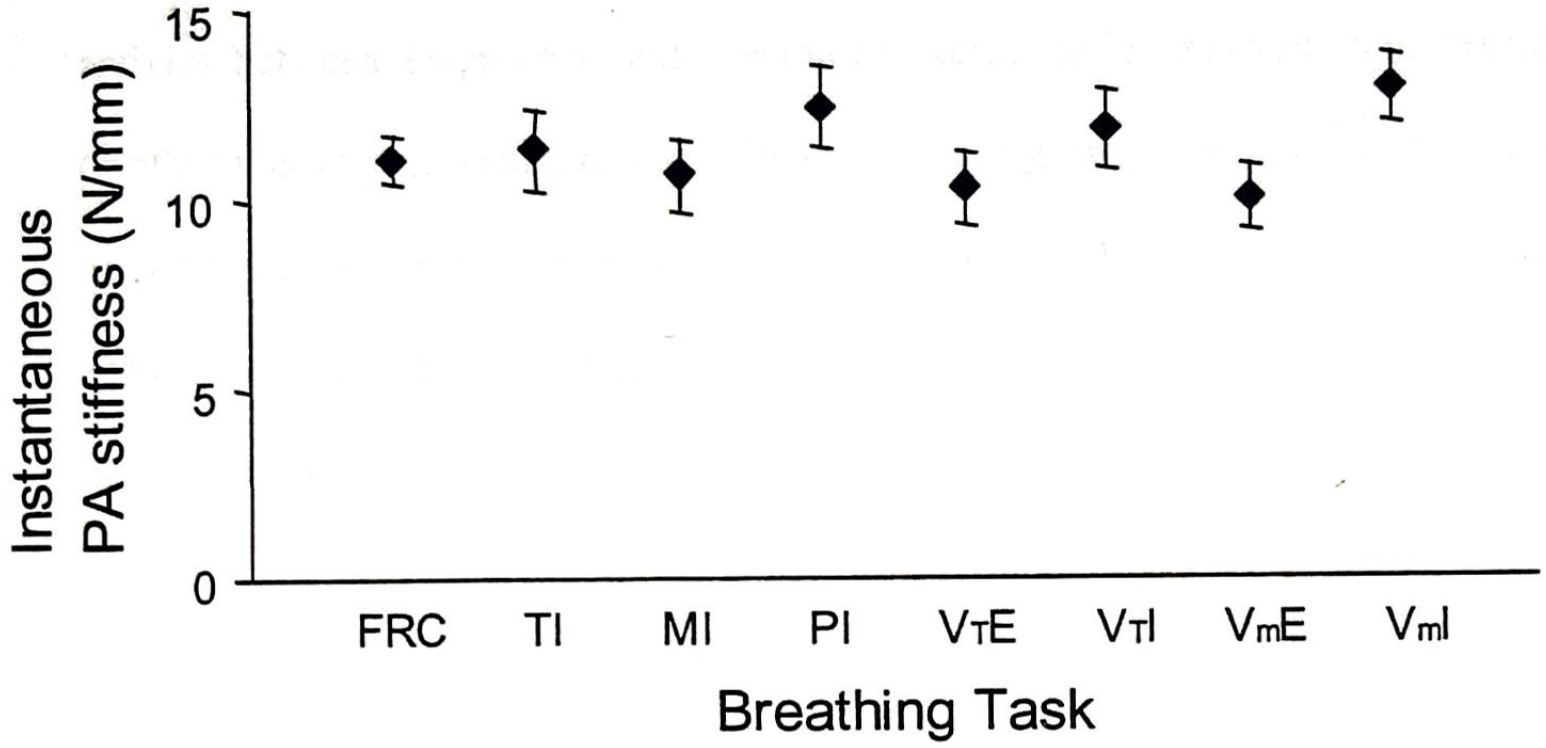

Figure 5.1. Mean instantaneous stiffness (SE) for the various breathing tasks. There is no difference between functional residual capacity (FRC), tidal inspiration (TI) and maximal inspiration (MI) however stiffness is greater for prolonged inspiration (PI). Stiffness for dynamic tidal inspiration (V_TI) and mechanical inspiration (V_MI) is greater than for dynamic tidal expiration (V_TE) and mechanical expiration (V_ME).

Comparison of respiratory and abdominal muscle EMG between different breathing conditions

When respiratory muscle EMG was compared across all breathing conditions there was no difference between breath held at FRC and tidal inspiration (P=0.46) and breath held at FRC and maximal inspiration (P=0.07) (Figure 5.2). However there was an increase in respiratory EMG with prolonged inspiration compared to FRC

(P<0.005). Prolonged inspiration and maximal inspiration produced higher respiratory EMG than either dynamic tidal breathing (P<0.005) or assisted mechanical ventilation (P<0.05). There was no difference in respiratory muscle activity between inspiratory and expiratory cycles during relaxed tidal breathing (P=0.06), however, respiratory muscle activity was greater during inspiratory cycles than expiratory cycles for mechanical ventilation (P=0.018). Respiratory muscle activity was also greater during inspiratory cycles of assisted mechanical ventilation than those of tidal breathing (P=0.024). There was no difference in respiratory muscle activity for the expiratory cycles of tidal breathing and mechanical ventilation (P=0.31) (Figure 5.2).

Abdominal EMG was greatest during maximal inspiration (Table 5.2, Figure 5.3). When compared across the different breathing conditions abdominal EMG was greater at maximal inspiration than at FRC (P=0.046). Abdominal EMG recorded with breath held at maximal inspiration was greater with inspiration than expiration during dynamic tidal and mechanical breathing (P<0.05). There was no change in abdominal EMG between breath held at FRC and breath held at tidal inspiration (P=0.57) or FRC and dynamic breathing (P>0.4). There was no difference in abdominal EMG between inspiratory and expiratory cycles during tidal breathing (P=0.123) or assisted mechanical ventilation (P=0.152). Abdominal EMG was also no different during inspiratory cycles of assisted mechanical ventilation than those of tidal breathing (P=0.51). There was no difference in abdominal EMG for the expiratory cycles of tidal breathing and mechanical ventilation (P=0.51) (Fig 5.3).

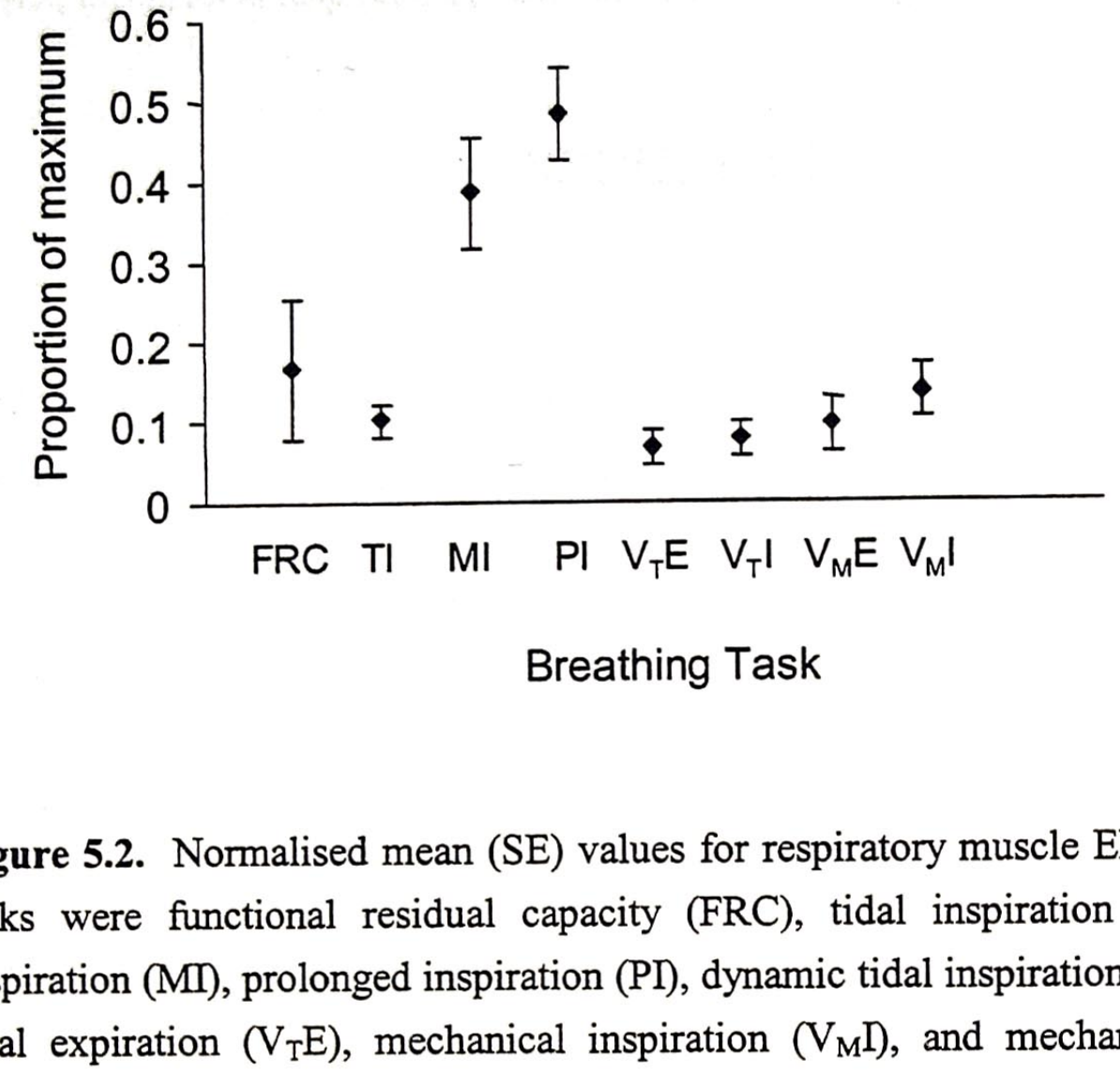

Figure 5.2. Normalised mean (SE) values for respiratory muscle EMG. Breathing tasks were functional residual capacity (FRC), tidal inspiration (TI), maximal inspiration (MI), prolonged inspiration (PI), dynamic tidal inspiration (V_TI), dynamic tidal expiration (V_TE), mechanical inspiration (V_MI), and mechanical expiration (V_ME).

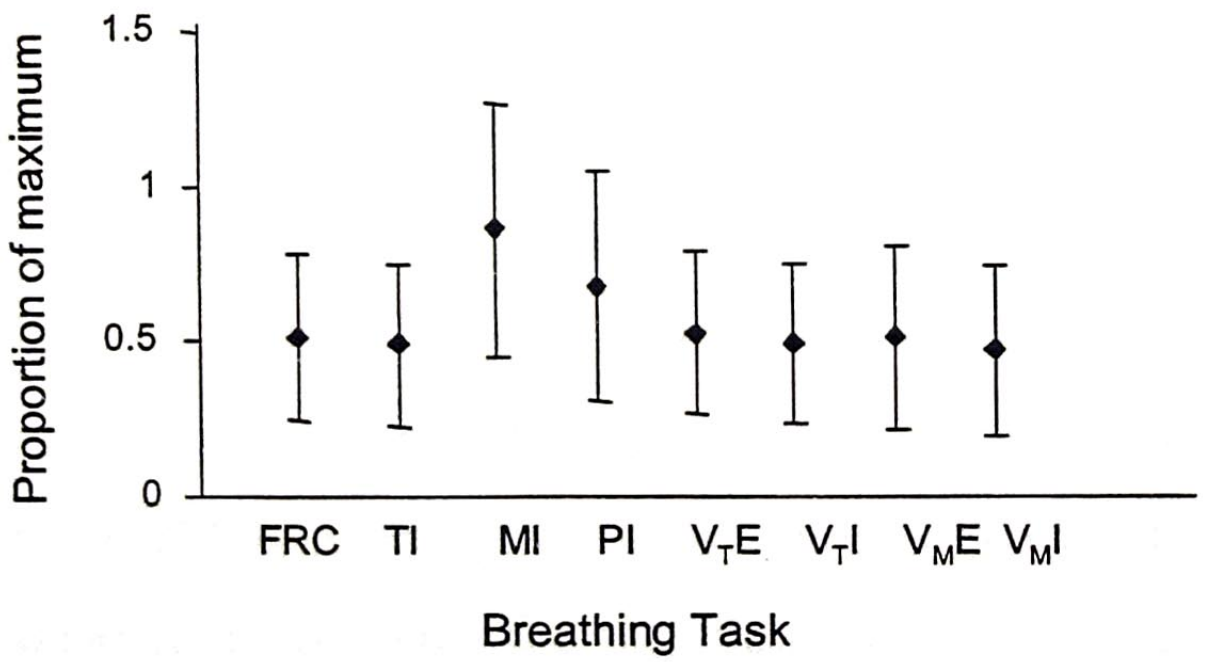

Figure 5.3. Normalised mean (SE) values for abdominal EMG. Breathing tasks were functional residual capacity (FRC), tidal inspiration (TI), maximal inspiration (MI),

prolonged inspiration (PI), dynamic tidal inspiration (V_TI), dynamic tidal expiration (V_TE), mechanical inspiration (V_MI), and mechanical expiration (V_ME).

DISCUSSION

This study describes a new method to determine lumbar PA stiffness (instantaneous PA stiffness). Instantaneous PA stiffness excellent reliability for measuring lumbar PA stiffness during a variety of static and dynamic conditions of respiration. In previous studies, PA stiffness has been calculated as the slope of a regression slope fitted to the force displacement curve between 30 and 90N. However, in the current study a consistent level of force was not achieved during all cycles of force application during the dynamic breathing tasks. Therefore, it was necessary to investigate an alternate method of calculating stiffness. Instantaneous PA stiffness is appropriate for measuring stiffness in a variety of static and dynamic conditions and particularly when the maximum level of force applied to the spine is variable.

Instantaneous PA stiffness values were highly correlated with PA stiffness from 30-90N for the static conditions. The high correlation between the two procedures indicates that either method will return a valid result, however calculations made for instantaneous PA stiffness will be lower than with the 30-90N method eg at FRC with the 30-90N method K was 13.52 N/mm compared with 11.0 N/mm for instantaneous PA stiffness. Therefore, comparisons between data need to take into account the difference in PA stiffness between the two methods. In addition, when reliability was compared between the two methods for within-test static data excellent reliability was demonstrated for both methods. However, when comparing

the two methods for the average of cycles there was excellent test-retest reliability for TI and MI but poorer reliability for FRC. The PA stiffness values at FRC were determined using the limits of force between 30 and 90N, whereas for the other tasks PA stiffness was calculated between 30 and 70N as not all cycles achieved 90N of force. Therefore the PA stiffness values at FRC using the 30-90N method were higher than with the instantaneous method ($P<0.0001$), accounting for the lower ICC value at FRC. Lumbar PA stiffness would be expected to be greater when including the higher levels of force as stiffness values increase with increasing force (Latimer et al. 1998). When PA stiffness at FRC is calculated between 30 and 70 N the ICC (2,1) value is 0.89. Therefore, reliability of the measure is dependent on the range of force used in calculation.

Lumbar PA stiffness at L4 is not related to lung volume but is affected by some breathing tasks. These results are consistent with those of an earlier study that demonstrated no difference in stiffness between FRC and end inspiration (Beaumont et al. 1991), however another study found that PA stiffness was lower during breath holding at maximal inspiration than at FRC (Keaveney et al. 1989). In the current study, subjects were instructed to breathe out to FRC and then breathe in to either a normal tidal inspiration or a maximal inspiration. Subjects were not instructed whether they should hold their glottis open or closed, however it is likely most people automatically close their glottis when holding their breath. When the glottis is closed the diaphragm does not need to be active to hold the lung volume. One explanation for stiffness at FRC being no different from stiffness during maximal inspiration is the subjects held their breath with the glottis is closed allowing the diaphragm to relax. This explanation could also account for decreased PA stiffness

during maximal inspiration reported in the earlier study (Keaveney et al. 1989). The results of the current study show that there was no difference in respiratory muscle activity between FRC and maximal inspiration. The fact that PA stiffness was often greater during tasks when there was greater respiratory muscle activity lends support to the hypothesis that diaphragm activity may contribute to PA stiffness. Another factor that may have influenced the lack of significant difference between PA stiffness at FRC and MI is the relatively small sample size used in this study.

Postero-anterior stiffness at L4 was greater during inspiration than expiration when the subject performed tidal breathing. Beaumont et al. (1991) found there was no significant difference between the inspiratory and expiratory phases of tidal breathing, although they observed a trend that spinal stiffness was greater during inspiration. The difference in results between the current study and the study by Beaumont et al. (1991) could be due to the method of data analysis or force application. In the current study only cycles observed to be wholly inspiratory or expiratory were included in the analysis. Also the maximum force of 110N is relatively low, however, the maximum force in the study by Beaumont et al. (1991) is not described. It was observed that a low force enabled the subjects to maintain a normal pattern of tidal breathing whereas this could not be achieved with higher levels of force. If Beaumont et al. (1991) applied higher levels of force in their study it may have been more difficult for subjects to maintain a normal breathing rhythm. In addition, the instantaneous method of analysis for spinal stiffness was used because tidal breathing is dynamic and causes movement of the spine so that each loading cycle did not reach the same maximum force. This method of stiffness calculation may have had increased sensitivity to changes in stiffness.

Lumbar PA stiffness was greater when subjects performed prolonged inspiration with an incentive spirometer. During prolonged inspiration continuous activity was observed from the respiratory muscle EMG and was greater than in the other tasks performed (Table 5.2). While it is possible that these electrodes recorded activity from abdominal muscles, intercostal muscles and the diaphragm, it may be reasonable to infer that activity is predominantly from the diaphragm due to the electrode placement (Osmond 1985) and activation of the diaphragm during inspiration (Loring and De Troyer 1985). In addition, abdominal EMG did not increase significantly from FRC (Table 5.2), which suggests abdominal muscle activity does not make a major contribution to the respiratory muscle signal. This study found that an increase in lung volume alone does not increase PA stiffness. Therefore, increases in PA stiffness during prolonged inspiration are likely to be due to respiratory muscle activity (including diaphragm) or the resulting changes in intra-abdominal pressure. The diaphragm is thought to play a role in increasing stiffness of the lumbar spine either by the direct attachments to the lumbar vertebrae or by increasing intra-abdominal pressure to assist with stability of the spine (Hodges and Gandevia 2000a).

Tidal breathing and assisted ventilation both demonstrated the same pattern of behaviour of PA stiffness i.e. that the inspiratory phases were stiffer than the expiratory phases. This difference between inspiration and expiration would be expected for tidal breathing, as the diaphragm should be more active during tidal inspiration than tidal expiration. The finding that stiffness during inspiration with assisted mechanical ventilation was greater than with tidal breathing was

unexpected. The prediction that the diaphragm would be more relaxed during assisted ventilation is in accordance with the results which indicated that there was no increase in respiratory EMG during mechanical breathing. Therefore, the reason for increased stiffness during assisted ventilation is unclear but could relate to the effect of application of positive pressure and the fact that the respiratory drive of these subjects was not depressed. There was phasic activity of the respiratory muscle EMG during assisted ventilation, which was higher than during tidal breathing. Therefore, even though this finding was unexpected the increase in stiffness for inspiratory phases of assisted ventilation is consistent with other findings of this study where increases in stiffness often corresponded to increased respiratory muscle activity. The findings that increased respiratory muscle activity (which most likely reflects diaphragm activity) was generally associated with increases in PA stiffness suggest that activity of the diaphragm is likely to be a contributing factor to PA stiffness.

The PA stiffness value represents the combined response of a variety of structures rather than an actual measure of stiffness at an individual vertebral segment. When a PA force is applied to the spine the resulting movement includes anterior rotation of the pelvis, extension of the spine, compression of the ribcage and abdominal contents and only a small amount of intervertebral movement (Lee et al. 1996). The only way to obtain a true measure of intervertebral PA stiffness would involve invasive stabilisation of the spine. Therefore the mechanical device provides a non invasive indication of PA stiffness of the spine. The dynamic breathing tasks investigated in this study during which the spine moved up and down with the breathing cycle may further compound the limitations of this measure. Movement of

the spine may account for some of the increase in stiffness in the dynamic tasks, however, the prolonged inspiration task indicated that stiffness increases are also related to increasing respiratory muscle (diaphragm) activity. Therefore, it is likely that at least some of the change in stiffness observed in the inspiratory cycles of the dynamic tests was due to the influence of muscle activity and not only movement of the spine.

It was proposed that the diaphragm may contribute to PA stiffness. In this study PA forces were applied at L4 where there is no direct attachment of the diaphragm, therefore, any effect on stiffness is more likely to be due to the indirect action of increasing intra-abdominal pressure. Lumbar PA stiffness may be different if the PA forces are applied at different spinal levels. If PA forces were applied to the upper lumbar spine were the crural diaphragm is attached directly to the vertebral bodies the increase in stiffness could be greater as the diaphragm may have a more direct action in stiffening the spine. In addition, when forces are applied higher in the spine the rigidity of the thorax will limit excursion of the spine in a PA direction, which is likely to further increase stiffness. When a PA force is applied over the lower lumbar spine it is likely to increase intra-abdominal pressure. Furthermore, during maximal inspiration intra-abdominal and intra-thoracic pressures should be increased, and these increases in pressures could contribute to increases in PA stiffness.

Clinical Implications

The breathing cycle affects L4 PA stiffness indicating that clinicians should be aware that their judgment of stiffness might be affected by the phase of the breathing cycle during stiffness assessment. This study confirms that the procedure using standardisation of breathing for research purposes is necessary to measure stiffness. However, for testing during static tasks the exact volume may not make a difference if breath holding above FRC.

This study also suggests that activity of the diaphragm is one possible factor that contributes to PA stiffness and that stiffness is not dependent on lung volume. Further research is currently being undertaken to investigate the role of the diaphragm and intra-abdominal pressure in contributing to lumbar PA stiffness.

CHAPTER SIX

TRUNK MUSCLE ACTIVITY AND INTRA-ABDOMINAL PRESSURE IN RESPONSE TO VARIED RESPIRATORY EFFORT CHANGES SPINAL STIFFNESS

ABSTRACT

Postero-anterior stiffness of the lumbar spine is influenced by many factors which include trunk muscle activity and intra-abdominal pressure (IAP). The purpose of this study was to determine whether stiffness varies throughout the respiratory cycle when these factors are modulated.

Stiffness at L4 and L2 was measured from the force and displacement properties when a postero-anterior force was applied to the spinous processes of the L4 and L2 vertebrae using a mechanical device. Electromyographic (EMG) recordings were made from erector spinae (ES) at L2 and L4, abdominal muscles and chest wall with surface electrodes. IAP was measured with a pressure transducer inserted into the stomach via the nose. Stiffness was measured with the lung volume held at the extremes of normal tidal volume and at volumes above and below tidal volume.

Stiffness at L4 and L2 was significantly increased above values recorded at functional residual capacity with both inspiratory and expiratory efforts. The magnitude of the increase was related to the respiratory effort and was greatest during maximum expiration. Across respiratory tasks, stiffness was related to changes in both pressure and EMG amplitude of the trunk muscles. Furthermore the increase in stiffness with maximal inspiration was greater when force was applied to L2 than L4.

The results indicate that stiffness of the spine is modulated with respiratory efforts and that this modulation is due to contributions from factors such as activity of the trunk muscles (including the diaphragm) and pressure in the abdominal cavity. In addition, the data suggest that the diaphragm may affect the stiffness of the spine via the attachment of its crural fibres to the upper lumbar vertebrae.

INTRODUCTION

Stiffness of the spine is affected by many factors including muscle activity, stiffness of the surrounding soft tissues (e.g. ligament and muscle) and structural factors (e.g. orientation of the facet joints) (Lee et al. 1996). The affect of muscle activity on stiffness has been investigated extensively (Cholewicki et al. 1999b; Chapter Three, Lee et al. 1993) and erector spinae activity of 10 % of a maximal voluntary contraction increases stiffness of the spine to a postero-anterior force by 12% (Chapter Three). As activity of the trunk muscles is modulated with respiration, we hypothesised that spinal stiffness would be modulated throughout the respiratory cycle. However, the relative contribution of inspiratory and expiratory muscles to spinal stiffness has not been determined.

Several other parameters complicate the effect of respiration on the stiffness of the spine. Firstly, intra-abdominal pressure (IAP) varies during respiration. In quiet respiration IAP increases during inspiration due to contraction of the diaphragm, and then falls during expiration (Campbell and Green 1953). However, when expiratory volume or flow is increased, IAP is also elevated during expiration as a result of abdominal muscle activity. It has been suggested that elevated IAP may increase spinal stiffness as a result of tensioning the lumbar spine (Daggfeldt and Thorstensson 1997), generation of a posterior shear force against the lumbar spine (Aspden 1987), decreasing the compliance of the abdominal contents (McGill and Norman 1987) or indirectly by the increasing the tension of the thoracolumbar fascia (Tesh et al. 1987). Secondly, stiffness may be influenced directly by contraction of

the diaphragm due to the attachment of the crurae to the lumbar vertebrae. In the majority of people the crurae extend to L3 on the right and L2 on the left (Williams et al. 1995). Thus diaphragm contraction may have a greater effect on stiffness at the upper lumbar levels.

The aims of the present study were; (i) to determine whether stiffness of the spine is modulated during quiet respiration, (ii) to compare the effect of inspiration and expiration above and below tidal volume on spinal stiffness, (iii) to investigate the relationship between changes in spinal stiffness, muscle activity and intra-abdominal pressure; and (iv) to investigate whether spinal stiffness was different at L2 and L4 (ie at a vertebral level that involves the crural diaphragm attachment and at one below the attachment of the crural diaphragm).

METHODS

Subjects

Eight subjects of mean (SD) age, height and weight of 39 (9) years, 1.78 (0.06) m, and 76 (12) kg, respectively, volunteered for this study. Subjects were excluded if they had a history of low back pain, or any significant neurological, respiratory or cardiovascular disease. The study was approved by the institutional Human Ethics Committee and written informed consent was obtained.

Measurement of spinal stiffness (response of the spine to postero-anterior pressure)

Postero-anterior stiffness of the lumbar spine at L4 and L2 was measured using a mechanical device that recorded the force required to achieve a set postero-anterior displacement of an indentor applied over the spinous process. The mechanical device, which consists of a motor that drives an indentor to a set distance with variable force, was previously described in Chapters One and Two. The indentor is positioned in contact with the skin over the spinous process of the target vertebra and force is applied in a cyclical manner (1 Hz) for five repetitions in the postero-anterior direction. Force and displacement are measured with a strain gauge and linear potentiometer, respectively. Force was applied at an angle of 4.5° in a caudal direction at L4 and at 11.5° in a cephalad direction at L2 (Stagnara et al. 1982). The range of displacement was selected to obtain a linear relationship between force and displacement. The maximal applied force was set at 150N (Latimer et al. 1996a). Measurement of postero-anterior stiffness by this device has good test-retest reliability and is highly accurate (Latimer et al. 1996a).

Intra-abdominal pressure

Intra-abdominal and intra-thoracic pressures were measured using a pair of thin-film strain gauge transducers (Gaeltec, UK) inserted into the nose via the stomach. One transducer was positioned in the stomach to record gastric pressure (Pga) and the other above the diaphragm to record oesophageal pressure (Pes). The optimal

position of the tube was confirmed by opposite changes in Pga and Pes with a sniff and a mueller manoeuvre. Once the tube was located in the desired position it was taped to the nose.

Respiratory measurements

Airflow was measured with a pneumotachograph (Hans Rudolf, MO, USA) and integrated on line to be recorded as volume. Feedback of volume was displayed on an oscilloscope. Respiratory movement of the rib cage was measured with an inductance plethysmograph (Respitrace, Ambulatory monitoring, NY, USA) placed around the chest.

Electromyography

Electromyographic (EMG) recordings of the trunk muscle were made using silver/silver chloride surface electrodes. Pairs of electrodes were placed over the erector spinae muscles ~4cm lateral to the spinous processes of L2 and L4 in parallel with the muscle fascicles, and over the lateral abdomen midway between the rib cage and iliac crest in an oblique direction. An additional pair of electrodes was placed over the 7^{th} and 8^{th} intercostal spaces in the midclavicular line to record EMG activity from the diaphragm. As this electrode records activity from muscles other than the diaphragm, including the intercostal and abdominal muscles it was referred to as 'chest wall' EMG. EMG data were filtered between 53 Hz – 1 kHz and sampled at 2 kHz using a CED 1401 and Spike2 software (Cambridge Electronic Design, UK).

Experimental procedure

Subjects were positioned in prone on a rigid plinth with their arms by their sides and neck supported in neutral. The pelvis and ribcage were supported on blocks so that there was no external pressure on the abdomen and a belt was also placed around the ribcage to limit changes in rib position and movement with different lung volumes. The spinous processes of the L2 and L4 vertebrae were identified by palpation of the spine and marked with a pen. The foot of the indentor was placed over the spinous process of the target vertebra (L4 or L2 for separate trials) and 4 cycles of force were applied to precondition the spine. Stiffness testing was then carried by applying five loading cycles of force at 1 Hz to the target vertebra (Figure 6.1).

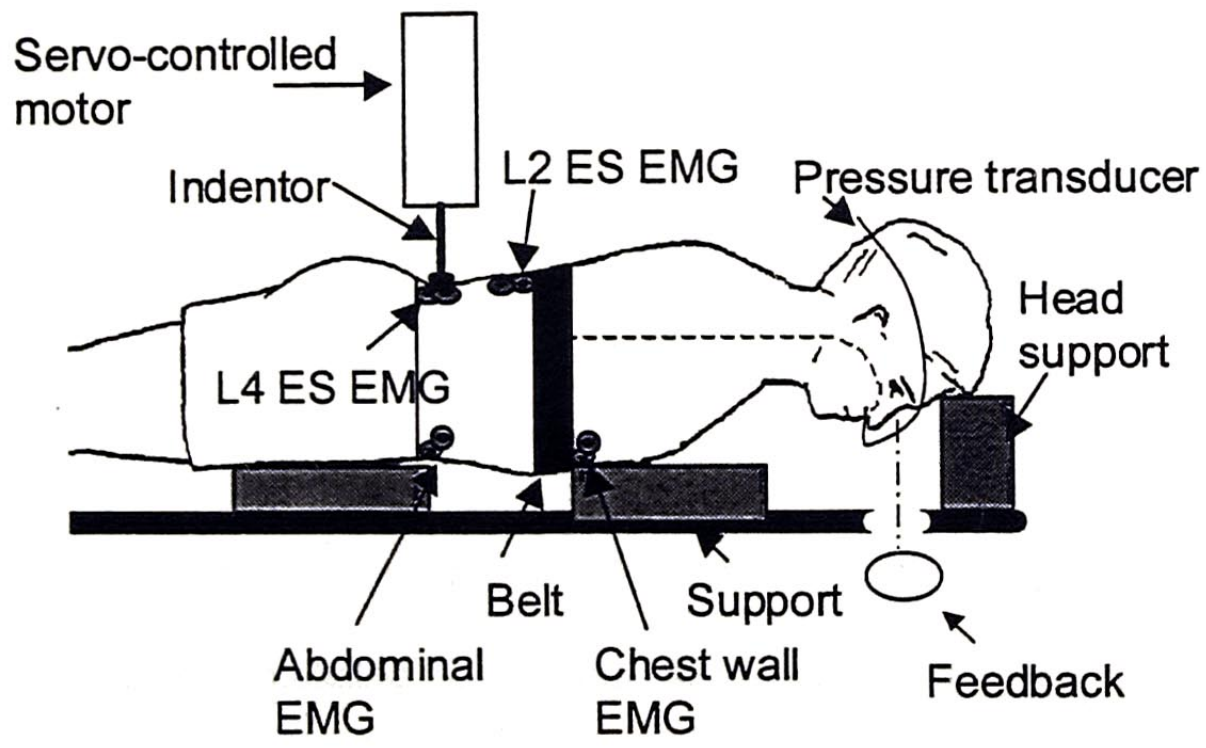

Figure 6.1. Subjects lay prone on the testing plinth with the mechanical device positioned over their L4 spinous process

The response of the lumbar spine (at L4 and L2) to PA pressure was assessed while subjects performed a series of respiratory tasks (Figure 6.2) in pseudo-random order. In all tasks lung volume was held with the glottis open. The tasks were (i) lung volume held at functional residual capacity (FRC), (ii) lung volume held at end tidal inspiratory volume (Vt), (iii) lung volume held at 50% of maximal inspiratory volume (50% Insp), (iv) lung volume held at total lung capacity (TLC), (v) lung volume held at 50% of the volume of expiration from FRC to maximal expiration (50% Exp), and (vi) lung volume held at residual volume (RV).

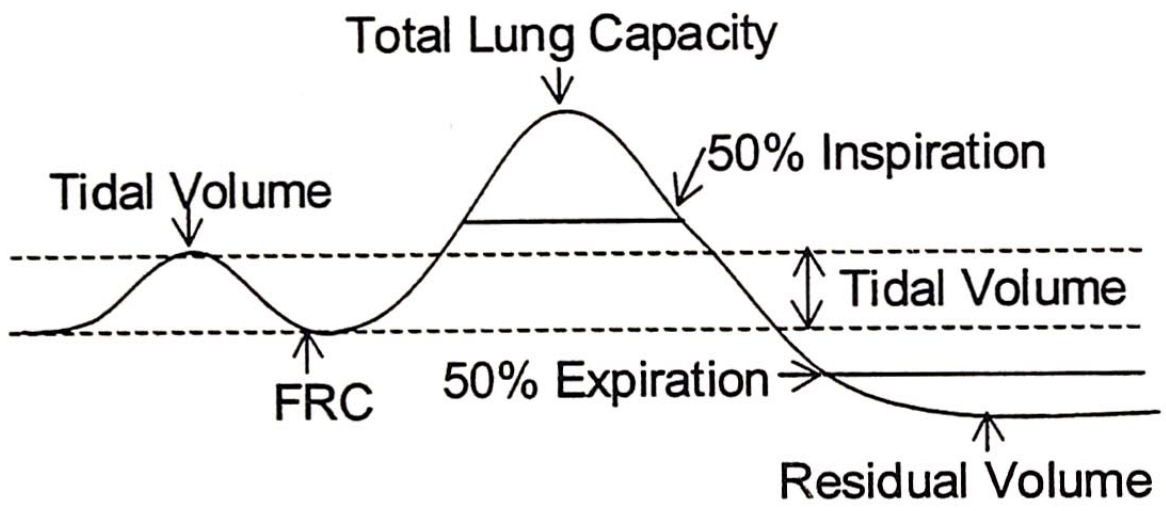

Figure 6.2. Points of measurements in the respiratory cycle.

In an additional trial the measure at TLC was repeated with the glottis held closed (TLCGC). This was included to confirm that changes in spinal stiffness that occurred with inspiratory efforts were due to the associated increase in muscle activity and Pga and not the effects of increased lung and rib cage volume. Closure of the glottis allows lung volume to be maintained, but allows the diaphragm and other inspiratory muscles to relax. Furthermore, Pga is decreased when the glottis is closed as a result of equalisation of the pressure in the abdominal and thoracic

cavities. Identification of small fluctuations in the airflow (due to cardiac movements) confirmed that the glottis had remained open during the procedure. Subjects practised each task while maintaining an open glottis. Feedback of the required lung volume and targets were provided for each task and subjects were instructed to maintain the volume during the stiffness measurement.

Data Analysis

Force-displacement plots were generated from the output of the stiffness device. Spinal stiffness was calculated as the slope of a regression line fitted to the force displacement curve between 50 and 110 Newtons using a custom designed computer program (Fig. 6.3). This range of force was chosen as the force displacement curves were linear in this range. Other studies that have measured lumbar stiffness have shown good reliability when stiffness is calculated in the linear range of the force displacement curve (Latimer et al, 1996a). Stiffness values were averaged over the last three consecutive cycles in each test. The first cycle was not used as it is more variable and inconsistent with stiffness recorded during subsequent cycles (Chapter Two). Stiffness values were expressed as a proportion of the stiffness at FRC.

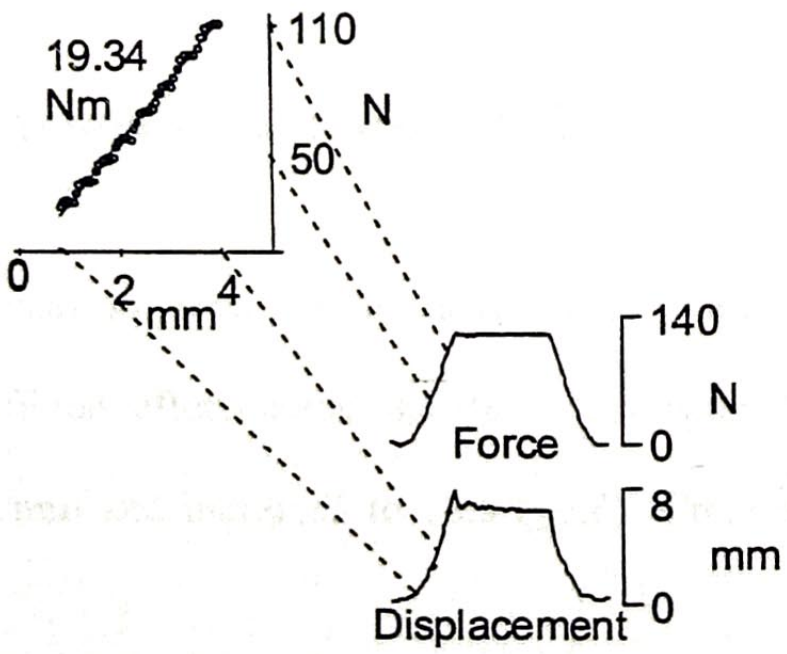

Figure 6.3. Stiffness is calculated by the slope of a regression line fitted to a force displacement curve between 50 and 110 Newtons.

Root mean square (RMS) EMG amplitude was measured for a 1-second epoch (i.e. an entire loading cycle) during the application of the PA pressure and normalised as a proportion of the value at FRC. The mean amplitude of Pga and Pes was recorded during the same 1-second as the EMG data and were normalised as a proportion of the value at FRC. Transdiaphragmatic pressure (Pdi) was calculated as the difference between Pga and Pes.

Statistics

Stiffness at L4 was compared between respiratory tasks and vertebral levels using a one-way analysis of variance (ANOVA) and Duncan's multiple range test. Pearson's r was calculated to determine the correlation between mean stiffness at L4 and the mean values for EMG and pressure. The amplitude of each parameter between respiratory conditions was compared with separate ANOVAs and Duncan's multiple range test. The alpha level was set at 0.05.

RESULTS

Changes in stiffness at L4

Spinal stiffness was measured at L4 during respiratory manoeuvres representing different efforts across the respiratory cycle. Stiffness for L4 at FRC was 15.3(±3.2) N/mm and increased to 18.5 (±5.0) N/mm at TLC and 23.9 (±7.3) N/mm at RV.

Representative force displacement curves are shown in Figure 6.4. There was no significant change in the stiffness at L4 between FRC and the end of a tidal inspiration (with the glottis open) (P=0.111). However, there was a trend for the stiffness to increase above FRC. When measurements were made during tasks in which lung volume was held above (inspiratory) and below (expiratory) the normal tidal volume, the stiffness at L4 was increased above the values recorded at FRC (Table 6.1) (P<0.05). Stiffness at L4 was increased from the FRC value by a factor of 1.22 for 50% inspiration, 1.24 for TLC and 1.53 for RV (Figure 6.5). The increase in stiffness was greater at RV than at TLC (P<0.005).

Table 6.1. Mean Values (SD) for lumbar stiffness (K in N/mm) at L4 and L2 during all respiratory tasks. The respiratory tasks measured were functional residual capacity (FRC), end tidal inspiration (Vt), 50% of maximal inspiration (50% Insp), total lung capacity (TLC), total lung capacity with the glottis closed (TLCGC), 50% of maximal expiration (50% Exp) and residual volume (RV).

	FRC	Vt	50% Insp	TLC	TLCGC	50% Exp	RV
K L4	15.3 (3.2)	18.4 (3.6)	18.6 (4.1)	18.5 (5.0)	15.6 (3.7)	19.4 (6.2)	23.9 (7.3)
K L2	14.9 (1.8)	16.1 (2.5)	18.3 (3.2)	19.9 (2.1)	15.1 (3.4)	17.0 (3.9)	24.9 (9.0)

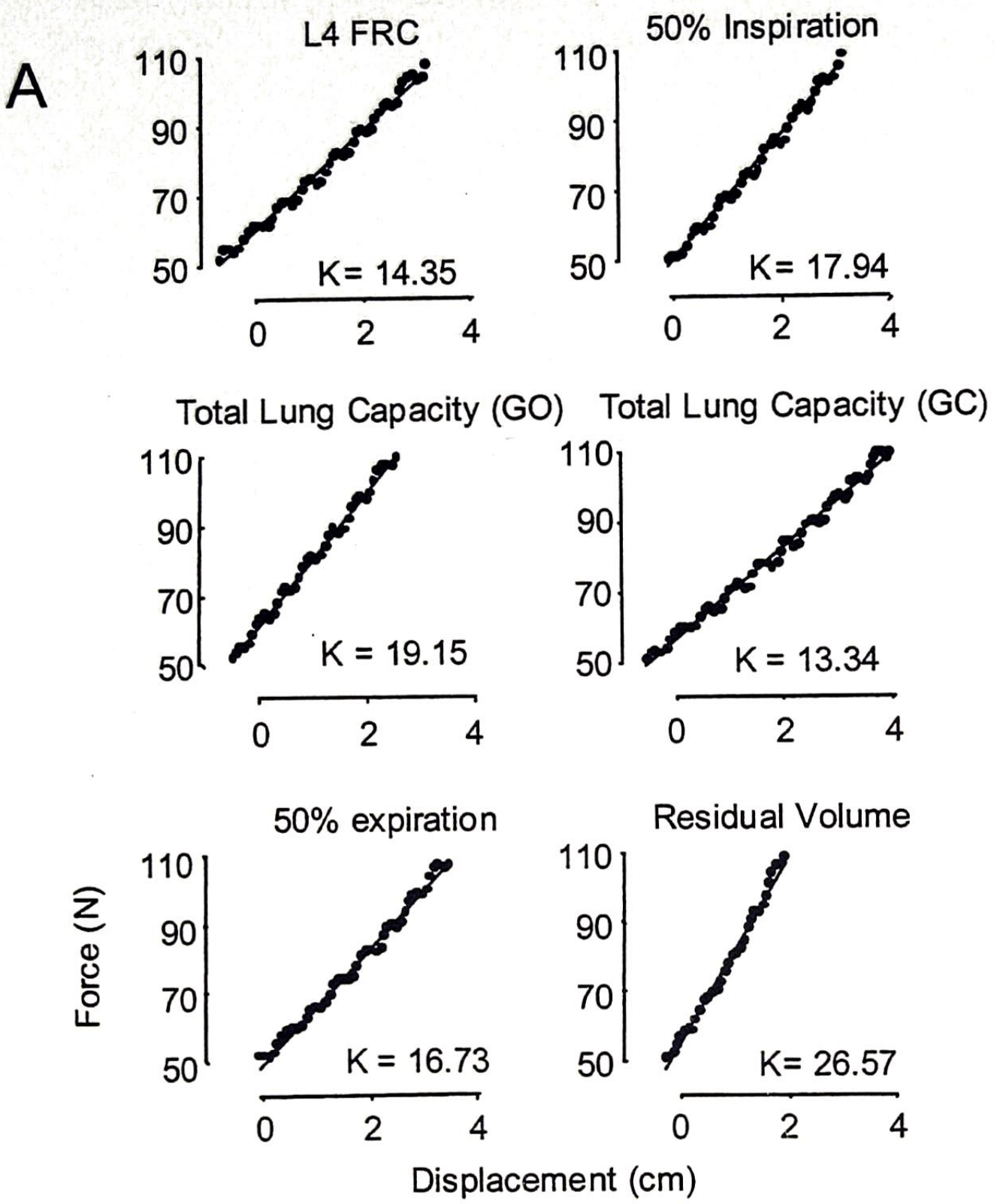

Figure 6.4 Force displacement curves for the respiratory tasks. The force displacement curves demonstrate the stiffness (coefficient K) for the respiratory tasks. Coefficient K is the slope of the regression line fitted to the force displacement curve between 50 and 100 Newtons. There was excellent correlation between the force displacement curve and the regression line for each task (R^2=0.99).

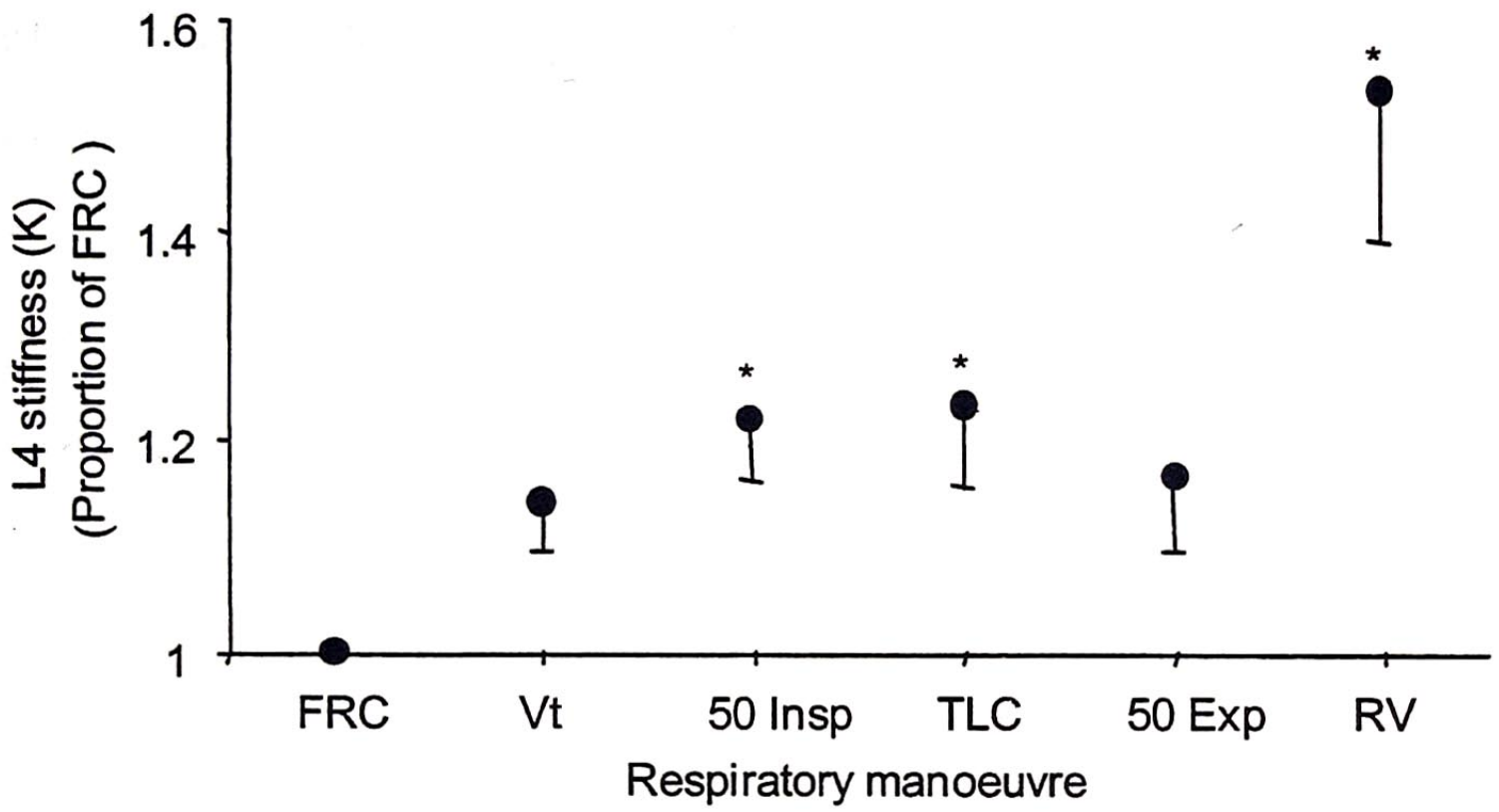

Figure 6.5. Relative increases in L4 stiffness with respiratory tasks. Mean (± SD) stiffness expressed as a proportion of the value for FRC for all respiratory manoeuvres. The values that demonstrated a significant difference from FRC are indicated by the asterisk.

Relationship between changes in stiffness, and EMG and pressure

To investigate the mechanism for the change in spinal stiffness with increased respiratory efforts we evaluated the relationship between stiffness and changes in EMG and pressure. Raw data for a representative subject are shown for each condition in Figures 6.6, 6.7 and 6.8. All EMG and pressure measurements were positively correlated with stiffness across the range of tasks (Fig. 6.9). Consistent with this data, when the amplitude of each parameter was compared between respiratory tasks the general trend was similar to that identified for changes in L4 stiffness, i.e. the values were increased above those recorded at FRC for the tasks in which lung volume was held above or below the normal tidal volume (Fig. 6.10). There were several exceptions to this general trend. EMG recorded with electrodes over L4 ES, AB and the chest wall was greater than that recorded at FRC for all lung volumes ($P<0.05$). In contrast the EMG amplitude recorded with the electrode over ES at the L2 level was only increased above that recorded at FRC during the expiratory tasks ($P<0.001$). Transdiaphragmatc and abdominal pressures increased above FRC values with all tasks, but the amplitude of the increase was greater with inspiratory compared to expiratory tasks ($P<0.05$).

Stiffness at L4 during maximal inspiratory efforts with the glottis open and closed was compared to confirm that the changes in stiffness were due to the elevated EMG and pressure and not simply the change in lung volume. The data indicate that the chest wall EMG ($P<0.001$) and Pdi ($P<0.001$) were decreased with closure of the glottis, however there was no change in Pga ($P=0.48$) (Figure 6.7, Table 6.2).

Correspondingly, when stiffness was compared between glottis conditions with maximal inspiration the stiffness at L4 was greater with the glottis open (18.5 N/mm) than with the glottis closed (15.6 N/mm) ($P<0.01$) and the values recorded with the closed glottis were not different to the values at FRC (15.3 N/mm) (Table 2) ($P=0.704$). There was no change in AB (P=0.51) or ES EMG (L2:$P=0.36$, L4:$P=0.25$) between conditions (Figure 6.7, Table 6.2). This suggests that changes in chest wall EMG and Pdi which are both indicators of activity of the diaphragm (in the absence of change in ES and AB EMG) are sufficient to influence L4 stiffness.

Table 6.2. Mean values (SD) during testing at L4 for all parameters when the lung volume was held at functional residual capacity (FRC), total lung capacity with the glottis open (TLC) and total lung capacity with the glottis closed (TLCGC). Stiffness and pressure data are presented as absolute values and EMG is normalised as a proportion of the values recorded at FRC.

	FRC	TLC	TLCGC
K L4	15.3 (3.2)	18.5 (5.0)	15.6 (3.7)
L4 ES EMG	1	3.3 (2.8)	2.5 (3.7)
L2 ES EMG	1	2.1 (1.2)	1.5 (0.9)
AB EMG	1	3.2 (2.9)	1.9 (1.2)
Chest wall EMG	1	5.1 (3.1)	2.6 (2.0)
Pga	24.8 (4.6)	59.7 (24.7)	45.1 (8.1)
Pdi	16.1 (5.8)	69.5 (34.5)	21.9 (14.3)

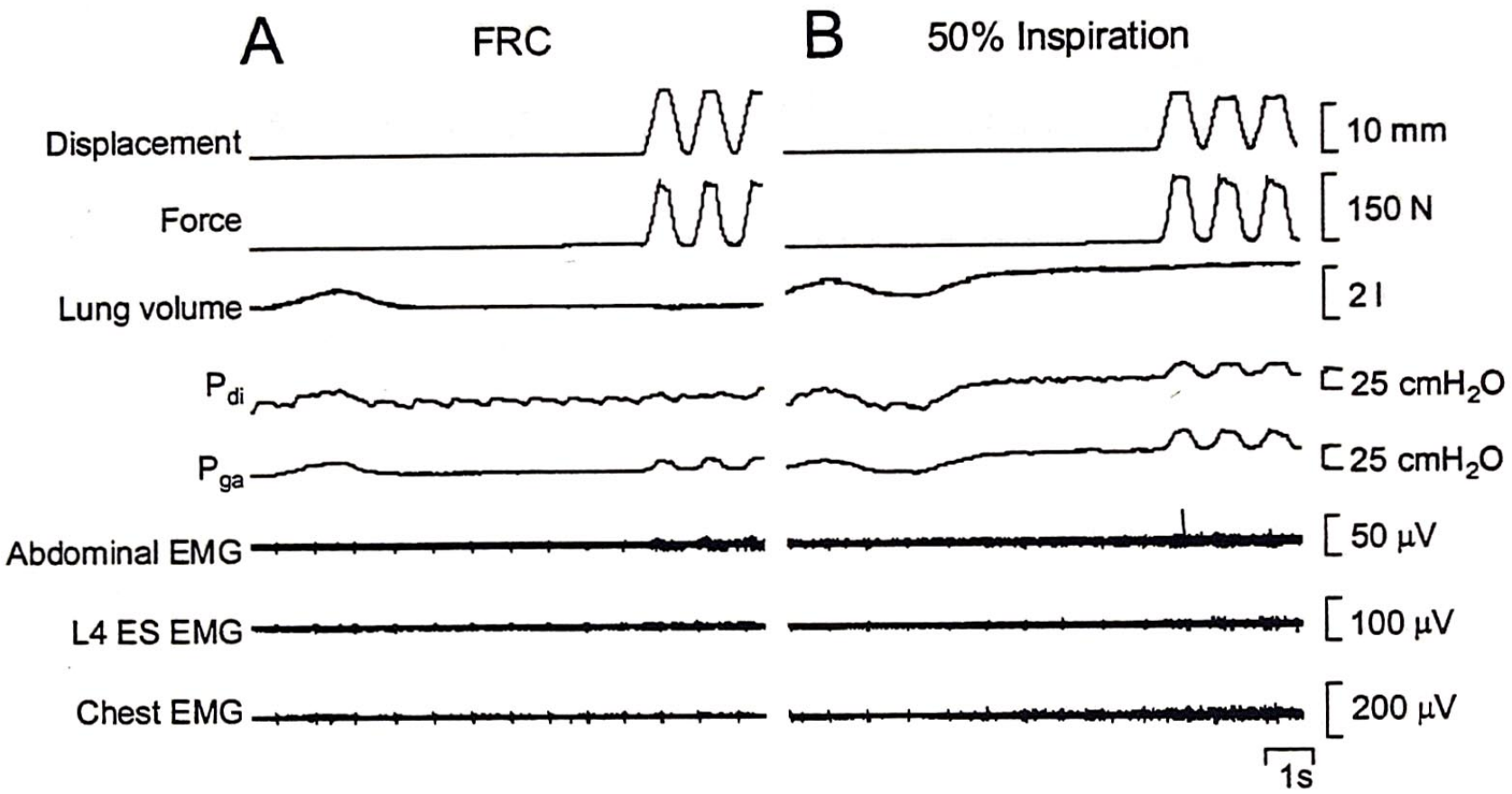

Figure 6.6. Representative data for the FRC and 50% Inspiration conditions. At FRC (A) the lung volume is low and there is minimal activity in the EMG. There is also only a slight increase in Pga and Pdi. At 50% Inspiration (B) an increase in lung volume is apparent and stiffness has increased with corresponding increases in EMG and pressures.

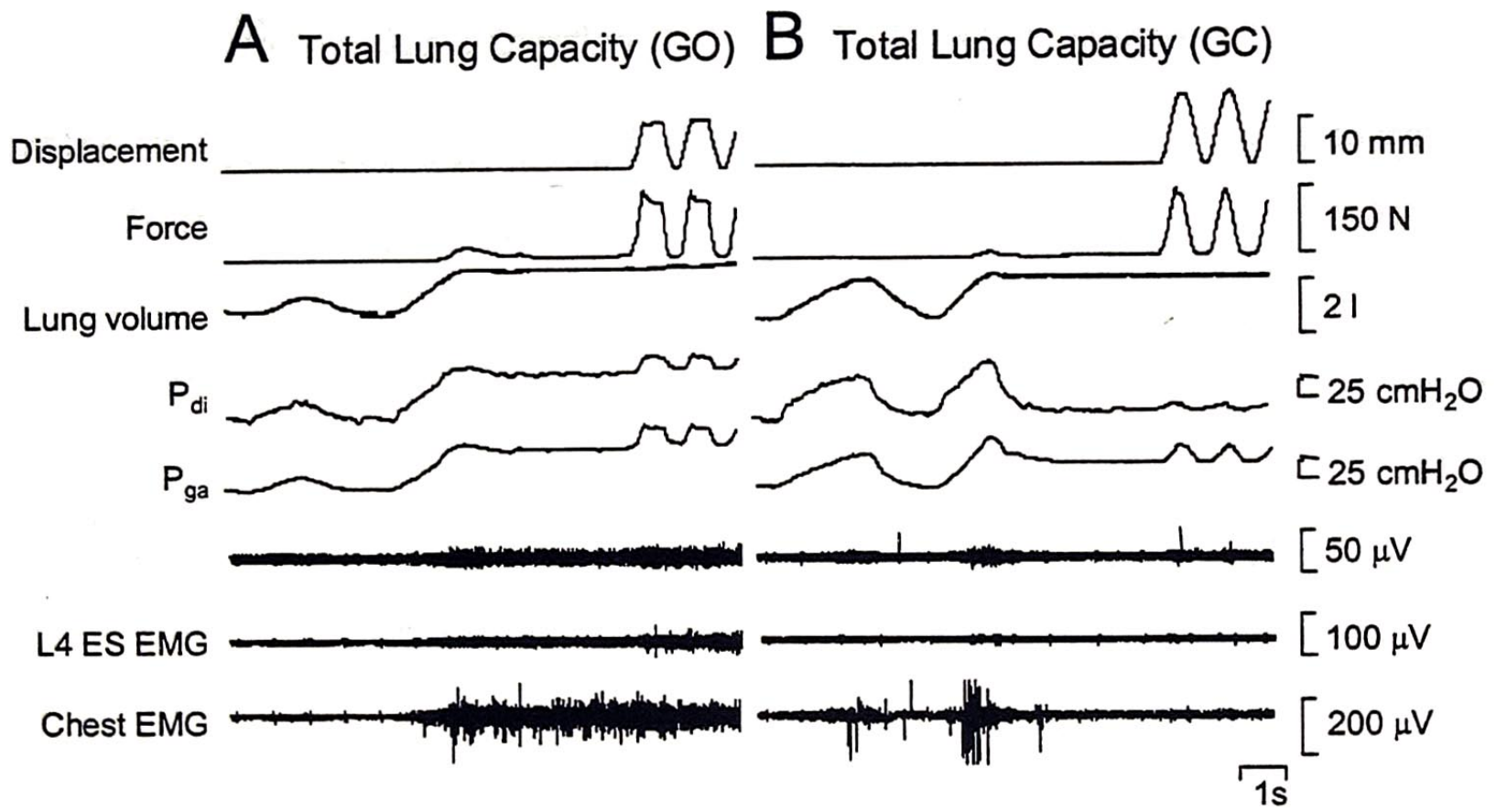

Figure 6.7. Representative data for the TLC condition with the glottis open (A) and closed (B). At TLC with the glottis open there is a marked increase in stiffness. The data show an increased lung volume along with increases in EMG and pressures during the period of loading. By comparison when testing was carried out at TLC with the glottis closed the stiffness decreased. The lung volume remains high however there is less activity in the EMG and Pdi is decreased. Pga does not decease significantly. This data suggests increased lung volume does not lead to increased stiffness but that EMG and pressure play a major role.

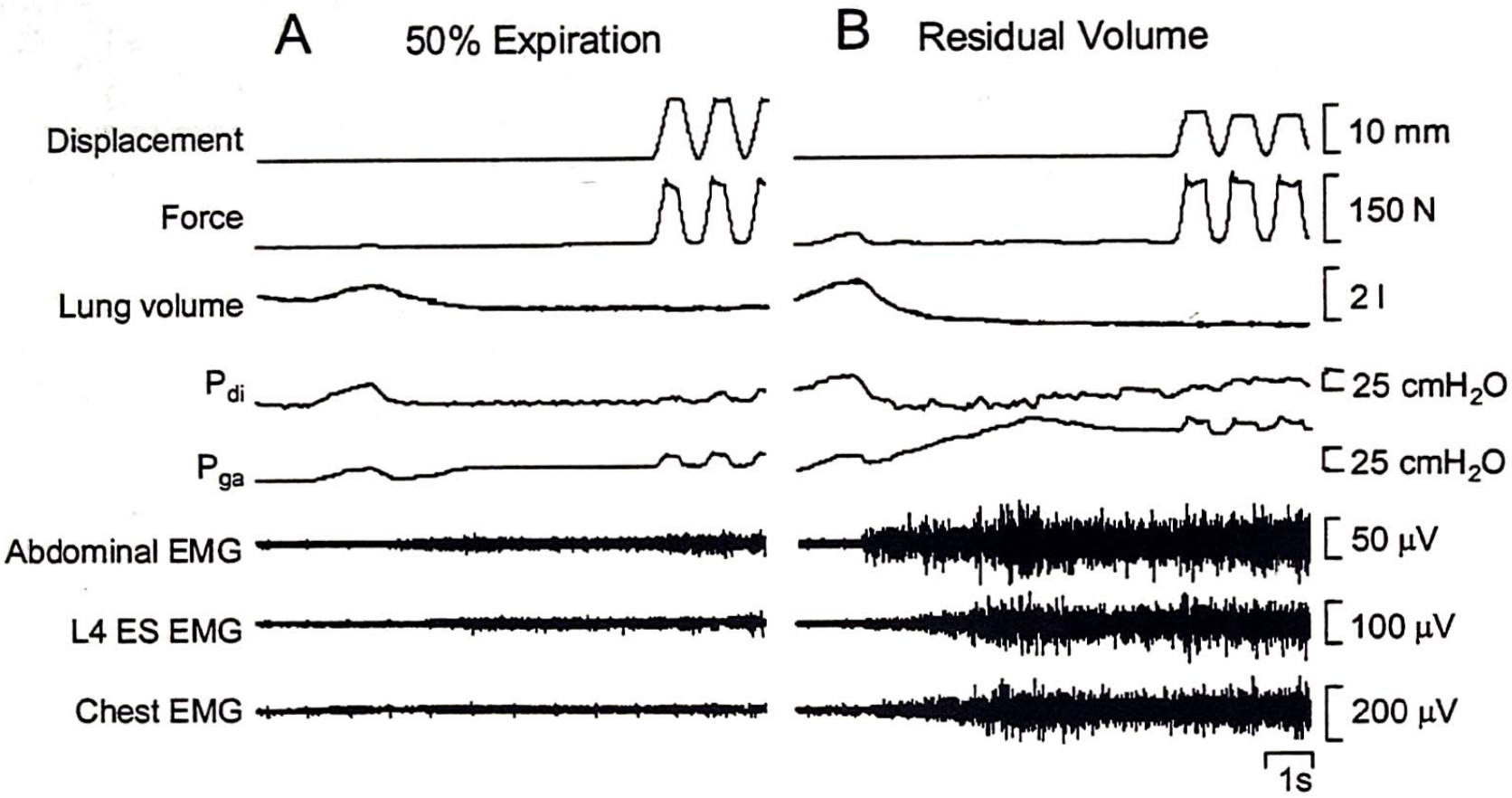

Figure 6.8. Representative data for the 50% Expiration and RV conditions. Increases in stiffness were greatest during testing at 50%Expiration (A) and RV (B). Increases in muscle activity were greater during ME which involved greater respiratory effort. There were also corresponding increases in pressures.

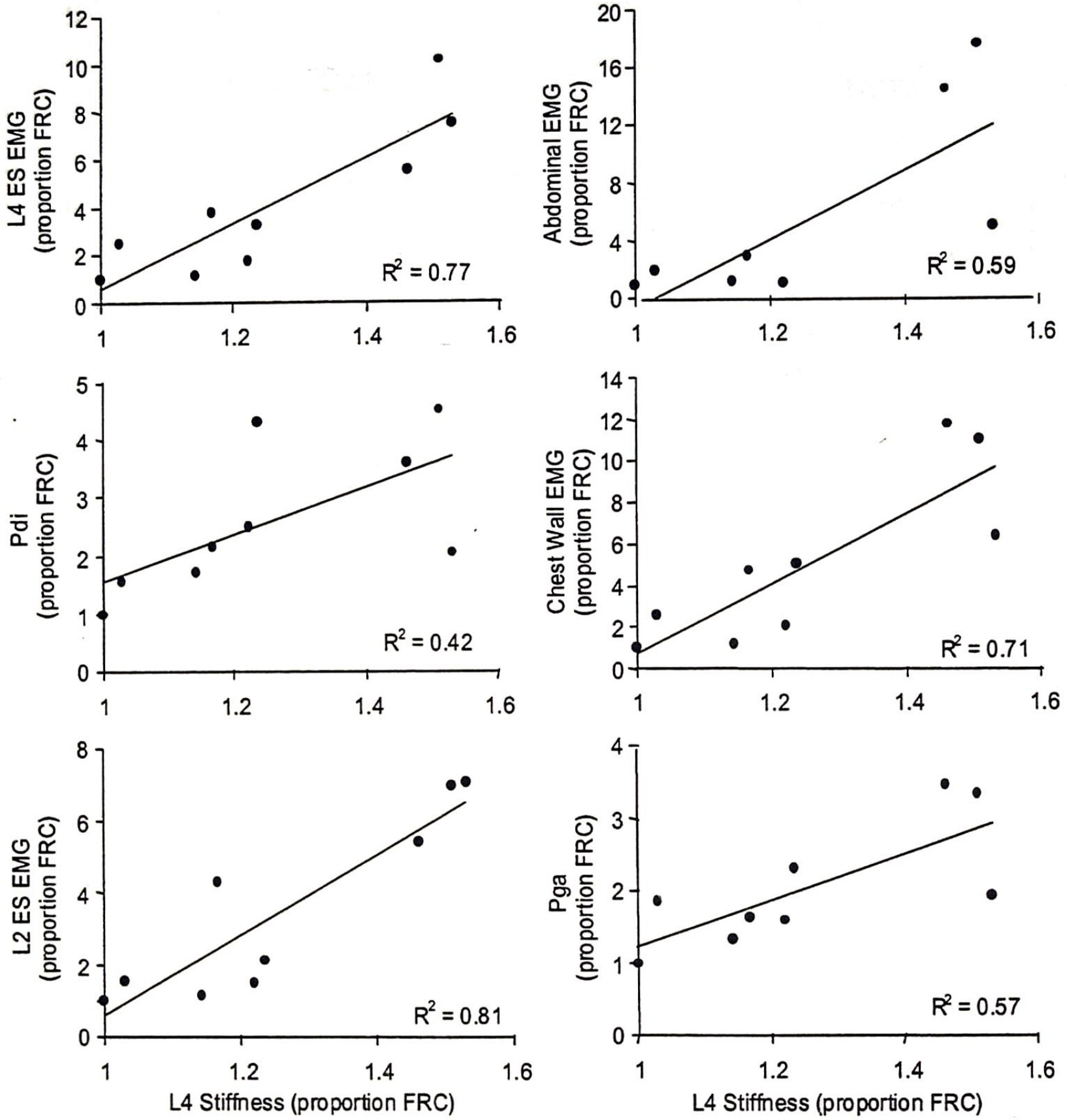

Figure 6.9. Relationship between L4 stiffness and EMG and pressure. EMG and pressure recordings were correlated with stiffness. All values (stiffness, EMG and pressure) are the mean values expressed as a proportion of the value at FRC.

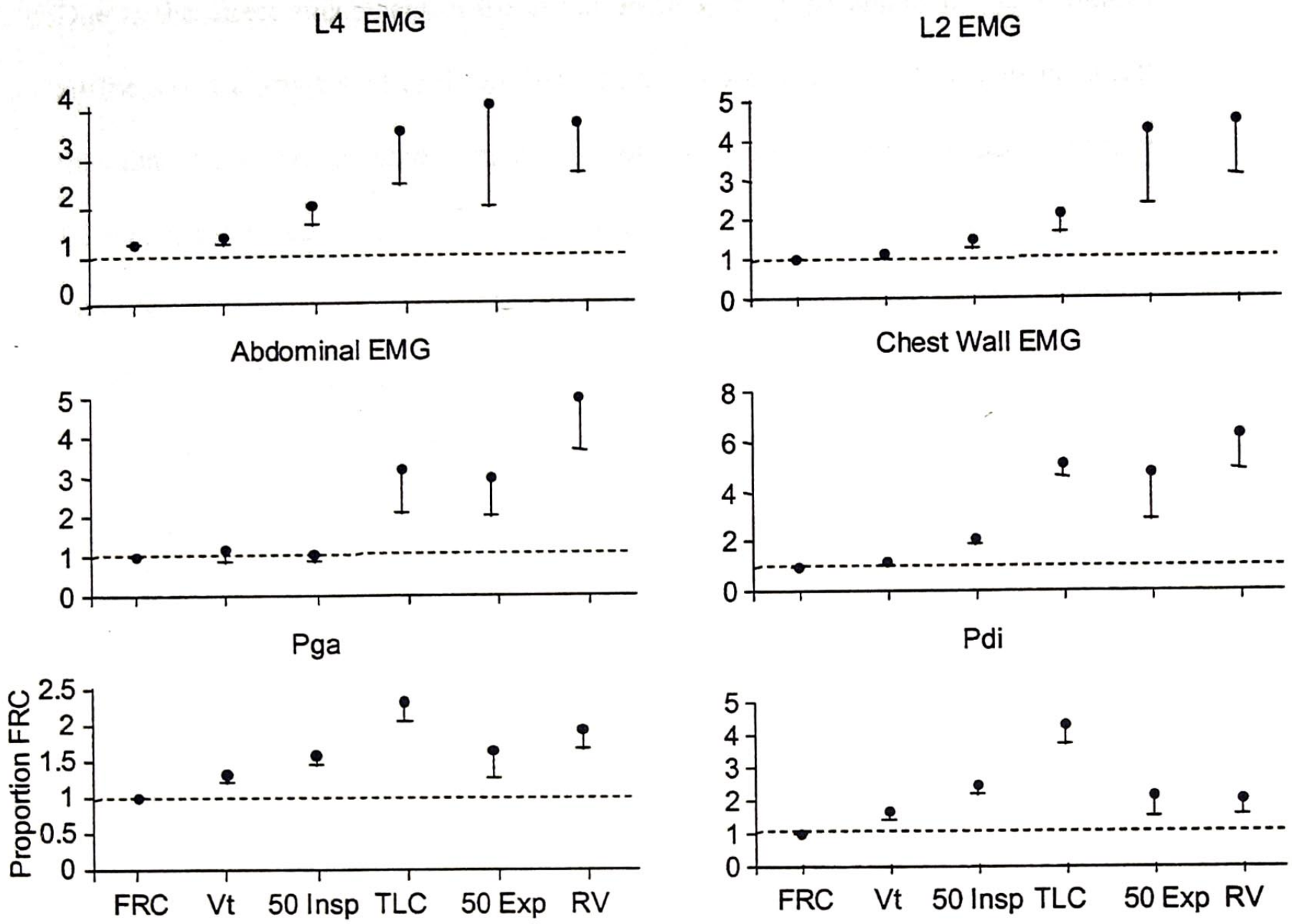

Figure 6.10. Changes in EMG and pressure between respiratory tasks. EMG and pressure recordings increase with increasing respiratory effort, except for ES EMG at L2 which was only greater than at FRC for tasks involving expiratory effort. Pga and Pdi were greater during inspiratory effort than expiratory effort.

Comparison of stiffness changes at L2 and L4

Due to the direct attachment of the crural diaphragm to the upper lumbar vertebrae stiffness at L2 was tested to determine whether it was increased to a greater extent than that at L4. When subjects held lung volume at maximal inspiration stiffness at L2 was greater than at L4 (P=0.038). However, there was no difference between these two lumbar levels with other conditions (Figure 6.11, Table 1) (FRC: P=1.0, Vt: P=0.14, 50% inspiration: P=0.66, 50%expiration: P=0.59, RV: P=0.23). Group data presented in Figure 6.11 show that the increase in stiffness at L4 reaches a plateau at the lung volumes above normal tidal volume. In contrast, L2 stiffness increases in an almost linear manner for all increments in lung volume.

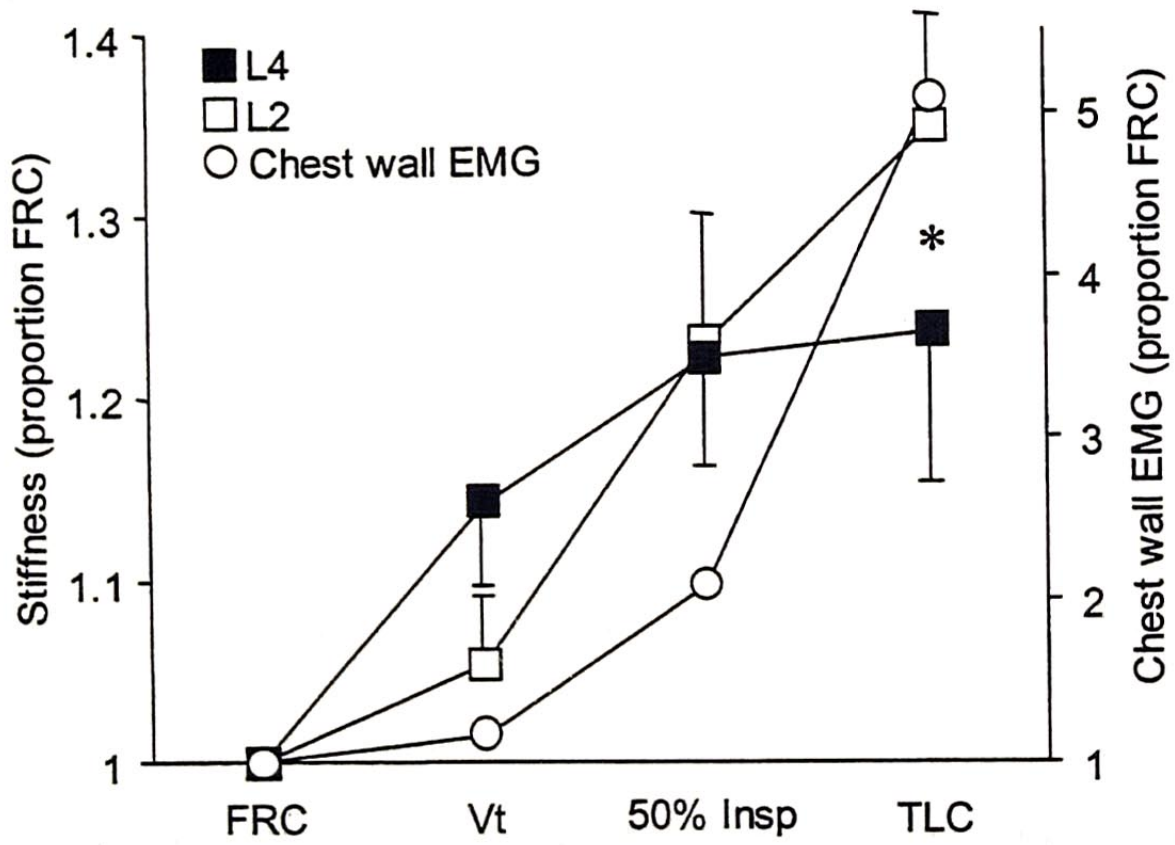

Figure 6.11. Proportional increases in mean (SE) stiffness at L2 and L4. There was a different pattern of increase in stiffness when the spine was loaded at L2 to when loaded at L4. The increase when loaded at L4 is most marked between FRC and end tidal inspiration (Vt) and then plateaus with increasing effort (50% Insp and TLC). In contrast the increase at L2 is more gradual over the range of respiratory tasks chosen. The pattern of increase in stiffness at L2 is similar to the pattern of increase in chest wall EMG.

DISCUSSION

The present study demonstrates the effect of respiration on lumbar stiffness during mechanical loading. Although stiffness at L4 did not change with lung volumes within the normal tidal range, when lung volume increased well above or below functional residual capacity stiffness was increased. The present data indicate that all of the factors measured contribute to this modulation of stiffness with respiration. Across tasks, changes in stiffness were positively correlated with changes in trunk muscle EMG and both transdiaphragmatic and abdominal pressures. However, in some conditions changes in transdiaphragmatic pressure and chest wall EMG, which are indirect indicators of activity of the diaphragm, were sufficient to cause a change in stiffness.

The results of the present study are consistent with a brief report that demonstrated no change in stiffness with quiet respiration during breath holding or tidal breathing (Beaumont et al. 1991). More recently, stiffness has been observed to increase during a Valsalva manoeuvre, although when breath was held at full inspiration stiffness did not increase in the majority of subjects (Kawchuk and Fauvel 2001). However, it is unclear whether glottis was maintained open or closed. While there has been some investigation of the effects of respiration on spinal stiffness our study is the first to compare the stiffness response of a variety of respiratory efforts including both inspiration and expiration.

Stiffness of the spine is a composite measure of the response of the spine that does not only measure intersegmental stiffness at a specific intervertebral level but is

influenced by stiffness of the entire spine and its supporting structures. For instance in the lumbar spine this measure of stiffness reflects a complex movement of the spine, which involves soft tissue compression, extension of the spine, anterior rotation of the pelvis, deformation and rigid-body displacement of the ribcage and a small amount of anterior shear of the target vertebra (Lee et al. 1996). Accurate measurement of intervertebral stiffness requires fixation adjacent segments of the spine. However, the stiffness of the segments at which the force is applied is likely to contribute significantly to the overall measurement. In the present study motion of the rib cage was restricted by application of a firm belt to minimise further compression and ensure that rib position was consistent between test conditions and the abdomen was left unsupported to reduce the effect of the compliance of the abdominal contents on the stiffness measure. Despite the limitations of this measure of spinal stiffness, it does provide a non invasive technique to evaluate spinal stiffness, at least in one direction.

This study indicates that the behaviour of muscle activity and pressures was consistent with the change in spinal stiffness and, in general it was not possible to distinguish between them. During increased respiratory effort there are increases in activity of the abdominal and erector spinae muscles and chest wall EMG (i.e. diaphragm and intercostal muscles), as well as increases in pressures (Pga and Pdi). The EMG recordings of erector spinae and abdominal muscles in this study are most likely to represent activity of the superficial muscles, however activity of the deeper trunk muscles, such as the multifidus and transversus abdominis, is also likely during some of the respiratory tasks and will have contributed to the net change in spinal stiffness. Many muscles contribute to the control of spinal stiffness (Bergmark 1989;

Cholewicki and McGill 1996). However, the relative contribution of individual muscles is likely to vary due to architectural and biomechanical factors. Recent evidence suggests that muscles such as transversus abdominis, which is a principal expiratory muscle (De Troyer et al. 1990), maybe important for control of intersegmental stiffness of the spine (Cresswell et al. 1992; Hodges and Richardson 1997b; Hodges et al. 2001a). This muscle may modulate spinal stiffness as a result of increased intra-abdominal pressure (Grillner et al. 1978; Hodges et al. 2001a) or increased tension in the thoracolumbar fascia (Tesh et al. 1987). However, other muscles also have an important contribution to modulation of spinal stiffness. The high correlation between erector spinae EMG activity and stiffness suggests that this muscle contributes significantly to lumbar stiffness (Chapter Three).

Spinal stiffness was unchanged over tidal volume and the associated changes in Pga and Pdi were small. Both gastric and transdiaphragmatic pressures were increased with respiratory efforts above and below normal tidal volume with the greatest increase in these pressures measured during the maximal inspiratory effort. In contrast the greatest increase in spinal stiffness was recorded with maximal expiration. This finding does not indicate that changes in pressure (Pga and Pdi) and stiffness were not related, rather it highlights the complex manner by which pressures and muscle activity contribute to the stiffness increase. That is, that the measured stiffness represents the net effect of all factors that are independently changed by respiratory efforts. While other studies have identified increased spinal stiffness during tasks that increase Pga such as a Valsalva maneourvre (Kawchuk and Fauvel 2001), it is difficult to confirm to what extent the pressure increase affects the spinal stiffness measurement in these studies as activity of the abdominal

and back extensor muscles is increased to produce the pressure change. One recent study that produced an increase in Pga by electrical stimulation of the phrenic nerves has confirmed that pressure alone may increase spinal stiffness via contraction of the diaphragm, without activation of the abdominal muscles (Hodges et al. 2001b).

Although this study has demonstrated a number of factors that may contribute to modulation of stiffness of the spine, other factors may also have changed during the respiratory tasks. For example quadratus lumborum attaches between the twelfth rib and pelvis (Bergmark 1989) and may be modulated with respiration. Activity of this muscle is likely to affect spinal stiffness. Other muscles such as psoas and the pelvic floor muscles (Sapsford and Hodges 2001) may also have an effect on the lumbopelvic region.

EMG activity recorded with the electrodes over the chest wall was also correlated with stiffness. Stiffness during maximal inspiration with the glottis closed was the same as at FRC but was increased with the glottis open. Lung volume was similar in both conditions and therefore, it is unlikely that passive changes in lung volume alone have an important influence on stiffness. When the glottis is held open diaphragm activity is necessary to maintain a constant lung volume whereas when the glottis is held closed lung volume can be maintained with the diaphragm relaxed. The main difference we observed between the two conditions was the chest wall EMG and the associated pressures. Although the chest wall EMG will have recorded activity from both the diaphragm and intercostal muscles, it is likely that a major contribution to this signal in the inspiratory effort is from the diaphragm.

The diaphragm may contribute to spinal stiffness by increasing IAP (Hodges et al. 1997b; Cholewicki et al. 1999a; Hodges and Gandevia 2000b). Correspondingly, as described above, when IAP is increased by activity of the diaphragm, without abdominal or erector spinae activity, lumbar stiffness increases (Hodges et al. 2001b). In addition, contraction of the diaphragm may increase spinal stiffness via the attachment of the diaphragm crurae to the upper lumbar vertebrae. To further investigate the contribution of the crural diaphragm to stiffness this study also indirectly evaluated the effect of the crural attachments of the diaphragm on lumbar stiffness by comparing of the changes in stiffness at L2 and L4 with inspiratory efforts. The crural diaphragm has attachments in the lumbar vertebrae as caudal as L2 or L3 (Williams et al. 1995). Therefore, if the diaphragm crurae contributes to lumbar stiffness it would be expected that stiffness would be greater at L2, compared to that at L4 where there is no direct attachment. The greater increase in stiffness at L2 reported here is consistent with this proposal. As mentioned above, non invasive measurement of spinal stiffness cannot provide an ideal measure of intersegmental stiffness and is affected by the stiffness at segments distant from the site of application of the PA force. However the finding that the change in stiffness was greater at L2 compared to L4 during similar tasks suggests that there was independence between the two measures and the measures are likely to reflect changes at the specific level, in addition to an influence from other distant sites. The proposal that stiffness is increased by contraction of the crural fibres is further strengthened by comparison of the pattern of the increase in stiffness at L2 and L4 (see Figure 6.11). The stiffness increase with increasing inspiratory efforts was linear at the L2 level, corresponding to an incremental increase in chest wall EMG. In

contrast the increase in stiffness at L4 reached a relative plateau at higher lung volumes, independent of further increases in chest wall EMG.

The results of this study have several functional and clinical implications. First, numerous studies have investigated the relationship between breathing and lifting. Generally these studies argue that breath holding or expiration are normal behaviour (Hemborg et al. 1985; McGill and Norman 1987). The present data suggest that stiffness of the spine is increased with both inspiratory and expiratory efforts but more so with expiration. This is consistent with the natural behaviour to expire when performing demanding tasks such as lifting. Furthermore the present data argue that stiffness is greater with the glottis open and trunk muscle contraction maintained, therefore it might be beneficial to recommend this action when attempting procedures requiring greater spinal stability. Secondly, clinical application of postero-anterior force to the spine is commonly used in the treatment of acute spinal pain. The present data suggest that it may be necessary to standardise the point in the respiratory cycle to make accurate judgements of spinal stiffness.

CHAPTER SEVEN

CONCLUSIONS

Manual application of postero-anterior forces to the spine is commonly used during assessment and treatment for low back pain. While forces have been used in this way since the time of Hippocrates (Schiotz and Cyriax 1975) there have not been any substantial changes to the method of application or in the understanding of mechanism. Recent evidence-based guidelines have recommended that manual therapy is most effective in the management of acute LBP but the use in chronic LBP is controversial (Bigos et al. 1994; CSAG 1994; Waddell et al. 1996). Despite the common use there is a poor understanding of the spine's response to applied forces both in the asymptomatic and symptomatic populations. In recent years, attempts have been made to describe responses to manipulation and mobilisation, although this has predominantly occurred using asymptomatic individuals.

This thesis presents a series of studies that investigated the responses of the lumbar spine to applied PA forces in both asymptomatic and symptomatic subjects. The responses examined include PA stiffness, displacement and muscle activity, with measurements made while the subjects were at rest or performing tasks involving activation of trunk and respiratory muscles. Each of the preceding chapters includes a discussion of the specific findings of that study. This final chapter aims to discuss the findings of the whole thesis and integrate them to determine and speculate on their implications for the understanding of mechanisms related to manual therapy and clinical practice.

Normal Lumbar PA Response

Chapter Two of this thesis was undertaken to determine the normal response of the lumbar spine to PA loading and to determine whether the response is stable over time. The findings indicated that when PA forces are applied to the lumbar spine there is a time dependent behaviour similar to the cyclic response previously described in cadaveric and living tissues (Yahia et al. 1991; Lee and Evans 1992). The PA response is stable over short periods of time eg five minutes, and also over a longer period ie up to eight days. Furthermore, Chapter Four measured a group of asymptomatic subjects and found that the PA response also did not change over much longer periods of up to seven months. Therefore the lumbar PA responses generally do not vary over time in asymptomatic individuals if the testing conditions are constant. This suggests that changes in stiffness that occur between treatments in LBP patients probably reflect something different than the time dependent behaviour of the tissues.

Another finding of Chapter Two was that the response during the first loading cycle is different from that of subsequent cycles ie stiffness was less and displacement was greater. This response was consistent each time PA stiffness was measured, even after a short interval of 5 minutes. Therefore, this finding indicates that it may be important to omit the first cycle from analysis of research data. In addition, if comparisons of data from different researchers are undertaken, it is important to make sure equivalent testing procedures and analyses are compared. Comparison of stiffness data from the studies in this thesis with other studies that have only reported

the first cycle is not recommended as studies that have reported results from only one cycle will report a lower stiffness. This is the first work to describe the variation between the first and subsequent cycles during application of PA forces as a normal response of the tissues of the lumbar spine to cyclic loading in asymptomatic subjects.

The normal responses of the lumbar spine to loading with PA forces have implications for the use of these forces in clinical examination of patients with LBP as well as for research purposes. As the lumbar spine exhibits time dependent behaviour to mechanical loading within a series of loading cycles, the clinical application of PA forces to assess PA stiffness may result in the short term decreases in PA stiffness perceived during assessment with PA forces. These effects due to testing the lumbar spine with PA forces are no longer apparent after five minutes. Therefore, an important implication is that other mechanisms than the time dependent behaviour of tissues are responsible for changes in PA stiffness lasting for longer periods of time (eg between treatments).

Muscle Activity and Lumbar PA stiffness

Much of the research on effects of manual therapy has involved investigating the muscle responses that occur during high velocity manipulative (thrust) techniques. Previous research has shown that these techniques provoke a reflex response in the muscles adjacent to the spine and in some cases also distant from the spine (Herzog et al. 1999). Alternate approaches have reported an increase in mean amplitude of

EMG in response to a slow sustained load (eight seconds) and a reduction in the mean amplitude after a manipulative thrust in asymptomatic subjects (Kawchuk and Fauvel 2001). This thesis provides the first evidence of a relationship between muscle responses (measured by reflex activity and mean amplitude) and PA stiffness in the lumbar spine in both a population with acute LBP and asymptomatic subjects.

This thesis investigates the muscle responses in two ways, firstly the response of PA stiffness to PA forces applied during voluntary activation of various trunk and respiratory muscles in asymptomatic subjects, and secondly the response of the lumbar spine to applied PA forces in passive symptomatic and asymptomatic subjects. Chapters Three, Five, and Six investigated the effects of various voluntary activities including activation of the erector spinae muscles, abdominal muscles, respiratory muscles and associated intra-abdominal pressure on lumbar PA stiffness. Chapter Four investigated the responses of the erector spinae in the resting subject with LBP.

Chapters Three, Five and Six demonstrated that voluntary activation of muscles that act on the lumbar spine increases lumbar PA stiffness. In Chapter Three, voluntary activation of the lumbar extensor muscles significantly increased lumbar PA stiffness even when the activation was as low as 10% MVC. The increase in stiffness was in the range of elastic stiffness able to be detected manually by some manipulative physiotherapists (Maher and Adams 1995) ie about 11%. Therefore, stiffness increases of 12% (Chapter Three) due to small amounts of voluntary activity are in the range that could be detected during examination with PA forces by some physiotherapists.

Chapters Five and Six examined the contribution of muscles activated during respiration to lumbar PA stiffness. Chapter Five indicated that there was no increase in PA stiffness with breath holding at different inspiratory lung volumes. In this study subjects were instructed to hold their breath at various inspiratory lung volumes but were not instructed to hold their glottis open or closed. It is a natural response to close the glottis to maintain lung volume during breath holding, as less respiratory effort is required, ie inspiratory muscles are able to relax. Confirming this, Chapter Six demonstrated that breath holding at the same lung volumes with the glottis held closed significantly decreased PA stiffness compared to breath holding with the glottis open. The difference between these tasks was greater activation of the respiratory, abdominal and erector spinae muscles with the glottis open. In addition, there was an increase in transdiaphragmatic pressure with the glottis open. Increases in PA stiffness were observed during dynamic tasks such as tidal breathing and other tasks that involve activation of the respiratory muscles. These results support the theory that it is muscle activity rather than lung volume that increases lumbar PA stiffness during these voluntary tasks. There was no difference in PA stiffness with breath held at different lung volumes with the glottis closed, however there were changes in stiffness between inspiratory and expiratory cycles during dynamic breathing with corresponding changes in respiratory muscle activity. This thesis provides further evidence that the diaphragm contributes to lumbar PA stiffness, as there is greater stiffness when loading is applied at the L2 level, where there is a direct attachment of the diaphragm, than at L4. This thesis also provides evidence that factors including trunk and respiratory muscle activity and intra-abdominal and transdiaphragmatic pressures contribute to lumbar PA stiffness, further supporting the multifactorial nature of stiffness.

The studies in this thesis indicate that voluntary activation of trunk and respiratory muscles increases PA stiffness. The mechanism of increasing PA stiffness is due to a combination of many factors and involves contributions from various muscles that contribute to lumbar stability either by their direct actions on the lumbar spine or indirectly via the thoraco-lumbar fascia or by increasing intra-abdominal pressure. The evidence from this thesis indicates that muscle activity increases PA stiffness even when activation is small (Chapter Three) and particularly when there is maximal activity of the muscles (Chapters Three and Six).

The implication of these findings for clinical practice is that it is important to standardise the respiratory effort of patients during testing of PA stiffness. If the patient is holding their breath with a closed glottis the lung volume does not appear to be important. However, it may be easier for subjects to breath hold at volumes greater than FRC as they will not be as oxygen depleted. In addition, specific instructions about glottis closure should be given as there is more muscle activity and greater stiffness with the glottis held open. It is also important for the patient to be relaxed during assessment of PA stiffness because activation of the lumbar extensors or other trunk muscles will potentially affect stiffness of the lumbar spine. Chapter Six demonstrated that stiffness is greatest when breath is held at maximal expiration with an open glottis. People often breath hold or breathe out during tasks involving considerable exertion (eg lifting, weightlifting etc). While there is no demonstrated relationship between PA stiffness in prone to stability during tasks in other positions it is interesting to speculate on the purpose of breathing out during exertion. This practice may have come about as it increases stiffness of the spine and it may be a natural mechanism to attempt to stabilise the spine. This thesis

provides the first evidence that activity of erector spinae, abdominal muscles and the diaphragm, which contribute the stabilisation the spine during active tasks (Hodges et al. 1997a; Hodges and Richardson 1997a), also contribute to PA stiffness.

The diaphragm is active during tasks that require stabilisation of the lumbar spine (Hodges et al. 1997a). The diaphragm produces phasic activity related to the breathing pattern as well as tonic activity while the task is performed (Hodges and Gandevia 2000a). In asymptomatic subjects without respiratory compromise, the diaphragm is able to perform both of these functions simultaneously (Hodges and Gandevia 2000a). The implications of respiratory disease on the function of the diaphragm and its role in stability of the spine have not yet been investigated. However, people with respiratory disease are more susceptible to LBP (Synnot and Williams 2001) or may be more likely to develop chronic LBP.

PA stiffness, muscle activity and low back pain

A further important aspect of this thesis was to determine if there is a relationship between PA stiffness, lumbar muscle activity and low back pain. Results from Chapter Four concur with the study by Latimer et al. (1996c) that subjects with low back pain have increased lumbar PA stiffness, however the effect was only small. An important finding of the current work is evidence that a combination of stiffness and variables of muscle activity are associated with low back pain during an acute episode of LBP. In addition, the presence of pain many months later is also associated with a combination of stiffness and muscle activity factors during the

episode of pain. The association was interesting because during the acute episode of LBP, higher pain intensity was associated with higher stiffness, however, over the longer-term low stiffness in the acute phase appeared to be associated with longer lasting pain. There was also a trend for lower muscle activity during the initial episode to be associated with ongoing symptoms.

Increased stiffness during an episode of LBP may be the body's mechanism to protect the spine against further damage. It is interesting to note that low stiffness and lower muscle activity during the initial episode seems to be associated with longer lasting pain. In Chapter Four only erector spinae activity was measured, however, it is possible there was also decreased activity of other spinal stabilising muscles that were not measured (eg multifidus and transversus abdominis). These muscles may be recruited to stabilise the spine in response to externally applied PA forces. It is possible that there is inhibition of muscle activity due to pain. Transversus abdominis and multifidus do not activate as early during voluntary activity in people with LBP (Hodges and Richardson 1998; Hodges and Richardson 1999a) and decreased cross sectional area of multifidus is associated with acute LBP (Hides et al. 1994). Consideration of the importance of decreased muscle activity and low stiffness allows speculation of the implications for treatment of acute LBP. When stiffness is low, exercises aimed at improving the function of stabilising muscles may be more appropriate than manual therapy as exercises should help to stabilise and stiffen the spine.

While previous reports have described a reflex muscle response to application of manipulation to the spine (Herzog et al. 1999), this thesis does not indicate that the

application of a PA force to the lumbar spine triggers a stretch reflex in all subjects. Some individuals appeared to have a stretch reflex response although these responses occurred in both symptomatic and asymptomatic subjects. However, there was a trend for increased stretch reflex behaviour (coherence) during an episode of LBP. The results of this thesis indicate that while reflex responses of the erector spinae may occur in response to a manipulative thrust or mobilisation procedure, they may be a normal response to forces applied to the spine in some people irrespective of their pain status. Therefore, at least in the resting patient, reflex activity of the erector spinae is unlikely to be a major contributing factor to PA stiffness.

Chapter Three indicated that voluntary activity of the erector spinae increased PA stiffness. There was an increase in erector spinae activity in subjects with LBP. It is possible that the increases in erector spinae activity detected in this study are not of great enough magnitude to result in clinically relevant increases in stiffness. A contraction of 10% MVC of erector spinae results in a significant increase in PA stiffness. Interestingly the levels attained during the study of subjects with LBP were only 22% of a submaximal contraction or approximately 4.4% of an MVC (Chapter Four), which may not be big enough to influence PA stiffness.

Limitations

In this thesis a number of different methods were used to calculate PA stiffness. In Chapters 2 and 4, stiffness was calculated as the slope of a regression line fitted to the force displacement curve between 30 and 90N. This method has been used in many previous studies investigating lumbar PA stiffness (Latimer etc). In Chapter 3

the force range used was 20 to 100N, a range commonly used on that testing apparatus. In Chapter 6 the force range chosen was between 50 and 110N because the force displacement curve was more linear in that range and previous studies has chosen the force range by selecting the linear part of the force displacement curve (Ltaimer) . In Chapter 5 a new method for calculating stiffness called instantaneous stiffness at 50N was described as well as the method 30-90N was used for comparison.

The fact that various different ranges of force were used for calculation of stiffness throughout this thesis means that it would be difficult to compare stiffness values between studies. However this was not attempted in the thesis. Stiffness is greater when higher forces are used to calculate stiffness (Latimer et al year). Therefore it is likely that in Chapter 6 the stiffness values would be higher than if stiffness were calculated between 30 and 90N. In Chapter 5 instantaneous stiffness was lower than when the 30-90N force range was used. This should not affect comparisons of stiffness within a study.

Another possible limitation is that the mechanical device used for testing stiffness in Chapters 2, 4, 5 and 6 was only calibrated on two occasions while conducting the studies for this thesis. The first was before any data collection commenced for the thesis. The force output of the device was tested against a set of scales of known accuracy and the displacement was measured against a dial indicator of known accuracy. Some minor mechanical modifications were made to the device after the Chapter 2 study and it was again calibrated before commencing the study for Chapter 4. The studies conducted in Chapter 5 and Chapter 6 were conducted concurrently with the Chapter 4 study.

Future directions

The findings of this thesis suggest some important considerations for the management of LBP. The association between the presence of pain several months after onset and stiffness and muscle activity during the episode suggests it may be possible to identify those patients whose pain will not resolve rapidly. Therefore, further work is needed to determine whether receiving treatment or particular interventions will change the outcome for these people. It is obvious that there is a need for further investigation in this area. It might be possible to determine which factors are most related to ongoing symptoms and establish a reliable and valid method to identify those in this category during their initial episode.

Although mobilisations are recommended in the management of acute LBP they do not prove to be effective for all patients. The findings of Chapter Four indicate that subjects with higher initial stiffness have higher pain. Further investigation of the effect of mobilisations on symptomatic subjects who have been categorised for range of stiffness should indicate whether mobilisations are more effective for specific ranges of stiffness. It would be expected that people with high stiffness would benefit most from mobilisations, as stiffness appears to decrease as pain resolves (Chapter Four). In contrast, subjects with low stiffness are more likely to have ongoing pain and perhaps would have a better result with a management approach that focuses more on exercises.

In addition, future studies should investigate the role of other muscles in spinal PA stiffness eg transverus abdominis and multifidus. These muscles are stabilisers of the spine during voluntary tasks that perturb the spine (Hodges et al. 1997a; Hodges and Richardson 1997a). Transversus Abdominis and multifidis were not measured in the studies of this thesis as they are deep muscles and cannot be specifically measured with surface EMG. Testing of the spine with PA forces involves the application of unexpected perturbations to the spine which may activate deep muscles to assist in stabilising the spine. Therefore, studies measuring stiffness could be undertaken using intra-muscular EMG. In addition, reflex behaviour of spinal stabilising muscles could be studied to gain a better understanding of how it is affected by LBP. This thesis found a trend for increased reflex activity in subjects with LBP. Further work could investigate the reflex response using more homogeneous groups of LBP subjects to determine whether the response is specific to particular groups of symptomatic subjects.

Further evidence for the contribution of the diaphragm to PA stiffness is also presented. The contribution of the diaphragm to PA stiffness in people with LBP needs to be investigated. In particular, the relationship between respiratory disease and LBP warrants further study.

Summary

This thesis provides evidence for the contribution of muscle activity to lumbar PA stiffness. Voluntary activation of trunk and respiratory muscles increased PA stiffness and although the effect was most marked during activities requiring maximal levels of activation the effect was present even with small amount of activation (ie 10% maximal activation of erector spinae). Other factors such as intra-abdominal pressure also contribute to increased PA stiffness. Importantly, evidence has been provided for a relationship between pain intensity, muscle activity and PA stiffness in symptomatic subjects.

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