Postural control impairment characteristics of chronic, recurrent low back pain: an investigation of automatic postural responses and sit-to-stand movements

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DEDICATION

This document is dedicated first and foremost to the individuals who participated in this study and those who experience chronic pain. Through your grateful contribution to our work we hope to understand your pain and develop effective treatments or strategies for prevention that will improve your quality of life.

This document is also dedicated to all graduate student mothers who have managed to juggle all aspects of motherhood and academia, remaining true to our families and ourselves.
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<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>aLBP</td>
<td>A group of people with LBP who are having an active pain episode of their spinal pain</td>
</tr>
<tr>
<td>ANOVA</td>
<td>Analysis of variance</td>
</tr>
<tr>
<td>AP</td>
<td>Anterior-posterior</td>
</tr>
<tr>
<td>APR</td>
<td>Automatic postural response</td>
</tr>
<tr>
<td>BMI</td>
<td>Body Mass Index</td>
</tr>
<tr>
<td>BOS</td>
<td>Base of support</td>
</tr>
<tr>
<td>CM</td>
<td>Centre of mass</td>
</tr>
<tr>
<td>COP</td>
<td>Centre of pressure</td>
</tr>
<tr>
<td>COPap</td>
<td>Centre of pressure (anterior-posterior)</td>
</tr>
<tr>
<td>COPlat</td>
<td>Centre of pressure (lateral)</td>
</tr>
<tr>
<td>EMG</td>
<td>Electromyography</td>
</tr>
<tr>
<td>EO</td>
<td>External Oblique</td>
</tr>
<tr>
<td>ES1</td>
<td>Erector Spinae at 1st lumbar segment</td>
</tr>
<tr>
<td>ES3</td>
<td>Erector Spinae at 3rd lumbar segment</td>
</tr>
<tr>
<td>Abbreviation</td>
<td>Definition</td>
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<tr>
<td>--------------</td>
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</tr>
<tr>
<td>ES8</td>
<td>Erector Spinae at 8&lt;sup&gt;th&lt;/sup&gt; thoracic segment</td>
</tr>
<tr>
<td>GA</td>
<td>Gastrocnemius</td>
</tr>
<tr>
<td>GRF</td>
<td>Ground reaction force</td>
</tr>
<tr>
<td>HA</td>
<td>Medial Hamstrings</td>
</tr>
<tr>
<td>IO</td>
<td>Internal Oblique</td>
</tr>
<tr>
<td>LBP</td>
<td>Low back pain</td>
</tr>
<tr>
<td>MSI</td>
<td>Movement Systems Impairment Classification System</td>
</tr>
<tr>
<td>MVC</td>
<td>Maximal voluntary contraction</td>
</tr>
<tr>
<td>NLBP</td>
<td>A group of people who do not have low back pain</td>
</tr>
<tr>
<td>NPRS</td>
<td>Numeric Pain Rating Scale</td>
</tr>
<tr>
<td>RA</td>
<td>Rectus Abdominus</td>
</tr>
<tr>
<td>ROM</td>
<td>Range of motion</td>
</tr>
<tr>
<td>RF</td>
<td>Rectus Femoris</td>
</tr>
<tr>
<td>SD</td>
<td>Standard deviation</td>
</tr>
<tr>
<td>STS</td>
<td>Sit-to-stand</td>
</tr>
<tr>
<td>TA</td>
<td>Tibialis Anterior</td>
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ABSTRACT

Evidence is accumulating that individuals with chronic low back pain (LBP) have altered movement coordination; however, the exact nature of this impairment is unknown. To clarify the relationship between pain and altered coordination this thesis characterized postural control in two cohorts of individuals with LBP (those with a history of pain who were in a quiescent period and those who were in an active pain period), relative to individuals without LBP. Automatic postural responses (APRs) as measured by joint torque and myoelectric responses were characterized following multi-directional support surface translations. Given that the active pain cohort had greater pain than the quiescent group, they might be more susceptible to volitional effects on movement. Therefore this cohort as well as the control group also performed a voluntary activity of daily living, the sit-to-stand (STS) movement, to quantify the influence of pain on a volitional movement. Their performance was then compared to the performance of the healthy control group. The first study demonstrated that the APRs of healthy individuals used a redistribution of the contributions of hip/trunk vs. ankle torques depending on the perturbation direction. The relative contribution of joint torques appears to be determined by the biomechanical constraints imposed by a specific
perturbation direction. In the second study, individuals with LBP in a quiescent period demonstrated increased sagittal plane ankle torque during the passive epoch (50-100ms after perturbation onset) and reduced peak trunk torques following perturbation, suggesting that they may have increased stiffness at the ankle and use a response strategy of active trunk stiffening, due to co-contraction of musculature. In the third study, individuals with LBP in an active pain episode demonstrated earlier but not altered peak torque magnitudes, and increased muscle activation, which may indicate increased muscle spindle sensitivity or increased postural gain. In the fourth study individuals with active LBP performed the STS movement with no speed alterations but with a strategy that relied more on distal joint contributions, which may reflect a cognitive decision to restrict trunk excursion. En masse, these findings suggest that altered movement strategies persist in the chronic, recurrent LBP population between painful episodes, which may reflect an intention to restrict trunk movement. Individuals with chronic LBP who are in an exacerbation of their pain appear to have a generalized heightened postural response to unexpected perturbation that may reflect a short-term modification to a heightened pain state. These individuals also move to restrict their trunk during volitional movements such as the STS. Such alterations, if
repeated, may lead to persistent abnormal movement patterns that could increase the susceptibility to future LBP episodes, as demonstrated by individuals in a quiescent period of their pain cycle. Interventions that focus on a return to increased variability and distribution of movement could enhance movement precision, through tissue and neuromuscular changes, and lead to a reduction of the frequency and severity of pain recurrence.
Il est de plus en plus démontré que la coordination des mouvements est altérée chez les individus souffrant de douleur chronique au dos (DCD). Toutefois, la nature exacte de cette déficience demeure inconnue. Afin de clarifier la relation entre la douleur et la coordination des mouvements, l’objectif de cette thèse était de caractériser et comparer les stratégies de contrôle postural chez deux groupes d’individus souffrant de DCD (ceux ayant déjà eu de la douleur mais étant dans une période quiescente et ceux souffrant présentement de douleur) et chez un groupe de sujets n’ayant jamais souffert de DCD. Les réponses posturales automatiques (RPAs), telles que mesurées par les moments de force articulaires et les réponses myoélectriques, ont été examinées suite à la présentation de brefs déplacements multidirectionnels, en translation, de la surface de support. Comme les individus souffrant de DCD en période active étaient plus susceptibles de montrer un comportement altéré dans leurs mouvements, ils ont également exécuté une activité de la vie quotidienne, soit de passer de la position assise à la position debout, afin de quantifier l’influence de la douleur sur un mouvement volontaire. Leur performance a pu être comparée à celle des personnes en santé. Les résultats de la première étude ont démontré que les RPAs des individus en santé
impliquent une redistribution des contributions des moments à la hanche et au tronc par rapport aux moments aux chevilles, qui dépend de la direction de la perturbation. La contribution relative des moments articulaires semble être déterminée par les contraintes biomécaniques imposées par la direction spécifique de la perturbation. Dans la seconde étude, le moment à la cheville, dans le plan sagittal et pendant la période passive (soit 50-100 ms après le début de la perturbation), était plus grand chez les individus avec DCD en période quiescente alors que la valeur maximale du moment au tronc était diminuée en réponse à la perturbation. Ces résultats suggèrent que ces individus ont peut-être augmenté la rigidité passive de leurs chevilles et utilisé une stratégie de raidissement actif du tronc, grâce à la co-contraction musculaire. Dans la troisième étude, les individus souffrant de DCD avec douleur active ont montré des pics de moments plus tôt, mais de même amplitude, ainsi qu’une activation musculaire augmentée, ce qui peut indiquer une sensibilité accrue des fuseaux musculaires ou un gain postural amplifié. Les résultats de la quatrième étude n’ont pas révélé de différence dans la vitesse d’exécution de la tâche assis-debout chez les individus souffrant de DCD avec douleur active. Cependant, ils semblent avoir opté pour une stratégie reposant plus sur la contribution des articulations distales, ce qui
peut paraître comme une décision cognitive visant à restreindre l'excursion du tronc. L'ensemble des résultats obtenus démontre que des stratégies de mouvement altérées persistent chez les individus souffrant de DCD avec épisodes de douleur active, reflétant une intention de restreindre le mouvement au niveau du tronc. Les personnes ayant une DCD et qui sont dans une période où la douleur est présente semblent avoir une réponse posturale augmentée généralisée suite à une perturbation inattendue, ce qui peut être l'expression d'une modification à la hausse, à court terme, de leur état de douleur. Ces individus bougent également en limitant les mouvements du tronc lors de mouvements volontaires comme celui de passer de la position assise à la position debout. Ces changements, s'ils sont répétés, peuvent conduire à l'utilisation persistante de patrons de mouvements anormaux qui peuvent entraîner une susceptibilité à des épisodes de douleur au dos dans le futur. Les interventions visant à augmenter la variabilité et la distribution des mouvements en provoquant des changements au niveau des tissus et des composantes neuromusculaires, pourraient permettre d'augmenter la précision du mouvement et ainsi réduire la fréquence et la sévérité des épisodes de douleur intense.
PREFACE

A) Statement of originality

This document contains no materials that have been published elsewhere, except where specifically referenced. The studies presented in Chapters 3, 4, 5 and 6 represent original materials and contribute to the advancement of knowledge about postural coordination in individuals with and without chronic low back pain (LBP), and the interrelationship between altered movement patterns and recurrent pain episodes within the chronic LBP population. These studies provide the first full-body analysis of corrective joint torque responses to multi-directional support surface translations that provide insight about the underlying postural control mechanisms contributing to the maintenance of upright posture. In addition, these studies have documented the corrective responses to this type of postural perturbation in individuals with a history of LBP who were in a quiescent period of pain, and individuals with LBP who were in an active period of pain, compared to individuals with no history of LBP, in order to understand the cycles of pain and quiescence that characterize the majority of the chronic LBP population. Movement patterns during sit-to-stand movements were compared between individuals with an active episode of LBP and individuals without LBP to provide insight into the
volitional alterations that may exist when in close temporal proximity to a pain episode, for comparison between volitional and automatic postural coordination. These studies were designed by me and my co-supervisors, Dr. Sharon Henry and Dr. Joyce Fung. Members of my supervisory committee (Dr. James Iatridis and Dr. Julie Côté) provided suggestions and comments regarding pain models and the study design and analysis. For all four studies, I was involved in subject recruitment, experimental setup, data collection, data analysis and creation of analysis software, as well as writing the manuscripts.

All data presented were collected at the University of Vermont, in the Department of Rehabilitation and Movement Science. The protocols used in these studies were approved by the University of Vermont Institutional Review Board.

B) Contribution of authors

I, Stephanie Jones, was in charge of the work included in this thesis, including study design, recruitment of subjects, data collection and analysis, statistical analysis and the writing of manuscripts, under the guidance of Dr. Sharon Henry and Dr. Joyce Fung. Dr. Henry critically
reviewed the manuscripts that were published or submitted from this thesis. Juvena Hitt assisted in the data collection and subject recruitment, as well as critically reviewed the manuscripts. Dr. Christine Raasch provided the software to generate the joint torque model used in the support surface translation protocol. Dr. Janice Bunn provided statistical assistance and critical review of the submitted and published manuscripts.
Chapter 1: Introduction

The overall objective of this thesis was to determine the impact of chronic LBP on neuromuscular control during unexpected balance perturbations and volitional movements. Understanding the impact of chronic pain on both automatic and voluntary postural control mechanisms as well as the extent to which altered movement patterns may persist between recurrent pain episodes will provide vital information to understand the episodic nature of this type of back pain. In addition, quantification of movement patterns is a critical first step needed to gain an understanding of whether the motor control impairment characteristics of chronic LBP are a result of chronically altered movement patterns or a secondary development to chronic pain.

Quantification of joint torques as a measure of postural control strategies is useful because they represent the net neuromuscular output of a given joint including all of the passive and active contributions to force production as well as the body's configuration. This is important because it is not possible to measure the individual contributions of all the muscles acting about a joint in vivo or the interaction between the segments that can influence the joint motion. Therefore, the torque or moment of force
provides an estimation of the net contribution of each joint to the total body’s movement. In combination with muscular activity, joint torque analysis can be a powerful tool used to understand the strategies underlying the control of human movement.

The studies contained herein quantified joint torque and muscle activation patterns in several cohorts in order to understand the influence of LBP on postural control. The support surface translation paradigm was used because it provided a window into the nervous system via complex, modifiable yet obligatory postural responses (i.e. not simple reflexes) without significant cortical influence. Given that pain is a complex, central nervous system phenomenon, and that the volitional effects of pain can be significant, this paradigm was chosen in an attempt to minimize these cortical influences. While widely documented under many experimental conditions (Horak et al. 1989; Szturm and Fallang 1998; Park et al. 2004) and in many populations, both healthy (Gu et al. 1996; Runge et al. 1999; Meyer et al. 2004) and pathologic (Allum and Honegger 1992; Horak and Diener 1994; Horak et al. 1996; Mummel et al. 1998; Bloem et al. 2000; Bloem et al. 2002; Carpenter et al. 2004a), the relative torque contributions of each joint to the APRs have not been quantified in a
systematic manner in response to multi-directional perturbations. The work detailed in Chapter 3 provides quantification of the joint torque contributions to APRs following multi-directional support surface translations in a population of healthy controls. Muscle activation patterns have been quantified following a similar protocol in this population (Henry et al. 1998b; Preuss and Fung 2008) and thus have not been analyzed in this study.

Understanding the influence of chronic pain on movement requires investigation of the volitional effects and the acute pain processes on movement patterns in order to tease out the underlying alterations in motor coordination. Therefore, Chapter 4 evaluated the joint torque and muscular contributions to APRs in a cohort of individuals with chronic, recurrent LBP who were not in an active pain episode, using the same support surface paradigm.

In order to begin to understand the differences between quiescent and active periods of chronic LBP and their relation to altered motor coordination, Chapter 5 quantified the joint torque contributions and muscle activation patterns of a cohort of individuals with chronic, recurrent
LBP, who are actively seeking treatment, in response to multi-directional support surface translations. It is assumed that this cohort may have had a greater influence of pain behaviours as well as direct pain effects that may exacerbate the altered APRs demonstrated by the quiescent cohort.

Given that the final cohort of individuals with LBP had significant confounding volitional effects of pain on motor coordination, Chapter 6 quantified the joint torques and muscle activation patterns used to perform sit-to-stand (STS) movements. A STS movement is a volitional, activity of daily living that requires sufficient strength and stability to transfer one’s centre of mass (CM) from the large, seated base of support (BOS) to the smaller, standing BOS. The investigation of whether altered movement patterns are present in performing STS movements enables the partitioning out of the potential volitional aims that may have influenced movement patterns from those elicited under conditions of unexpected balance perturbation.
Chapter 2: Literature review

2.1 Epidemiology and etiology of chronic, recurrent LBP

Chronic low back pain is a prevalent and costly condition that affects a large proportion of the population. In fact, it is estimated that 80% of individuals in North America will experience LBP at some point in their adult lives, with an annual incidence in the US population ranging from 15-45% (Andersson 1997). Impairments resulting from LBP have been cited as the most common cause of activity limitations in individuals under the age of 45 (Kelsey et al. 1979). This population is particularly at risk of developing chronic LBP; indeed, approximately 73% of individuals report recurrence of pain within 12 months after an initial pain episode (Pengel et al. 2003) and many of these individuals will embark upon a cycle of pain recurrence (Von Korff 1994) that may last for many years.

The episodic nature of LBP has lead to difficulties in defining chronic vs. acute pain (Kent and Keating 2005). One of the least ambiguous definitions of chronic, recurrent LBP is back pain that is present on either less than (recurrent) or greater than (chronic) half the days in a 12-month period, occurring in multiple episodes, or “flare-ups” (Von Korff 1994). A
flare-up is defined as a phase of heightened pain that is superimposed on a course of chronic pain (Von Korff 1994). This differs from acute pain, which is defined as a sudden onset of back pain without either chronic or recurrent behaviour (Von Korff 1994). For the purposes of this thesis we will use the term “chronic, recurrent” to include individuals from both groups as defined by Von Korff (1994), given the arbitrary demarcation of number of days of pain distinguishing the groups. Although individuals with this type of LBP will experience at least a partial resolution of their symptoms following a flare-up, recurrent episodes will likely continue to occur, suggesting that the underlying cause of the LBP has not resolved, either via spontaneous healing or therapeutic intervention, even in the absence of pain.

Despite the high incidence of LBP in our population and its tremendous impact on individual and society, little evidence is available to identify effective non-pharmacological or non-surgical treatments for the prevention of chronic LBP. The reason for these poor treatment outcomes likely results from inappropriate matching of treatment to the etiology of LBP given that the vast majority of cases of LBP have an indeterminable cause, resulting in treatments being applied to heterogeneous
populations. In fact, it was estimated that greater than 85% of individuals seeking treatment for LBP were diagnosed as having non-specific LBP (Manek and MacGregor 2005; Chou et al. 2007). A further 15% have either possible degenerative changes or instability identified using traditional radiographic techniques, although the back pain experienced by these individuals cannot be definitively linked to these causes (Hart et al. 1995). Thus it appears that standard diagnostic methods have failed to determine the underlying etiology of the majority of LBP cases, suggesting that new methods aimed at quantification of movement patterns might provide insight into causes of LBP.

2.2 Kinesiopathologic model

The Kinesiopathologic Model [KPM; (Sahrman 2002)] provides a conceptual framework with which to understand the underlying mechanisms that may contribute the development and persistence of LBP in the absence of a definitive pathoanatomical diagnosis. This model contends that for optimal functioning of the body, precise movements of the body’s segments must be maintained to minimize stress on the tissues within the system, in order to prevent pain and injury. According to the KPM, maintaining movement precision can best be accomplished by
intermittent movements and variability in movement directions as opposed to repetitive movements within a small range of directions. If movements are limited to a small range of directions then maladaptive changes within the system, such as altered passive tissue properties or muscle recruitment patterns could result in changes to the biomechanical and neuromuscular components of the system. These alterations could lead to a failure to maintain movement precision, leading to an increased susceptibility to microtrauma, pain and potentially significant injury to the spinal system. This is in contrast to traditional models that suggest that a loss of movement precision is the result of underlying pathology (Hislop 1975).

The KPM model suggests that with regard to LBP, individuals demonstrate a “directional susceptibility to movement” (Sahrmann 2002) that may result from repeated movements in a specific direction and/or the use of prolonged postures that increase lumbar flexibility within a specific direction. These patterns become reinforced throughout an individual’s daily movements, contributing to tissue stress and possibly LBP. Because these movement patterns are well established, symptom recurrence and further injury are likely and may form the basis of the chronic, recurrent
pain bouts characteristic of this group of individuals with LBP. These directional susceptibilities to movement have been characterized as specific movement impairment syndromes, of which five have been identified for the lumbar spine (lumbar rotation, lumbar extension, lumbar flexion, lumbar rotation with extension and lumbar rotation with flexion). These impairment syndromes are identified through observation of abnormal movement patterns and movements that result in symptom reproduction.

The theories of the KPM can be applied to rehabilitation intervention given that a major tenet of the model is that movement precision can be re-established through the normalization of movement patterns. It suggests that by ameliorating impaired movement patterns it is possible to induce changes both at the tissue and neuromuscular levels that could allow the individual to return to increased variability and distribution of movement and therefore, appropriate control of movement precision. Thus the movement impairment syndromes provide targets for movement re-education that attempts to modify functional activities and trunk movements in a direction-specific manner with the goal of normalization and redistribution of trunk movement and alignment (Maluf et al. 2000;
Van Dillen et al. 2005). The strength of this approach is that it attempts to classify individuals into subgroups that may benefit from specifically targeted interventions as opposed to more generalized treatment programs applied to a heterogeneous population of individuals with LBP.

While the outcomes for treatment based on this model have been promising (Harris-Hayes et al. 2005), an understanding of the extent of the underlying tissue and neuromuscular changes induced by altered movement patterns remains to be determined. Given that the KPM emphasizes the relationship between altered movement patterns and these underlying changes in the development and persistence of pain it is important to investigate what movement patterns, both during volitional movements and unexpected balance perturbations, persist between, or are related to, pain episodes.

### 2.3 Automatic postural responses in healthy individuals

In response to unexpected perturbations of balance either via support surface translation or applied force, an APR is elicited in order to regain static equilibrium (Horak and Macpherson 1996). APRs are obligatory responses to unexpected balance perturbations whose latencies are of
sufficient duration to involve polysynaptic connections but are shorter than cortically-initiated volitional movements. These robust responses that occur at a latency of 70-100 ms after perturbation onset, can be modified based on higher level task intentions and previous knowledge of the perturbation (Horak et al. 1989), indicating that although the corrective responses are not generated by the cortex, descending input can alter the pattern but not the timing of response (Horak and Macpherson 1996). While the timing of this response is relatively fixed, the response magnitude elicited can be changed by the context of the applied perturbation including the direction (Henry et al. 1998b) and velocity (Horak et al. 1989; Park et al. 2004) of the perturbation, initial position of the body prior to perturbation (Horak and Moore 1993; Tokuno et al. 2006), or central set (Horak et al. 1989; Horak and Macpherson 1996).

Three postural strategies have been characterized depending on the type and characteristics of the perturbation. The “ankle strategy”, in which the body behaves like an inverted pendulum (Kuo and Zajac 1993), uses ankle plantar & dorsiflexion torque to oppose the motion caused by the perturbation (Horak and Macpherson 1996). This strategy, which does not sacrifice postural alignment of the trunk, is often used as a first line of
defence against balance perturbations that are small in magnitude and slow in velocity, even though it is not the most effective for controlling the body’s centre of mass (CM) within the base of support (Horak and Macpherson 1996). The “hip strategy” involves torques created at the hip, which act to divide the body into two segments with articulation at the hip (Horak and Macpherson 1996), in other words behaving like a double-inverted pendulum (Kuo and Zajac 1993). In doing so the centre of pressure (COP) is moved in the direction of the perturbation but the CM is decelerated rapidly using the inertia of the trunk (Kuo and Zajac 1993). This strategy is most commonly demonstrated in healthy individuals following perturbations of greater magnitude and velocity or when standing on a narrow beam or compliant surface (Horak and Macpherson 1996). Although originally thought to be discrete strategies, responses to most perturbations use some combination of both the ankle and hip strategies. Indeed, Park et al. (2004) have demonstrated relative scaling of the hip and ankle contributions to the corrective responses dependent on perturbation magnitude. A third option for responding to support surface translation is to employ a “stepping strategy”, elicited primarily in response to very large or fast translations (Maki and McIlroy 1993), however, given
that stepping responses were omitted from analysis in this set of studies this discussion will be confined to feet-in-place responses only.

Although responses to support surface translations have been well-characterized in both healthy and pathologic populations and under varied experimental conditions, most studies have been limited to backwards perturbations that elicit forward body sway, with little attention paid to forward and lateral, or diagonal perturbation directions. Of interest is whether the strategies elicited following backward translations are the same as those used in response to varied directions of perturbation or whether the strategies employed are unique to the direction and type of perturbation. Two main viewpoints exist, one that espouses differential control in the sagittal and frontal planes of the body (Matjacic et al. 2001; Allum et al. 2003; Gruneberg et al. 2005; Kung et al. 2009), including different triggers for corrective responses depending on the plane of the perturbation and the second suggests that common strategies are used to respond to balance perturbations regardless of the direction of perturbation (Moore et al. 1988; Henry et al. 1998b; Henry et al. 1998a).
Differences in corrective responses (including surface reaction forces, myoelectric and kinematic variables) to sagittal vs. frontal plane perturbations have been documented, however, it is possible that these differences result only from the anatomical constraints of the body unique to each direction and that the overall neural control strategies governing the responses do not differ with direction. For instance, corrective torque responses to lateral perturbations demonstrate earlier latencies than those demonstrated in the sagittal plane (Allum and Honegger 1992). However, this likely results from the constrained motion at the knee in the frontal plane which acts to couple ankle inversion/eversion and hip abduction/adduction (Day et al. 1993), transmitting the perturbation to the inertially dominant proximal joints. In other words the body’s geometry provides for a greater passive response to balance perturbation in the lateral plane, possibly requiring less of an active muscular contribution (which would be delayed relative to the passive inertial response). In fact, postural responses to lateral perturbations under conditions of narrow vs. wide stance widths support greater reliance on active mechanisms with narrow stance and an increasing passive contribution with increasing stance width (Day et al. 1993; Henry et al. 2001), suggesting that body
geometry does influence corrective responses but may not represent a shift in neural control strategy.

Common to all perturbation directions appears to be a shared responsibility for the corrective responses between both proximal and distal joints, although consistent with the arguments of Park et al. (2004) the relative contribution of one joint vs. another may vary depending on the constraints of the task. In the lateral plane, the knee is not capable of abduction/adduction movement and the capability of ankle inversion/eversion to resist perturbation is small given the small moment arm available in this plane (Winter et al. 1996; Rietdyk et al. 1999). Thus in the lateral plane it appears that hip abduction contralateral to the perturbation largely governs the corrective response to support surface translation (Meyer et al. 2004). In contrast, responses to backwards perturbations appear to be more reliant on the ankle joint, given the large moment arm available for ankle plantar flexion (Mihelj et al. 2000). Yet, despite the relative scaling of the corrective responses between joints, similar active control strategies appear to be used to respond to perturbations in both the anterior-posterior (AP) and lateral planes. Both AP and lateral control used a load/unload force couple strategy about the
fore/aft foot for AP and about the left/right foot for lateral directions, acting in each case to move the body CM back towards the center of the BOS (Henry et al. 1998a). Therefore, although the corrective responses to balance perturbations may be distributed among the joints dependent upon the direction-specific constraints imposed by body geometry, the governing strategy appears to be the same regardless of perturbation direction. Indeed, early proximal muscle activation at the trunk with a concomitant distal to proximal activation sequence, regardless of perturbation direction (Henry et al. 1998b) suggests that the neural strategy governing the corrective response includes use of both the hip and ankle strategies irrespective of direction.

Admittedly it is difficult to determine the governing neural strategy and whether or not these direction-specific differences are due simply to differences in plane-specific body geometry. Quantification of joint torques may provide further insight into this issue by providing a net representation of the ultimate output of each joint as part of the corrective response. However, few studies have quantified net joint torques of more than the ankle joint and in multiple directions of perturbation, nor have they included separate hip and trunk torques. In general, corrective responses
to backwards perturbations consist of ankle plantarflexion, knee flexion and hip and trunk extension torques (Allum and Honegger 1992; Runge et al. 1999). Torque responses to forward perturbations have rarely been studied beyond ankle contributions that demonstrate well-documented dorsiflexion torque in response to perturbation-induced backward body sway (Horak and Moore 1993; Gu et al. 1996). Corrective torque responses to lateral perturbations are dominated by primarily hip abduction and ankle inversion (contralateral to the side of the perturbation), as well as ipsilateral trunk bending torques (Meyer et al. 2004). While it seems that these directional differences could be related to direction-specific differences in body geometry, a systematic analysis of corrective torque responses including forward, backward, lateral and diagonal combinations of these perturbation directions, could clarify whether the neural control strategy governing these responses is the same or if in fact sagittal vs. frontal plane responses are controlled independently.

Compared to the study of torque responses a greater effort has been devoted to the study of the muscular activation responses following these types of external perturbations. In general, a distal-to-proximal activation
sequence from ankle to hip (Horak and Nashner 1986; Moore et al. 1988) has been reported regardless of the direction of perturbation, although often with a concurrent early activation of the trunk (Henry et al. 1998b; Carpenter et al. 1999) and/or neck musculature (Keshner et al. 1988).

Most muscle responses to unexpected support surface translations have been characterized by activation patterns consisting of one or two directions of maximal amplitude for each muscle following perturbation, occurring at the same latency (Henry et al. 1998b). Muscles that demonstrated bipolar activation did so with two distinct latencies, for example the Rectus Abdominus was recruited at an early latency for posterior perturbations, implicating it as a prime mover contributing to the hip strategy, while it was activated at a later latency for anterior perturbations, possibly being used as a stabilizer (Henry et al. 1998b).

Activation of the trunk musculature may be of particular importance to individuals with LBP and may be the most affected by chronic spinal pain. Although to our knowledge, trunk muscle myoelectric responses have not been studied in this cohort, these muscular responses have been well characterized in healthy individuals (Henry et al. 1998b; Carpenter et al. 2008; Preuss and Fung 2008). In general, the deeper abdominal muscles (Transversus Abdominus, Internal Oblique) appear to be active similarly
regardless of perturbation direction with the more superficial External Oblique and Rectus Abdominus demonstrating specific directional tuning of activity levels (Carpenter et al. 2008; Preuss and Fung 2008). Early responses (0 to 250 ms following perturbation onset) in the paraspinal muscles were generally monopolar and oriented to directions of loading (i.e. right side muscles were most active following forward translations with a leftward component) where they could contribute to an early hip/trunk strategy (Preuss and Fung 2008). Responses from 250-500 ms following perturbation onset demonstrated bipolar tuning curves with responses direction along the contralateral-anterior, ipsilateral-posterior diagonal (Preuss and Fung 2008), generally supporting previous findings (Henry et al. 1998b) although these responses were integrated over different windows based on unique perturbation characteristics, likely explaining the differences demonstrated between the two studies.

Although muscle responses have been shown to be direction-specific, Torres-Oveido and Ting (2007) reported that responses to all directions of perturbation could be recreated using only 6 muscle synergies, whose amplitudes were modulated based on the direction of perturbation. In general, two synergies each were present for both the anterior and
posterior perturbation directions representing the ankle and hip strategies in each case. Lateral perturbation directions used a fifth synergistic muscle grouping, representing a hip/trunk strategy, and a final grouping was used to support a “knee flexion” strategy that was used by some subjects, particularly following anterior perturbations (Torres-Oviedo and Ting 2007). Thought not formally grouped into synergies, similarly organized muscle responses have been reported (Henry et al. 1998b). Therefore, it appears that while the muscle synergies are highly direction-specific, their ultimate output regardless of perturbation direction conformed to combinations of the ankle and hip strategies, modulated with direction. Further insight into the common use of modulated ankle and hip strategies regardless of perturbation direction can be accomplished through a systematic analysis of torque and electromyography (EMG) contributions to the postural responses elicited by platform perturbations.

2.4 Automatic postural responses in individuals with neurological or musculoskeletal pathology

Investigations of patient populations have provided significant insight into movement control, providing information on the importance of various nervous system structures to postural control. Individuals with Parkinson’s
disease demonstrate increased co-activation of agonist-antagonist leg musculature, decreased trunk rotation and ankle torque (Carpenter et al. 2004a) but similar latencies to healthy individuals in their corrective responses to postural perturbations (Horak et al. 1996). In addition, these individuals are unable to appropriately scale the amplitude of their corrective responses to perturbation amplitude and velocity, implicating the basal ganglia in the control of baseline muscle tone as well as the development of appropriate levels of force. In contrast, individuals with cerebellar disorders demonstrated larger amplitude responses to surface translations, although with no change in the onset times of the postural responses (Horak and Diener 1994). Individuals with cerebellar lesions were unable to scale their responses based on previous experience of the perturbation, achieving scaled responses only when aided by somatosensory feedback from altered perturbation velocity (Horak and Diener 1994). It is thought, therefore, that the cerebellum is related to accurate control of postural response gain (Mummel et al. 1998). In patients with vestibular deficits the hip strategy is either absent (Horak et al. 1990) or delayed (Allum and Honegger 1992), although the lower leg responses appear to be unaltered compared to healthy individuals. Individuals with induced somatosensory loss of the feet and ankles
demonstrated responses with increased reliance on the hip strategy with no alterations in the timing of the response (Horak et al. 1990; Meyer et al. 2004). However, a patient with total leg proprioceptive loss demonstrated delayed but not absent postural responses, suggesting that corrective responses may be triggered by a combination of leg, hip and trunk proprioceptive feedback (Bloem et al. 2002). These results suggest that APRs are initiated by both somatosensory and vestibular feedback, that the vestibular system is involved in appropriate implementation of the hip strategy and that the somatosensory system is involved in appropriately scaling the amplitude of the corrective responses (Horak et al. 1990). These findings provide a basis for comparison of the responses in individuals with chronic LBP that may help to clarify which systems may be affected in this population.

Though less well-studied, altered corrective torque responses have been demonstrated in older adults (Gu et al. 1996; Allum et al. 2002), individuals with Parkinson’s disease (Horak et al. 1996; Carpenter et al. 2004a), cerebellar deficits (Horak and Diener 1994), diabetic polyneuropathy (Bloem et al. 2000) and dorsal root ganglionopathy (Bloem et al. 2002), and in healthy adults under conditions of anesthesia-
induced reduced foot plantar sensitivity (Meyer et al. 2004), although most studies reported only ankle torques. Specifically, reductions in active ankle torque production following unexpected perturbations have been identified in individuals with Parkinson’s disease (Horak et al. 1996; Carpenter et al. 2004a) and dorsal root ganglionopathy (Bloem et al. 2002) attributed to heightened background agonist-antagonist activity. Since these studies did not quantify trunk torques it is unknown whether these populations of individuals demonstrated similar torque reductions at the proximal joints, however in both populations increased baseline co-contraction of the proximal musculature is reported that could lead to reduced proximal torques. Individuals with cerebellar deficits and healthy individuals with experimentally reduced plantar sensitivity demonstrated increased ankle torque following support surface translations, indicating that both central and peripheral deficits could cause hypermetric torque responses. In addition, a distal to proximal redistribution of torque contributions to postural responses was demonstrated following induced cutaneous anaesthesia of the soles of the feet, resulting in an increased reliance on the loaded hip and a reduced reliance on the loaded ankle to respond to lateral perturbations (Meyer et al. 2004). Neither aging (Gu et al. 1996) nor vestibular deficits resulted in significant alterations in corrective torque
production, although with increasing perturbation velocity some individuals with vestibular deficits were unable to successfully incorporate hip torque into the postural response strategy (Runge et al. 1998). These findings indicate that both central and peripheral deficits may result in altered response strategies to postural perturbations.

While it is logical that neurological dysfunction can result in altered movement control, it is interesting to consider whether similar deficits in postural control could result from chronic LBP or conversely whether LBP could result from altered postural control. Support surface translations have not been well characterized in the chronic LBP population. Henry et al. (2006) studied the centres of mass and pressure responses to multi-directional perturbations in individuals with a history of LBP but who were not in an active episode of pain. These individuals demonstrated increased CM and decreased COP displacements in the sagittal plane following multi-directional support surface translations suggesting that individuals with LBP used a stiffening strategy to prevent significant movement of the trunk segment. In doing so their stability may have been compromised as evidenced by a reduced margin of stability between the COP and CM [a measure of postural efficacy; (Corriveau H. 2000)].
However, further study including analysis of torque and myoelectric responses is required to confirm whether these patients indeed used a stiffening strategy to respond to perturbations of this type.

Although responses to surface translations have not been well documented in the chronic LBP population, APRs have been studied under different experimental paradigms. Radebold et al. (2000) quantified muscle recruitment patterns in persons with and without LBP who were required to apply an isometric force in either the trunk flexion, extension or lateral bending directions which was subsequently, unexpectedly released. In healthy individuals the responses were to first, silence the agonist muscles and second, initiate activation of the antagonist muscles to slow the momentum of the upper body that results when the resisting force is suddenly removed (Radebold et al. 2000). The patients with LBP demonstrated delayed offset of agonist and onset of antagonist of the first muscle reaction in both cases and demonstrated co-contraction of a significant proportion of antagonist and agonist musculature (Radebold et al. 2000). Presumably, co-contraction of agonistic and antagonistic musculature may be a strategy used to increase trunk stiffness and prevent excessive motion of the lumbar spine.
2.5 Volitional sit-to-stand (STS) movement patterns and LBP

To fully appraise postural control in individuals with LBP, particularly those experiencing an active pain episode, observation of movement and stability during self-initiated movements must be considered. It has been shown that individuals with LBP demonstrate altered coordination patterns during voluntary movements of sit-to-stand (Coghlin and McFadyen 1994; Gioftsos and Grieve 1996; Shum et al. 2005a; Shum et al. 2007). In fact, the patterns demonstrated by asymptomatic, chronic LBP sufferers were so distinct from those of healthy individuals that neural networks assessing kinematic and kinetic parameters were able to successfully allocate individuals to their respective groups approximately 80% of the time (Gioftsos and Grieve 1996). These patterns are so robust that even in the presence of increased interindividual variability in the LBP group, neural networks could successfully predict who did and did not have LBP (Gioftsos and Grieve 1996).

Two different strategies of accomplishing a successful STS have been demonstrated in healthy individuals (Schenkman et al. 1990; Coghlin and McFadyen 1994). The knee strategy was characterized by high moments
at the knee and the use of the low back and hip extensors to control forward momentum of the body CM and to redirect momentum from primarily forward motion to upward motion (Coghl and McFadyen 1994). This strategy required relatively small extensor moments at the hip and trunk which may be desirable to individuals with LBP but also requires the ability to control forward momentum to prevent loss of balance (Schenkman et al. 1990). The hip-trunk strategy, used increased trunk flexion requiring increased hip and pelvis-trunk extensor moments to control forward momentum and initiate upward motion of the CM (Coghl and McFadyen 1994). This strategy would presumably enable the CM to be moved closer to the standing BOS prior to seat unloading to increase the stability of the movement, coincidently decreasing the required knee moment. Although it would seem logical that LBP patients would preferentially opt for the knee strategy to reduce the magnitude of moments applied about the trunk, LBP patients instead used a modified strategy for the STS motion that served to distribute the moments across all joints (Coghl and McFadyen 1994).

Individuals with LBP demonstrated decreased movement time for the entire STS manoeuvre but spent a greater proportion of the movement in
the ascending phase (from seat-off to full knee extension) which may indicate that these individuals place a “higher priority” on stability of motion than on moments generated at the trunk (Coghlin and McFadyen 1994).

During the ascending phase, the individual must create extensor moments at the lower limb and trunk in order to propel the body CM upward but is constrained to maintain his/her CM within the BOS. The authors suggest that by increasing the duration of the ascending phase by slowing the forward velocity of the CM, the individual could avoid potentially large decelerations of the CM that could necessitate increased hip and trunk joint moments to reduce momentum and maintain stability (Coghlin and McFadyen 1994). To adequately judge whether this is true, the CM velocity in both the horizontal and vertical directions should be quantified. Unfortunately these findings were not presented (Coghlin and McFadyen 1994).

Reduced lumbar spine and hip motions were demonstrated by individuals with LBP performing STS manoeuvres (Shum et al. 2005a). Not only were peak hip and lumbar flexion reduced but there was also a reduction in the relative contribution of the lumbar spine to the STS movement. The relative phase angles of individuals with LBP were altered such that the
lumbar spine led the hip less during the flexion phase, while the hip led the lumbar spine more compared to individuals without LBP (Shum et al. 2005a). In a similar study individuals with LBP also demonstrated reduced sagittal plane lumbar moments at the joint between fifth lumbar and first sacral segments with compensation in other planes of motion (Shum et al. 2007). Unfortunately, these investigations were limited to the lumbar spine and hip with no indication as to whether any modifications were made distally. Given the proximal vs. distal redistribution in torque responses demonstrated by chronic LBP to support surface translations (Jones et al. Submitted) and the proximal vs. distal reweighting of sensory contributions to posture disturbed by tendon vibration (Brumagne et al. 2004), we expect that there may have been compensations for the restricted proximal motion distally. Therefore, a full kinematic, kinetic and myoelectric analysis of the proximal and distal joint contributions to the STS motion should be undertaken, to determine whether similar redistribution of joint contributions occurs in this volitional movement. In addition, no measure quantifying stability throughout the STS motion has been investigated in this population. We believe that these measures are important to understand the underlying aims of the postural control system.
in accomplishing this volitional task within the constraints imposed on the individual who has been experiencing chronic LBP at the time of testing.

2.6 Motor control impairments in individuals with LBP

A growing body of evidence supports motor control impairments associated with chronic LBP, yet the etiology of these impairments has remained elusive. It has been posited that the impairments demonstrated by individuals with LBP may be related to deficits in proprioceptive feedback. Evidence of a reduced ability to accurately reposition their trunk (Brumagne et al. 2000; Newcomer et al. 2000; Descarreaux et al. 2005), redistribution of their postural responses to lumbar and ankle vibration (Brumagne et al. 2004) and altered Hoffman-reflex recruitment patterns attributed to reduced excitability of Ia-afferent fibers (Ginanneschi et al. 2007) support altered proprioception in this population. Individuals with LBP have demonstrated a reduced ability to accurately and precisely reposition their trunk during volitional movements (Newcomer et al. 2000); (Brumagne et al. 2000). These individuals were able to improve their repositioning performance either through extensive practice at repositioning (Descarreaux et al. 2005) or through the use of vibration applied to the Multifidus muscle (Brumagne et al. 2000). While vibration of
the Multifidus leads to reduced accuracy in trunk positioning in healthy individuals, those with chronic LBP actually improved both the accuracy and precision of their movements (Brumagne et al. 2000), possibly mediated by increased noise of the afferent feedback (Cordo et al. 1996). Individuals with LBP have been shown to reweight the proprioceptive feedback from their lumbar spine musculature to more distal ankle muscles more than healthy individuals, as evidenced by larger sway than demonstrated by healthy individuals, following Tibialis Anterior and Triceps Surae vibration than following lumbar paraspinal vibration (Brumagne et al. 2004). The authors suggest that these changes could result either peripherally at the muscles or may involve the central processing of proprioceptive afferent feedback (Brumagne et al. 2004).

Individuals with chronic LBP move more slowly (Novy et al. 1999; Al-Obaidi et al. 2003; Descarreaux et al. 2005; Lamoth et al. 2006b) and produce less isometric force at the knee joint than do individuals with no history of LBP (Verbunt et al. 2005), suggesting that descending inhibition may also be characteristic of chronic LBP. Using superimposed electric stimulation while performing an isometric knee extension task, individuals with LBP demonstrated lower torque production and a greater increase in
torque following stimulation (Verbunt et al. 2005). These findings and those that indicate a still greater reduction in torque for individuals experiencing high levels of pain or with psychologic distress have implicated descending inhibition as a plausible contributor to altered movement patterns in individuals with chronic LBP. In addition, individuals with LBP may have a deliberate intention to restrict lumbar movement, perhaps due to fear of movement or pain secondary to chronic LBP (Descarreaux et al. 2007; Thomas et al. 2007; Thomas and France 2008), which could influence the implementation of a given movement strategy.

Cortical changes have also been demonstrated by individuals with chronic LBP that may support altered descending input as characteristic of this cohort. Increased activity of the somatosensory cortex measured by magnetic source imaging has been demonstrated by individuals with chronic LBP in response to painful electrical stimuli applied to the back, that increased in intensity with length of chronicity (Flor et al. 1997). Increased activity was accompanied by a medial shift in the representation of painful stimuli in the somatosensory cortex to include areas more distal to the representation of pain (Flor et al. 1997). Reductions in thalamic and dorsolateral prefrontal grey matter density that were related to pain
duration have also been demonstrated by magnetic resonance imaging in individuals with chronic LBP (Apkarian et al. 2004), further implicating central mechanisms as being related to LBP. In fact, Jacobs et al. (Jacobs et al. 2010) demonstrated significant correlations of motor readiness potentials with anticipatory postural adjustment latencies and enhanced cortical motor readiness prior to self-initiated arm movements in individuals with chronic LBP compared to those without. These changes are indicative of an increased contribution of the cerebral cortex to postural coordination. Therefore, investigating both APRs and volitional movements in individuals with chronic LBP may provide evidence to determine the extent of peripheral vs. central involvement in the motor control impairments demonstrated by this population.
Chapter 3: Responses to multi-directional surface translations involve redistribution of proximal versus distal strategies to maintain upright posture

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Prior to characterizing the torque and muscular activation strategies of individuals with chronic LBP, it is important to have a basic understanding of the strategies used by individuals who do not have a history of LBP. Although support surface translations have been widely studied in healthy individuals, few studies have examined multi-directional perturbations and none have undertaken a full body analysis of the sagittal and frontal plane corrective torque strategies. This study characterizes these corrective torque responses and attempts to address whether a single, unifying strategy is used or whether each individual direction of perturbation requires a unique response strategy.
3.1 Abstract

Evaluation of postural control in multiple planes is necessary to determine the movement strategies used to respond to unexpected perturbations. The present study quantified net joint torques of the lower limbs and trunk in the sagittal and frontal planes following multi-directional surface translations. Twenty-one healthy subjects stood with feet on separate force plates mounted on a moveable platform, translated unexpectedly in one of 12 directions. Peak net torque magnitudes and latencies following perturbation onset were determined as were the relative contributions of each joint to total torque production. Magnitude of net torque generated by each leg varied by perturbation direction, with the largest individual joint magnitude elicited in directions of limb loading. Relative contributions of individual joint torques to the total response were dependent upon perturbation direction. Results suggest that a redistribution of the relative contributions of hip/trunk vs. ankle strategies occurs dependent on perturbation direction, with a significant contribution by the knee joint in response to forward perturbations. Direction-specific redistribution of proximal vs. distal strategies appears to depend upon the biomechanical constraints imposed by a given perturbation direction. Thus it appears that
sagittal and frontal plane posture-righting responses may not be uniquely controlled, and may instead be governed similarly, with modulation of relative torque contributions among joints when necessary given direction-specific anatomical constraints.

3.2 Introduction

The translating platform paradigm has been an invaluable tool in elucidating the underlying mechanisms of postural control, leading to the understanding of medium latency or automatic postural responses to unexpected perturbations (Nashner 1976; Nashner 1977). Using the translating platform paradigm, these responses, though originally thought to be immutable, have been demonstrated to be robust yet modifiable synergistic responses that are influenced by many intrinsic and extrinsic factors (Horak et al. 1997). It has been well-documented that perturbation characteristics, such as amplitude, velocity (Horak and Diener 1994; Park et al. 2004) and acceleration (Szturm and Fallang 1998), as well as subject characteristics such as prior knowledge of perturbation and central set (Horak et al. 1989) can influence automatic postural responses, demonstrating the adaptability of these responses.
Despite the focus on characterizing postural responses to the translating surface perturbation, several issues remain unresolved. One issue involves the control of posture in response to multi-directional perturbations of the support surface, and whether differences exist between control in the frontal vs. sagittal planes. This is an important question given that many of the unexpected perturbations that occur in vivo involve multiple planes of movement. One viewpoint asserts that responses to anterior/posterior (AP) and lateral perturbations are controlled independently (Matjacic et al. 2001; Allum et al. 2003; Gruneberg et al. 2005). Matjacic et al. (2001) argued that control in the sagittal and frontal planes is decoupled based on the observation that the net joint torque sums in the sagittal (ankle only) and frontal (ankle + hip) planes, respectively, were identical to those elicited in combination forward/backward and lateral perturbations. However, this finding does not necessarily implicate different control mechanisms between the two planes and instead may lend support to the view that a common strategy is utilized regardless of perturbation direction (Henry et al. 1998b; Henry et al. 1998a). Evidence of the onset of trunk velocity in the frontal plane preceding onset in the sagittal plane, following uni- and multi-planar perturbation directions has been used to support a separate organization
of balance responses between the frontal and sagittal planes (Carpenter et al. 1999; Allum et al. 2003). However, differential timing of frontal and sagittal plane trunk movements could reflect the inherent differences in skeletal geometry that serve to transfer the perturbation to the hip and trunk without the damping effect of the knee or significant movement of the ankle in the frontal plane.

A second hypothesis regarding multi-directional control is that the mechanisms for controlling posture in multiple planes are the same yet must act within biomechanical constraints of the body that differ in each direction of movement (Moore et al. 1988; Henry et al. 1998a; Henry et al. 1998b). Henry et al. (1998a) suggest that the fore/aft force couple used to respond to translations in the forward/backward directions is analogous to the load/unload force couple demonstrated in response to lateral translations. The muscle synergies used to generate these reaction forces differ depending upon the direction of perturbation, but the output (surface reaction forces) is similar regardless of direction, suggesting that the strategies used by the nervous system to respond to multi-directional perturbations are similar. Park, Horak & Kuo (2004) determined that scaling of postural response gain occurs in proportion to the
biomechanical constraints induced by the perturbation magnitude, suggesting that a single feedback control scheme is responsible for a range of responses.

To address these conflicting views, analysis of the joint torques generated by the lower limbs and trunk to respond to multi-directional perturbations (including forward, backward and lateral directions) may lead to the determination of a global strategy or set of strategies to maintain balance. Little effort has been devoted to quantification of joint torques of the lower limbs and trunk in response to multi-directional support surface translations, having been reported only in response to backward translations that induce forward body sway (Allum and Honegger 1992; Runge et al. 1999; Bothner and Jensen 2001; Park et al. 2004) and lateral translations inducing left/right body sway (Meyer et al. 2004). In posterior translations a continuum of responses have been identified, characterized by primarily ankle responses to lower velocity perturbations with increasing contribution of hip responses as perturbation difficulty increases, either by increased velocity or decreased support surface size (Horak and Nashner 1986; Runge et al. 1999). Meyer, Oddsson & De Luca (2004) reported a combined ankle and hip response to lateral
translations driven primarily by contralateral hip abduction and contralateral ankle inversion torque. Although some element of the hip strategy is thought to contribute to the response to anterior translations (Henry et al. 1998a), this has not, to our knowledge, been substantiated through analysis of joint torques.

Therefore, the purpose of this descriptive study was to characterize the joint torques at the ankles, knees, hips and trunk, in both the frontal and sagittal planes (knee in sagittal only), in response to unexpected translations of the support surface. We hypothesized that the magnitude of the torque responses (both spatial and temporal parameters) in the frontal and sagittal planes would be modulated based on the specific direction of perturbation.

3.3 Methods
Twenty-one healthy subjects (13 female, 8 male) were recruited from the local community (mean ± SD: age 33.0 ± 10.5 yrs, height 1.71 ± 0.08 m, mass 68.1 ± 12.9 kg). All subjects signed an informed consent document in accordance with University of Vermont Institutional Review Board policy and the rights of the subjects were protected.
Subjects were instructed to stand on a moveable platform, looking forward and with arms hanging comfortably at their sides. Subjects self-selected their stance width and toe out angle (facilitated by having subjects march in place until comfortable). The foot positions were traced and marked on the force platform to ensure correct repositioning of the feet following resting or stepping. All subjects were asked to maintain their balance in response to the perturbation but were intentionally not given any guidance about how to respond. Any trials in which the subjects stepped were discarded and repeated randomly at a later point in the protocol. Subjects were given 2-3 practice trials in each of two directions (180°, directly left; 90° directly forward) prior to which they were told the direction of impending platform movement. Following these practice trials, linear translations of the support surface in the transverse plane were presented in 12 directions of 30° increments (Figure 3-1) with three trials in each direction, and two stimulus profiles. Only the responses from one with nearly constant velocity will be presented here. The platform was translated 9 cm using a ramp and hold waveform (duration of the waveform was approximately 400 ms; Figure 3-1, boxed inset) with a peak velocity of 25.5 cm/s and a peak platform acceleration of 127 cm/s².
Figure 3-1: Time series of joint torque responses to perturbations across all perturbation directions for an exemplar subject. Sagittal plane torques of the trunk and left hip, knee and ankle are presented in response to predominately forward or backward perturbations (unshaded). Frontal plane torques of the trunk, and left hip and ankle are presented in response to primarily lateral perturbations (shaded). All traces range from 250 ms prior to platform perturbation to 1000 ms following perturbation onset on the horizontal axis (vertical line marked as 0 represents the onset of platform movement) and ± 50 Nm on the vertical axis. Schematic stick figures demonstrate the resulting direction of body sway and the dominant torque demonstrated at each joint for each of the cardinal directions. Stick figures are depicted with the subject facing to the right for the sagittal plane views and are viewed from the back for the frontal plane views. The boxed inset contains a displacement (cm) vs. time (ms) trace for platform movement during a rightward (0°) perturbation, as determined by the force plate kinematic markers.
Perturbation trials were randomized for both velocity and direction to ensure that subjects could not predict the perturbation type and were presented in four blocks of approximately 18 trials separated by three mandatory rest periods of approximately 5 minutes each. Subjects were offered additional rest periods if needed but none were required.

Subjects stood with one foot on each of two force platforms (AMTI, Watertown, MA) that were mounted within the moveable platform that was driven by electromechanical motors controlled by commercially available software (Compumotor, Parker Hannifin Corp., Rohnert Park, CA). The output of the two force platforms was sampled at 1000 Hz and filtered with a low pass filter of 10.5 Hz. The center of pressure for each foot was calculated from the recorded forces and moments.

A 3-camera, passive marker system (BTS, Milan, Italy) was used to collect 3-dimensional spatial coordinate data for calculation of the body kinematics. Three non-collinear reflective markers were placed on the head, trunk, both thighs and both shanks in order to determine joint velocities and accelerations in the frontal and sagittal planes. Two markers were placed, one on each force platform, to mark the level of the platform.
Because trials in which the subject stepped were omitted from analysis, it was assumed that the feet remained stationary on the surface allowing calculation of the location of the foot center of mass in a fixed position. Kinematic data were sampled at 50 Hz and filtered using a dual pass 2\textsuperscript{nd} order Butterworth low pass filter with a cut-off frequency of 2 Hz (a cut-off frequency of 5 Hz was used for the force plate markers, which were assumed to have the greatest acceleration due to their close proximity to the translating surface). A pilot study with kinematic data collected at 100 Hz at the fast perturbation velocity (peak velocity of 43 cm/s and peak acceleration of 127 cm/s\textsuperscript{2}) was used to evaluate the frequency spectrum of all markers. It was determined that 90.4 - 94.8\% of the signal power was demonstrated at frequencies of 10 Hz or less, while 94.7 - 97\% of the signal power was demonstrated at 25 Hz or less. Therefore, a sampling frequency of 50 Hz was deemed sufficiently accurate to capture the kinematic data of perturbations of the type presented in this report.

An eight-segment, rigid link model was constructed for each subject, with segment centers of mass determined using regression equations from the literature based on the subject’s height and mass (Zatsiorsky and Seluyanov 1983). The model represented the feet, shanks and thighs.
bilaterally as well as the pelvis and trunk segments. Due to limitations in the resolution of the camera setup, markers delineating the pelvis could not be used. To compensate for this limitation, a “virtual pelvis” was computed as the line that intersected the marker over the 4th lumbar vertebra (distal trunk marker) and was perpendicular to a second line that connected the left and right proximal hip markers. The virtual pelvis was a separate segment with mass and inertial properties assigned using the above anthropometric model and could move independently from the trunk and thigh segments.

Marker position data were used to calculate joint angles, and first and second time derivatives of the joint angle data were calculated numerically with a 4-point weighted least-squares approach to provide angular velocity and acceleration data. This approach fits a quadratic to four data points which can then be used to calculate the 1st and 2nd derivatives at the 2nd point in the series. Force data were low pass filtered at 10 Hz (2nd order, dual pass, Butterworth filter) and re-sampled at 50 Hz in synchrony with the kinematic data. Kinematics and force data were used to compute ankle, knee (sagittal only), hip and trunk (relative to pelvis) net joint torques in the frontal and sagittal planes (SD/Fast, Needham, MA, USA)
through inverse dynamics techniques. SD/Fast prescribes joint motion to follow experimentally-measured trajectories (joint angular displacement, velocity, acceleration) and applies measured ground reaction forces to the feet, calculating the net “hinge torques” necessary to generate the required motions. Knee abduction/adduction torques were not computed given the negligible motion of the knee in the frontal plane. These net joint torques represent the sum of all torques acting at a given joint, including the gravitational torque (due to the action of gravity at each segment center of mass), inertial torques (due to intersegmental interactions and linear and angular accelerations of the segments) and the muscle torque (which includes both the passive and active forces of all muscles acting about a joint as well as passive forces within the joint; (Hoy and Zernicke 1986) and thus represent the net torque output at a given joint.

Peak torque and latency to peak torque were computed for each joint using programmable software (Matlab, Natick, MA, USA). In addition, torque integrals (defined as the area under a given torque vs. time curve) were calculated across seven 75 ms epochs from 25 – 550 ms after perturbation onset. All torque values were normalized to individual subject height (m) and weight (N). The absolute values of the peak torques were
computed to analyze whether the torque magnitude varied with direction, regardless of whether the torque polarity was negative or positive. However, when the direction of perturbation was largely in the plane orthogonal to the plane of the torque being calculated (e.g., a 0° or 30° perturbation with sagittal plane torques, or a 90° or 120° perturbation with frontal plane torques) the polarity of the peak torque responses often varied among trials, although the timing of the peaks across trials was consistent. To address these changes in polarity, statistical comparisons of peak torques included only perturbation directions of 60°, 90°, 120°, 240°, 270° and 300° for sagittal plane torques and 0°, 30°, 150°, 180°, 210°, 330° for frontal plane torques (see Figure 3-1 for a definition of perturbation directions). By limiting the directions of analysis in this way we are confident that we compared only peak torques with the same polarity and did not equate peak torques of opposite polarities. Descriptive statistics were computed for all measures and the responses to the perturbations were analyzed using a repeated measures analysis of variance for each joint torque with direction as the repeated factor (SAS System for Windows, Cary, NC, USA). An alpha level of p < 0.05 was considered statistically significant for main effect comparisons. Post-hoc tests were multiple comparisons of all pair-wise differences with a
Bonferroni correction to preserve the overall significant p-value of 0.05. Therefore the effective significance level for post-hoc tests was set at \( p < 0.0033 \). Given that the Bonferroni correction is a conservative adjustment we will consider trends in post-hoc comparisons with p-values ranging between 0.0033 and 0.05.

### 3.4 Results
Multidirectional support surface perturbations elicited joint torque responses that were dependent on direction, with changes in both spatial and temporal parameters.

#### 3.4.1 Spatial parameters
Frontal plane torque responses to perturbation directions with a large lateral component \( (150^\circ, 180^\circ, 210^\circ, 330^\circ, 0^\circ, 30^\circ) \) were characterized by ankle inversion/hip abduction in the loaded leg (side to which body sway was induced, contralateral to the perturbation) and ankle eversion/hip adduction in the unloaded leg (ipsilateral to the perturbation). In addition, contralateral lateral bending torque (to the side opposite the perturbation) was initially generated at the trunk followed by a larger ipsilateral lateral bending torque at the trunk in response to lateral perturbations (Figure 3-
1). Sagittal plane torque responses to predominantly forward perturbations (60°, 90°, 120°) were characterized by ankle dorsiflexion, knee extension, hip flexion and trunk flexion torques, whereas responses to predominantly backward perturbations (240°, 270°, 300°) demonstrated ankle plantarflexion, knee flexion, and hip and trunk extension torques (Figure 3-1).

In general, peak frontal torque amplitudes differed according to perturbation direction (trunk, F88,5 = 3.61, p = 0.0051; all other joints, F5, 82-88 = 5.89 to 58.55, p < 0.0001; Figure 3-2a-c). The loaded leg demonstrated smaller ankle torque contributions in response to combined lateral and forward perturbation directions (30° for left leg depicted in Figure 3-2b and c, dark shading; 150° for right leg, not shown) relative to combined lateral and backward directions (210° for the right leg, not shown; 330° for the left leg, Figure 3-2b and c, dark shading). In contrast, the unloaded leg demonstrated a larger ankle contribution in the lateral/forward directions (150° for the left leg, Figure 3-2b and c, light shading; 30° for the right leg, not shown did not reach significance) compared to the combination backward/diagonal directions (210° for the left leg, Figure 3-2b and c, light shading; 330° for the right leg, not shown)
or the purely lateral translations (180° for the left leg, Figure 3-2b and c, light shading; 0° for the right leg, not shown, although this did not reach significance for the right ankle). Trunk torque differed significantly only between directions 30 and 150° (p = 0.0031).

Peak sagittal torque amplitudes for the lower extremity joints varied significantly across directions ($F_{5,77.91} = 3.13$ to 42.23, p-values ranged from $p < 0.0001$ to $p = 0.013$; Figure 3-3) and tended to be greater in diagonal perturbations in which the leg was loaded, reaching significance at the ankle and knee (e.g., for a 60° perturbation, the left leg was loaded as the body CM sways backward and to the left side, Figure 3-3b-d, dark shading) than in strictly forward or backward perturbations. This asymmetry reflects the need for increased torque resulting from a weight-shift in the frontal plane toward the loaded leg, particularly for the knee and ankle (Figure 3-3c, d). At the trunk there was a tendency for forward perturbation directions to elicit a reduced peak torque compared to backward directions; in fact responses to backwards perturbations (270°) were greater than all other perturbation directions (p-values ranged from 0.0003 to 0.044).
Figure 3-2: Influence of perturbation direction on joint torque responses in the frontal plane. Group means (± SD) of the absolute values of normalized peak torque magnitude for the trunk (a) and left lower limb joints (b, Hip; c, Ankle) in the frontal plane. Areas of light grey shading represent directions that result in unloading of the left lower limb, while areas of dark grey shading represent directions that result in loading of the left lower limb due to induced body sway. Inset diagrams depict the actual normalized peak torque group averages across all perturbation directions, with polarity included. Significant main effects for direction (*) are indicated by superscripts listed adjacent to the joint of interest.
Figure 3-3: Influence of perturbation direction on joint torque responses in the sagittal plane. Group means (± SD) of the absolute values of normalized peak torque magnitude for the trunk (a) and left lower limb joints (b, Hip; c, Knee; d, Ankle) in the sagittal plane. Light grey shading represents diagonal directions that result in unloading of the left lower limb, dark grey shading represents diagonal directions that result in loading of the left lower limb. Unshaded areas represent purely anterior or posterior perturbations that result in symmetrical loading. Inset diagrams depict the actual normalized peak torque group averages across all perturbation directions, with polarity included. Significant main effects for direction (*) are indicated by superscripts listed adjacent to joint of interest.
Sagittal Plane Torque Responses

a. Trunk: $\text{dir. } p = 0.013$

b. Left Hip: $\text{dir. } p = 0.0003$

Left Leg Unloaded

Left Ankle: $\text{dir. } p < 0.0001$

Left Leg Unloaded

Direction of Perturbation

Forward      Backward      DF
3.4.2 Temporal parameters

In response to lateral perturbations, the latency of peak torque production varied between the directions that resulted in leg loading vs. leg unloading. In general, the hip abductor/adductor peak torque responses occurred later in directions where the leg was loaded than those in which the leg was unloaded (left hip $F_{5,84} = 7.27, p < 0.0001$, Figure 3-4a; right hip $F_{5,82} = 9.78, p < 0.0001$, not shown). Conversely, at the ankle, eversion/inversion peak torque responses tended to occur earlier in the loaded leg than the unloaded leg (left ankle $F_{5,88} = 6.47, p < 0.0001$; right ankle $F_{5,85} = 11.56, p < 0.0001$, Figure 3-4a; not shown), with the exception of the latencies to 30° perturbations in the left leg which occurred at approximately the same time as those of the unloaded leg. In contrast, responses to predominantly forward or backward perturbation directions elicited fewer differences in peak torque latencies. Peak latencies in the sagittal plane torque responses did not demonstrate a direction effect at the distal joints (left ankle $F_{5,91} = 1.86, p = 0.11$; left knee $F_{5,82} = 1.69, p = 0.15$, Figure 3-4b), however at the proximal joints direction effects were demonstrated. Hip torque production peaked earlier in the forward
directions (60°, 90°, 120°) compared to backward (left hip F_{5,80} = 2.92, p = 0.018, Figure 3-4b; with the exception of 240° which was more similar to the forward directions in time) while the trunk demonstrated earlier peak latencies in the backward directions (240°, 270°, 300°) relative to forward (trunk F_{5,77} = 3.29, p = 0.0096, Figure 3-4b; only 90° perturbations reached significance).

3.4.3 Pattern of torque development over time

In order to document the pattern of joint torque responses to multidirectional perturbations, the relative magnitude of joint torque integrals across 75 ms intervals spanning 25–550 ms following perturbation onset were determined (Figure 3-5). In lateral perturbation directions, the earliest response (between 25-175 ms post-perturbation) was initiated largely by unloaded hip adduction (ipsilateral to the perturbation), with some initial contribution of lateral trunk bending contralateral to the perturbation (Figure 3-5a). The largest contributor to the equilibrium response between 175 and 400 ms following perturbation onset was loaded hip abduction, with concurrent increases in loaded ankle inversion torque, both contralateral to the direction of perturbation. In the later phases of the posture-righting response (400–550 ms), the trunk, loaded hip and loaded
ankle continued to dominate, contributing ipsilateral side bending, abduction and inversion torques, respectively. The unloaded ankle

Figure 3-4: Influence of perturbation direction on peak torque latencies in the frontal (a) and sagittal (b) planes. Group means (± SD) of the peak torque latencies are plotted for the frontal plane trunk, left hip and ankle joints (a) and the sagittal plane trunk, left hip, knee and ankle joints (b). Significant main effects for direction (*) are indicated by superscripts listed adjacent to the joints of interest. Light grey shading represents perturbation directions that result in unloading of the lower limb, whereas dark grey shading represents directions that result in loading of the lower limb due to induced body sway. Unshaded areas represent purely anterior or posterior perturbations that result in symmetrical loading between the two lower limbs (sagittal plane only).
a. Frontal Plane Peak Latencies

- **Trunk**
  - $p < 0.0001$

- **Left Hip**
  - $p < 0.0001$

- **Left Ankle**
  - $p < 0.0001$

b. Sagittal Plane Peak Latencies

- **Trunk**
  - $p = 0.0096$

- **Left Hip**
  - $p = 0.018$

- **Left Ankle**
  - $p = 0.11$

**Perturbation Direction**:
- Frontal Plane: 330, 0, 30, 150, 160, 210
- Sagittal Plane: 60, 90, 120, 240, 270, 300
Figure 3-5: Group means of the contributions of lower limb and trunk torques (shaded according to panel legends) to the summed, normalized torque responses across joints. Frontal plane torques are presented in response to purely lateral perturbations (in this case, 180°; a) whereas sagittal plane torques are presented in response to purely forward (90°, b) and backward (270°, c) perturbations. Polarity of the torque responses for each joint (e.g. Ext vs. Flx) are listed in the epoch of greatest total torque production and extend across epochs unless otherwise indicated by bi-directional arrows across epochs where the opposite polarity was elicited. Inset plots depict the relative contribution (%) of each joint to the summed torque response for each of the seven epochs, with no reference to torque polarity.
Summed Normalized Torque Integrals
Total and Relative

180° Perturbation

- Trunk
- Unloaded Hip
- Unloaded Ankle
- Loaded Hip
- Loaded Ankle

UnL Ankle Inv
Trunk R Bend

90° Perturbation

- Trunk
- Hip
- Knee
- Ankle

Trunk Extension
Hip Ext
Knee Ext

270° Perturbation

- Trunk
- Hip
- Knee
- Ankle

Trunk Flexion
Hip Flexion
Knee Ext

Post-Perturbation Epoch (ms)

0 25-100 100-175 175-250 250-325 325-400 400-475 475-550
demonstrated fairly constant eversion torque throughout the movement response.

In forward perturbation directions, the initial response was dominated by early trunk extension torque (from 25-175 ms post-perturbation), with development of a large ankle dorsiflexion torque between 175-400 ms following perturbation onset (Figure 3-5b). Knee extension and hip flexion torques began to increase from 175 ms after perturbation, the knee providing a particularly large contribution from 250-550 ms post-perturbation. Trunk flexion torque developed late (from 400-550 ms post-perturbation) and likely did not provide substantial contribution to recovery of balance given the timing of torque development (after platform deceleration) and its percentage relative contribution to the total torque response summed across joints (Figure 3-5b, inset). In contrast, the early response to backward perturbations was dominated by hip flexion and knee extension torque (between 25-175 ms post-perturbation), followed by a large ankle plantarflexor torque (Figure 3-5c). Ankle plantarflexor torque remained the dominant torque contribution throughout the movement response (175-550 ms post-perturbation) although some knee flexion, hip extension and trunk extension torques increased slightly in the later
portion of the movement response (250-550 ms and 325-550 ms after perturbation onset for the knee and hip/trunk, respectively).

3.5 Discussion

The present study described the net joint torque output of the trunk, hips, knees and ankles in response to multidirectional support surface translations. Individual leg contributions varied with direction, with the largest torque contribution elicited in perturbation directions that resulted in limb-loading. Temporal differences of peak torque development were demonstrated in the frontal plane and at the proximal joints in the sagittal plane.

3.5.1 Strategy selection: sagittal plane responses

Directions of maximum torque contribution for the lower limbs occurred in diagonal perturbations (i.e. those combining forward or backward and lateral components; Figure 3-3b, c and d). Although it has been suggested that the off-axis perturbation directions that elicited maximal responses represent a “directional sensitivity” of muscle and torque responses (Allum et al. 2003; Gruneberg et al. 2005), it is plausible that these leg responses (which may include both stretch reflex and balance-correcting responses)
are a direct consequence of the perturbation-induced body sway that results in asymmetric loading between the lower limbs (Figure 3-3b, c and d). Therefore it is possible that the directional variation in sagittal plane response magnitudes often attributed to specific lines of muscle action, insertion points or joint axes (Henry et al. 1998b; Carpenter et al. 1999) may instead reflect increased torque production and presumably, muscle force, in response to asymmetric limb loading due to induced body sway. Asymmetric loading in this manner may not only apply to the directions of body sway (i.e. inducing limb loading, muscles primarily active as extensors) but also directions of unloading for muscles that are primarily active as flexors. Jacobs and Macpherson (Jacobs and Macpherson 1996) report directions of maximal activity for both flexor and extensor muscle groups that correspond to directions of maximal loading (for extensors) or maximal unloading (flexors) due to the direction of body sway following perturbation. These directions represent multi-directional perturbations and require asymmetric muscle activation regardless of the specific role of the muscle (i.e. extensor vs. flexor) in the corrective response.

Sagittal plane responses to forward and backward surface translations demonstrated patterns that may be viewed as similar when considered in
context of the assumed biomechanic constraints influencing postural responses dependent on the direction of perturbation. An early contribution of the trunk and/or hip was demonstrated (Figure 3-5b and c) with the trunk providing a large contribution to the torque responses following forward perturbations and the hip providing a greater proportion of the response to backwards perturbations. It is possible that since hip extension is limited to only 20° (Reese and Bandy 2002) when the body sways posteriorly (forward perturbation; 90°), the trunk may provide a larger contribution to the response in what could be termed as a “reverse hip strategy”, although with a significant contribution of the knee, consistent with the findings of Henry, et al. (1998a). The knee extension torque elicited following forward perturbations would likely aid transfer of energy from the distal segments to the trunk and pelvis, which would act to dampen the effects of the perturbation, due to their inertia (Hall and Jensen 2002). In the opposite perturbation direction (backward perturbation; 270°), the hip demonstrates an early flexion response characteristic of a “hip strategy”, with limited contribution of the trunk, which may reflect the position of the hip as the first link in the chain from distal to proximal joints that can exert a significant direct effect on the large trunk/pelvis complex. Further study is needed to evaluate whether
these assumed mechanical limitations govern the pattern of torque production elicited.

The role of the knee has been largely ignored in response to surface translations with most assuming minimal contributions to the posture-righting response, particularly in slower perturbations (Matjacic et al. 2001; Park et al. 2004). Park, Horak & Kuo (2004) suggested that while inclusion of the knee into postural models enhanced the ability of the model to predict experimental data, it was deemed too computationally complex to include for the reduction in error gained. Knee flexion torques similar to those elicited in the current study have been reported following backward surface translations, both experimentally (Runge et al. 1999) and theoretically (Allum and Honegger 1992), although the relative contributions of this joint to the posture-righting responses were not directly compared to the other joints. Hall & Jensen (2002) reported significant contributions of the knee to the recovery of balance following forward perturbations, demonstrating the contribution of the knee to energy absorption used to minimize destabilization of the trunk. Our findings support an increased role for the knee in response to both forward and backward perturbations, such that the relative contribution of the knee
to the corrective response parallels and may exceed that of the hip, especially in response to forward perturbations (Figure 3-5b and c).

3.5.2 Strategy selection: frontal plane responses

Responses to predominantly lateral perturbations were controlled using the load/unload strategy described previously (Winter et al. 1993; Rietdyk et al. 1999; Henry et al. 2001) as demonstrated by increased torque production by the lower limbs in the direction of lateral body sway, and the decreased torque on the opposite side (Figure 3-2b and c). Consistent with the arguments above describing the need for increased torque in directions of limb loading, the directions of purely lateral body sway (rightward, 0° and leftward, 180°) demonstrated increased frontal plane torque production, particularly at the hip and trunk (Figure 3-2). Balance was recovered primarily through the use of contralateral hip abduction, ankle inversion and ipsilateral trunk lateral bending torques (Figure 3-5a), consistent with previous findings (Rietdyk et al. 1999; Meyer et al. 2004). However, a small but not insignificant proportion of the response was initiated by ipsilateral hip adduction and contralateral trunk lateral bending torques, a finding that has not previously been reported (Figure 3-5a). Although these contributions, occurring between 25 and 175 ms post-
perturbation, are small relative to those produced later in the response, the fact that they are maintained and are the dominant corrective torques during this period suggests that they may represent some combination of passive and active responses. Sagittal knee torque contributions to lateral perturbation directions could not be compared statistically (see methods for explanation) and were small relative to the frontal plane corrective torques (inset of Figure 3-3c, directions 0° and 180°). However, it is likely that the bilateral sagittal knee torques (of differing magnitudes or polarities) may have provided some additional contribution to the load/unload strategy of balance recovery following lateral perturbations. The early contralateral trunk lateral bending torque could represent initiation of what has been termed a “hip strategy” (Horak and Nashner 1986; Kuo and Zajac 1993; Runge et al. 1999) albeit in the frontal plane, reflecting the bending at the hip and trunk in the direction of body sway that aims to reduce the moment of inertia of the body to quickly reverse the direction of body CM motion (Kuo and Zajac 1993). Early ipsilateral hip adduction torque could be the result of muscle spindle discharge caused by sway-induced hip abduction, motion that could act to facilitate the hip strategy in combination with the demonstrated trunk torque.
3.5.3 Control of automatic postural responses: sagittal versus frontal planes

In contrast to the peak latencies in the sagittal plane, which were independent of direction at the distal joints, frontal plane torque latencies demonstrated modulation with perturbation direction at all joints, (Figure 3-4). The explanation for this observation could be three-fold. First, it is possible that control in the frontal plane is more highly regulated than the sagittal plane, due to the fact that loss of balance to the lateral side does not easily lend itself to a stepping strategy (Winter et al. 1996; Zettel et al. 2002). Alternatively, it may be that the perturbation velocity used in this study is less difficult to respond to in the frontal plane given the stability provided by the relatively wide base of support compared to the base of support in the sagittal plane that is dependent on foot length. Thirdly, it is also plausible that the added passive contribution to lateral perturbations (Rietdyk et al. 1999; Henry et al. 2001), due to the relatively wide base of support in this plane (heel to heel distance in the current study, mean ± SD: 21.5 ± 6.6 cm), could diminish the need for a substantial active response by allowing the geometry of the skeletal system in the frontal plane to dictate the response.
Although it has been suggested that postural control in the sagittal vs. frontal planes is de-coupled (Matjacic et al. 2001; Gruneberg et al. 2005), we do not find compelling evidence to support this assertion; nor can we prove definitively that a single control schema is used to control both frontal and sagittal planes. However, our findings point to similarities in control across directions, in both the frontal and sagittal planes. While direction-specific differences in the elicited responses were demonstrated, they can be explained by a simple redistribution of distal and proximal strategies required by the biomechanical constraints influencing postural responses for each direction of perturbation. Both the hip/trunk and ankle strategies emerge in response to all directions of perturbation with the addition of the knee when it is most mechanically advantageous to contribute to the response (i.e. can provide substantial extensor torque in forward perturbations). In fact, it may be that the responses are ordered such that the most distal joint that is best-suited to respond to a given direction of perturbation (given anatomical limitations of joint ranges of motion; (Reese and Bandy 2002) will provide the largest contribution to the postural response (Figure 3-5). For example, in a forward perturbation (90°), the ankle is somewhat limited in its ability to generate sufficient dorsiflexor torque to resist backward body sway, requiring the addition of
significant knee torque to prevent balance loss. In response to a backwards perturbation (270°), the ankle is well-suited to resist forward body sway due to the long lever arm of the foot in this direction, thus the relative contribution of the knee to the posture-righting response is reduced and that of the ankle is enhanced. In addition, the hip and trunk demonstrate early contributions to the postural responses in all directions of perturbations, suggesting that regardless of the perturbation direction an early proximal strategy may be used to rapidly initiate body CM movement over the base of support. Therefore, the direction-specific torque magnitudes elicited by multidirectional perturbations represent a range of responses generated through a relative redistribution of proximal and distal strategies based on the unique set of constraints imposed by skeletal anatomy in response to each direction of perturbation.

3.6 Conclusions

In conclusion, these findings emphasize the importance of considering trunk torques independently from those torques generated across the hip joint given that in all perturbation directions the trunk demonstrates an independent and significant contribution to the posture-righting response. Redistribution of the hip/trunk and ankle strategies appears to occur when
biomechanical constraints render one joint less able to contribute to the postural response. The knee has been overlooked as a potential contributor to the automatic postural response, and appears particularly important in response to forward perturbations, where both the ankle and hip joints are limited in their ability to resist backward sway. Postural responses do not appear to be controlled separately in the sagittal and frontal planes, given that the responses to all perturbation directions are accomplished with an early contribution of the hip and trunk joints and significant corrective torque generated by the joints that are mechanically best-suited to respond given direction-specific biomechanical constraints.

3.7 Acknowledgments

The authors wish to thank Michael J. Rubin, Heather E. Aubin and Kerry A. McCarthy for their assistance in data reduction.

3.8 Grants

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Chapter 4: Individuals with chronic low back pain demonstrate altered distal and proximal torque production to maintain upright posture following unexpected balance perturbations

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The previous study detailed the corrective torque strategies used by healthy individuals to respond to multi-directional support surface translations and demonstrated that proximal and distal torques were redistributed based on biomechanical or anatomical constraints imposed by a specific perturbation direction. This study aims to understand the influence of chronic, recurrent LBP on the automatic postural responses elicited by unexpected support surface translations. Sagittal and frontal plane joint torques and muscular activation patterns were quantified in a cohort of individuals who had a history of chronic LBP but were in a quiescent period of their pain. As such we quantified the underlying
changes in automatic postural coordination that persisted between pain episodes while minimizing the influence of pain on the responses.

4.1 Abstract

Background: There is increasing evidence that individuals with chronic low back pain (LBP) have altered movement coordination, however, the exact nature of this impairment is unknown. Assessing postural control deficits in this population is difficult given the potential confounding influences of pain on voluntary movements and the acute effects of local pain mediators. Therefore, to minimize these effects we characterized automatic postural responses to multi-directional support surface translations in a cohort of individuals with a history of LBP but who were not in an active episode of their pain. Methods: Twenty subjects with and 21 subjects without chronic, recurrent LBP stood on two force plates that were translated unexpectedly in 12 directions. Three-dimensional kinematics were combined with anthropometric equations and ground reaction forces to calculate net joint torques of the ankles, knees (sagittal only), hips and trunk in the frontal and sagittal planes. Peak torques and torque integrals were used to estimate active and initial contributions, respectively, to the postural responses. Findings: Individuals with LBP
demonstrated increased sagittal plane ankle torque during the initial epoch and reduced frontal and sagittal plane peak trunk torques following perturbation. *Interpretation:* Therefore, it appears that individuals with LBP may have increased intrinsic stiffness at the ankle and may use a strategy of equivalent agonist-antagonist torque production, likely due to co-contraction of musculature, to respond to unexpected perturbations of the support surface. Notably, these motor control alterations have persisted between active pain episodes and may be linked to the chronicity of LBP experienced by this cohort of individuals.

4.2 Introduction

Low back pain (LBP) is a common condition that incurs a great financial burden on the individual and society at large, and is the most common cause of job-related disability in the United States. Despite the high prevalence of this disorder, little is known about the etiology of the largest proportion of individuals with LBP; those characterized as having LBP that is “non-specific” in origin and recurrent in behaviour (Von Korff 1994).

Altered movement patterns have been quantified in individuals with LBP that suggest underlying changes in neuromuscular control of both
voluntary (Coghlin and McFadyen 1994; Gioftsos and Grieve 1996; Hubley-Kozey and Vezina 2002; Lamoth et al. 2006b) and reactive movements (Radebold et al. 2000; Henry et al. 2006). It is unknown whether these changes developed following the initial pain episode or may have contributed to the development of the original onset of pain. To begin to address this issue, quantification of movement patterns in this population may clarify the relationship between neuromuscular impairments and recurrent episodes of pain and may lead to more efficacious treatment. However, studying a population with recurrent pain can be difficult given the residual effects of pain on movement even after the pain has resolved. Individuals with LBP may choose to move in a given way in order to minimize forces applied to painful structures (Porterfield and DeRosa 1998; McGill 2002) or merely in the anticipation of pain (Al-Obaidi et al. 2003; Moseley et al. 2004b), obscuring or exaggerating the underlying neuromuscular impairments that may be contributing to the development or persistence of LBP. These confounding factors are of particular concern when studying volitional movements. In fact, while individuals with LBP may choose to move at slower velocities (Simmonds et al. 1998; Al-Obaidi et al. 2003; Lamoth et al. 2006b) or with reduced force production (Verbunt et al. 2005), they are often able to
achieve higher than preferred movement velocities when challenged
(Lamoth et al. 2006a; Lamoth et al. 2006b; Lee et al. 2007), suggesting
that volitional factors may confound the ability to detect underlying
neuromuscular impairments resulting from the LBP episode.

In order to minimize volitional effects it is important to use a paradigm that
is less susceptible to these influences. The support surface translation
paradigm has been used to study automatic postural responses (APRs;
for a review see (Horak and Macpherson 1996), obligatory responses to
unexpected balance disruption, and can provide a window into movement
strategies that result without initiation by the motor cortex. The APR
responses are robust and well-documented and provide a systematic
means of investigating postural control. Feet in place responses to
unexpected support surface translations largely incorporate two strategies,
an ankle strategy (relying primarily on generation of corrective ankle
torque to return the body to equilibrium; (Runge et al. 1999) and a
hip/trunk strategy (relying on hip/trunk flexion in the direction of body sway
following perturbations acting to quickly accelerate the body centre of
mass over the base of support; (Kuo and Zajac 1993). These two
strategies are not discrete entities; rather, a continuum of responses using
some combination of these two strategies has been demonstrated based on subject specific thresholds (Runge et al. 1999), biomechanical constraints (Park et al. 2004) and different directions of perturbation (Hall et al. 1999; Matjacic et al. 2001; Meyer et al. 2004; Jones et al. 2008).

We have reported on postural responses to multi-directional translations of the support surface in individuals with non-specific, recurrent LBP (Henry et al. 2006). Following unexpected perturbations, individuals with LBP demonstrated delayed centers of pressure and mass responses, increased center of mass excursion and a reduced margin of stability (center of pressure minus center of mass), largely in the sagittal plane, compared to individuals with no history of LBP. These results suggest that individuals with LBP may use an alternate strategy to respond to unexpected support surface translations; one that may afford them less stability in the sagittal plane. In order to quantify the postural strategies being used by this population net joint torque responses to these perturbations must be investigated to determine whether strategy selection is altered in this group.
Therefore, the purpose of this study was to compare the APRs of individuals with and without LBP to multi-directional support surface translations using net joint torques computed at the trunk, hip, knee and ankle as an indicator of the movement strategy used to govern the corrective response. We have chosen to study a cohort of patients reporting a history of chronic LBP but not in an active pain episode at the time of testing, to reduce the direct influence of pain on the elicited responses. Given the redistribution of proximal vs. distal strategies identified in individuals without a history of LBP (Jones et al. 2008) and the potential inability or reluctance of individuals with LBP to move and/or create force at the trunk (McGill 2002), we hypothesized that individuals with LBP would demonstrate altered responses that reflect adoption of a strategy of stiffening at the trunk in order to reduce the amount of trunk torque required to effectively respond to unexpected perturbations of equilibrium.

4.3 Methods

Twenty subjects with chronic, recurrent (Von Korff 1994) LBP (11 female, 9 male) were recruited from the local community through posted advertisements and word of mouth [mean (SD): age 39.9 (14.5) yrs, height
1.73 (0.14) m, mass 75.9 (16.8) kg. Twenty-one healthy subjects (13 female, 8 male) were similarly recruited from the local community [mean(SD): age 33.0 (10.5) yrs, height 1.71 (0.08) m, mass 68.1 (12.9) kg]. Subjects with LBP were excluded (by clinical exam or interview) if they had pain below the knee consistent with a disc herniation, presence of any neurological signs, serious spinal complications (e.g., vertebral fracture, tumor or infection), spinal stenosis, previous spinal surgery, systemic infection, balance or cardiovascular disorders, current pregnancy, history of any surgery in the 3 months prior to testing, uncorrected vision problems, or a severe musculoskeletal deformity (scoliosis or kyphosis) or injury to the lower extremity that would interfere with testing. Subjects were also excluded if they were receiving worker’s or disability compensation for their LBP, or if they were in litigation because of their LBP problem. Subjects were tested when they were not in a recurrence of their LBP (Von Korff 1994; McGorry et al. 2000) and in general, reported pain ratings ≤3/10 on the Numeric Pain Rating Scale (NPRS) the day of testing (3 LBP subjects reported 4 on the NPRS).

Control subjects were excluded if they had a neurological disease or balance disorder, uncorrected vision problems, cardiovascular disorders,
severe musculoskeletal injuries, back pain during the past week, or back pain during the prior 12 months that required medical attention or resulted in missed work. All subjects were currently employed at the time of testing or participating fully in their usual role (e.g., full-time student, homemaker).

At the time of testing subject groups did not differ with respect to age, height, weight or body mass index (0.067<P<0.562, Table 4-1). However, subjects with LBP demonstrated greater pain and disability as measured by the McGill Pain Questionnaire (Melzack 1987), NPRS (Stratford and Spadoni 2001) and Roland Morris Disability Questionnaire (Roland and Morris 1983) (P<0.001, Table 4-1). All subjects signed an informed consent document in accordance with University of Vermont Institutional Review Board policy and the rights of the subjects were protected.

Detailed description of the methods used in this study have been published previously (Jones et al. 2008). Subjects were instructed to stand on a moveable platform, looking forward, at self-selected stance width and toe-out angle, and with arms hanging comfortably at their sides. Any trials in which the subjects stepped were discarded and repeated randomly at a later point in the protocol. Subjects were given 2-3 practice trials in each of
two directions (180°, directly left; 90° directly forward) prior to which they were told the direction of impending platform movement. Following these practice trials, linear translations of the support surface in the transverse plane were randomly presented in 12 directions of 30° increments (Figure 4-1) with three trials in each direction, at two velocities (only results from the faster velocity trials will be presented due to a lack of velocity effects).

Table 4-1: Subject demographic information

<table>
<thead>
<tr>
<th>Parameter [Mean ( SD)]</th>
<th>LBP (n = 20)</th>
<th>NLBP (n = 21)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>39.9 (14.5)</td>
<td>33 (10.5)</td>
<td>0.09</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.73 (0.14)</td>
<td>1.71 (0.08)</td>
<td>0.56</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>75.9 (16.8)</td>
<td>68.1 (12.9)</td>
<td>0.10</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>25.2 (3.2)</td>
<td>23.2 (3.4)</td>
<td>0.07</td>
</tr>
<tr>
<td>Gender (# Male/ # Female)</td>
<td>9 / 11</td>
<td>8 / 13</td>
<td>0.65</td>
</tr>
<tr>
<td>McGill Pain Questionnaire</td>
<td>3.45 (2.78)</td>
<td>0.05 (0.22)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>(# of Words Score)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Numeric Pain Rating (/10)</td>
<td>2.00 (1.37)</td>
<td>0.19 (0.40)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Roland Morris Disability</td>
<td>2.35 (2.30)</td>
<td>0 (0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Questionnaire (/18)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stance Width (cm)</td>
<td>20.8 (5.5)</td>
<td>21.5 (6.6)</td>
<td>0.72</td>
</tr>
<tr>
<td>Toe-out Angle (°)</td>
<td>102.1 (4.8)</td>
<td>103.9 (5.5)</td>
<td>0.28</td>
</tr>
</tbody>
</table>

Subjects stood with one foot on each of two force plates (AMTI, Watertown, MA, USA) that were mounted within the moveable platform.
that was driven by electromechanical motors controlled by commercially available software (Compumotor, Parker Hannifin Corp., Rohnert Park, CA, USA). The center of pressure for each foot was calculated from the recorded forces and moments.

A 3-camera, passive marker system (BTS, Milan, Italy) was used to collect 3-dimensional spatial coordinate data for calculation of the body kinematics. Kinematic data were sampled at 50 Hz and filtered using a dual pass 2nd order Butterworth low pass filter with a cut-off frequency of 2 Hz (a cut-off frequency of 5 Hz was used for the force plate markers, which were assumed to have the greatest acceleration due to their close proximity to the translating surface). An eight-segment, rigid link model was constructed for each subject, with segment centers of mass (CM) determined using regression equations from the literature based on the subject's height and mass (Zatsiorsky and Seluyanov 1983). The model represented the feet, shanks and thighs bilaterally as well as the pelvis and trunk segments.

Marker position data were used to calculate joint angles, and first and second time derivatives of the joint angle data were calculated numerically.
Figure 4-1: Time series of joint torque responses to perturbations across all perturbation directions for two exemplar subjects; an individual with LBP (black) and an individual without a history of LBP (grey). Sagittal plane torques of the trunk and left hip, knee and ankle are presented in response to predominately forward or backward perturbations (unshaded). Frontal plane torques of the trunk, and left hip and ankle are presented in response to primarily lateral perturbations (shaded). All traces range from 250 ms prior to platform perturbation to 1000 ms following perturbation onset on the horizontal axis (vertical line marked as 0 represents the onset of platform movement) and ± 50 Nm on the vertical axis. Schematic stick figures demonstrate the resulting direction of body sway and the dominant torque demonstrated at each joint for each of the cardinal directions. Stick figures are depicted with the subject facing to the right for the sagittal plane views and are viewed from the back for the frontal plane views. The boxed inset contains a displacement (cm) vs. time (ms) trace for platform movement during a rightward (0°) perturbation, as determined by the force plate kinematic markers.
with a 4-point weighted least-squares approach to provide angular velocity and acceleration data. Force data were low pass filtered at 10 Hz (2\textsuperscript{nd} order, dual pass, Butterworth filter) and re-sampled at 50 Hz in synchrony with the kinematic data. Kinematics and force data were used to compute ankle, knee (sagittal only), hip and trunk (relative to pelvis) net joint torques in the frontal and sagittal planes (SD/Fast, Needham, MA, USA) through inverse dynamics techniques.

Peak torques and latencies to peak torque were computed for each joint using programmable software as a means of quantifying the corrective torque responses (Matlab, Natick, MA, USA). A torque integral (defined as the area under a given torque vs. time curve) was calculated to quantify the early part of the response reflective of the passive contributions and of the initial conditions prior to perturbation including baseline tonic muscle activity during an initial response period (50-100 ms following perturbation onset). All torque values were normalized to individual subject height (m) and weight (N). The absolute values of the peak torques were computed to analyze whether the torque magnitude varied with perturbation direction, regardless of whether the torque polarity was negative or positive. Statistical comparisons of peak torques included only
perturbation directions of 60°, 90°, 120°, 240°, 270° and 300° for sagittal plane torques and 0°, 30°, 150°, 180°, 210°, 330° for frontal plane torques (see Figure 4-1 for a definition of perturbation directions).

Descriptive statistics were computed for all measures and the responses to the perturbations were analyzed using a repeated measures analysis of variance for each joint torque with perturbation direction as the repeated factor and LBP vs. NLBP as the grouping factor (SAS System for Windows, Cary, NC, USA). Demographic measures (age, height, weight, BMI, stance width, foot angle) were compared using independent samples t-tests, while the proportion of males vs. females was compared using a Chi-square test of independence. Measures of pain and disability were compared using a Wilcoxon Ranked Sum Test. An alpha level of $P < 0.05$ was considered statistically significant for main effect comparisons.

As a post-hoc exploratory analysis to understand the influences of the subject's initial position on the postural responses to surface perturbation during the passive period (50-100 ms following perturbation onset), we computed the mean and standard deviation of the position of the net center of pressure for each perturbation trial, in both the sagittal (COPap)
and frontal (COPlat) planes, across a 200 ms window that ended 40 ms prior to onset of support surface translation. These values were normalized to each subject’s average foot length (for COPap) and stance width (heel-to-heel separation for COPlat) and were compared statistically in the same manner reported for the joint torque variables.

4.4 Results

Following multi-directional platform perturbations, individuals with LBP demonstrated modulation of torque responses with perturbation direction similar to individuals without LBP. However, individuals with LBP demonstrated trends towards reduced initial and active torque production at the trunk and increased initial torque at the ankle.

4.4.1 Effects of LBP on spatial responses

Initial torque responses were modulated with perturbation direction at all joints (Figure 4-2) in both the sagittal and frontal planes by both LBP and NLBP groups (direction: all joints, $F_{5,152-171} = 7.02$ to $146.05$, $P < 0.001$). In the sagittal plane, individuals with LBP demonstrated no differences in initial trunk torque production following perturbations in primarily forward/backward directions ($60^\circ$, $90^\circ$, $120^\circ$, $240^\circ$, $270^\circ$, $300^\circ$; group:
F_{1,39} = 2.01, P = 0.164) but demonstrated a non-significant trend towards reduced left side bending torque at the trunk following only rightward perturbations (0°, 30°, 330°; group X direction: F_{5,174} = 1.96, P = 0.087; Figure 4-2a). In contrast, individuals with LBP demonstrated increased ankle torque production in the sagittal plane that was most evident following backward perturbations (240°, 270°, 300°; group: F_{1,39} = 4.23, P = 0.046; Figure 4-2b), but demonstrated no between-group differences in frontal plane ankle torques (group X direction: F_{5,170} = 1.71, P = 0.134; Figure 4-2b) elicited by left and rightward surface perturbations.

Peak torque production (reflecting active torque production) at all joints in the sagittal and frontal planes were modulated by direction in individuals with and without LBP (direction: sagittal plane peak trunk torque, F_{5,142} = 2.93, P = 0.02; all other joints, F_{5,144-175} = 9.06 to 106.52, P < 0.01).

Individuals with LBP tended to reduce sagittal plane trunk torque following forward/backward perturbations (group: F_{1,41} = 2.94, P = 0.09; Figure 4-3a). In the frontal plane, individuals with LBP demonstrated reduced peak trunk torque across all perturbation directions (group: F_{1,39} = 5.40, P = 0.03; Figure 4-3a). No other differences in active torque production were evident between individuals with and without LBP.
Figure 4-2: Trunk and ankle torque responses in the sagittal and frontal planes during the initial period (50-100 ms following perturbation onset). Polar plots depict group means of the normalized joint torques integrated and averaged over the initial period for the trunk (a) and left ankle (b) comparing the responses of individuals with LBP (black circles) and without LBP (NLBP; grey circles). Although all perturbation directions are shown for demonstration purposes, analyses of perturbation directions for sagittal plane torques included 60°, 90°, 120°, 240°, 270° and 300° and for the frontal plane torques included 0°, 30°, 150°, 180°, 210° and 330°. The shaded directions indicate those perturbation directions resulting in loading of the left leg. The innermost point of each graph represents the maximum negative torque integral value (indicating trunk flexion and right side bending or left ankle dorsiflexion, inversion). The middle black circular axis represents 0 net torque whereas the outer black circular axis represents that maximum positive torque integral value (indicating trunk extension and left side bending, or left ankle plantarflexion, eversion). Trends or significant main effects for group and interactions for group by direction (*) are indicated by superscripts listed adjacent to the joint torque of interest. Significant main effects of direction (P < 0.001) were found at both the trunk and ankle in the sagittal and frontal planes. Note the trend
of reduced initial torque response at the trunk and increased initial torque response at the ankle by individuals with LBP as compared to those without (NLBP).

Polar Plots of Individual Joint Torques During the Passive Period: 50 - 100ms post-perturbation

a. Trunk

b. Left Ankle

Sagittal Plane Torques  
Frontal Plane Torques
Figure 4-3: Peak joint torque responses in the sagittal and frontal planes at the trunk (a), and left lower limb (b, hip; c, knee, and d, ankle). Polar plots depict group means (± SD) of the absolute values of normalized peak torque magnitudes comparing individuals with LBP (black circles) to those without LBP (NLBP; grey circles). Although all perturbation directions are shown for demonstration purposes, analyses of perturbation directions for sagittal plane torques included 60°, 90°, 120°, 240°, 270° and 300° and for the frontal plane torques included 0°, 30°, 150°, 180°, 210° and 330°. Trends or significant main effects for group (*) are indicated by superscripts below the joint of interest. All joints demonstrated significant main effects of direction (P < 0.01). Note the reduced peak torque magnitude by the individuals with LBP at the trunk compared to individuals without LBP (NLBP).
Polar Plots of Normalized Peak Torque Amplitudes

a. Trunk
   Late Extension-Flexion
   Late Left-Right Side Bending

b. Left Hip
   Extension-Flexion
   Abduction-Adduction

c. Left Knee
   Flexion-Extension
   NLBP
   LBP

d. Left Ankle
   Plantar-Dorsiflexion
   Eversion-Inversion
Mean initial position of the net COPap varied with direction only in the sagittal plane (direction: F$_{5,170} = 9.57$, $P < 0.01$), such that the initial position prior to perturbations primarily in the forward directions (60, 90, 270°) was located slightly more forward than prior to perturbations primarily in the backward directions (240, 270, 300°) for both groups. However, this difference was very small (on average 3% of total foot length representing less than approximately 1 cm discrepancy between directions) and, although it was statistically significant, was not likely physiologically significant (Horak and Moore 1993). No differences between groups (mean COPlat, group: F$_{1,36} = 0.00$, $P = 0.99$) or among perturbation directions were detected in the mean initial net COPlat position in the frontal plane (direction: F$_{5,172} = 0.45$, $P = 0.82$). However, the standard deviation of initial net COPap was significantly reduced in individuals with LBP regardless of perturbation direction (SD of COPap, group: F$_{1,41} = 5.21$, $P = 0.03$). No differences in the standard deviation of initial COPlat were detected either across directions (SD of COPlat, direction: F$_{5,177} = 0.55$, $P = 0.74$) or between groups (group: F$_{1,39} = 0.31$, $P = 0.58$).
4.4.2 Effects of LBP on temporal responses

There were no group differences in either the frontal or sagittal planes for the latency to peak torques (group: $F_{1,31-40} = 0.03$ to $2.72$, $P$-values ranged from $0.11$ to $0.86$) with the exception of left and right hip extension/flexion. Individuals with LBP demonstrated shorter times to right peak hip torque (group: $F_{1,35} = 5.6$, $P = 0.024$) with a similar trend in the left hip (group: $F_{1,37} = 3.31$, $P = 0.08$), in both extension and flexion, compared to healthy individuals.

4.5 Discussion

In response to multi-directional translations of the support surface, individuals with LBP demonstrated altered postural strategies that incorporated responses with increased initial ankle torque response amplitudes in the sagittal plane and with reduced active trunk torque amplitudes, in both the frontal and sagittal planes, with no alterations in the timing of the torque responses.

4.5.1 Individuals with LBP may have altered intrinsic stiffness

Increased intrinsic stiffness, generated by the viscoelastic properties of the joint, muscles and connective tissue, varies with joint position and with
activation level of the associated muscles (Ludvig et al. 2007). Therefore, intrinsic stiffness includes not only the passive tissue properties but also the mechanical consequences of tonic muscle activity even without additional dynamic (reflex or voluntary) muscle activity. The epoch chosen to evaluate these mechanical and neural contributions to the intrinsic joint stiffness was between 50 and 100 ms following perturbation onset, to minimize any effects of initial platform acceleration or perturbation-induced stretch activation given an assumed electromechanical delay of 25-50 ms (Allum and Mauritz 1984). Thus it is presumed that this period would quantify the passive tissue alterations potentially associated with LBP, as well as the initial conditions prior to the surface perturbation including baseline tonic muscle activity. During this period we found increased ankle torque amplitude, largely following backward perturbations that induced forward body sway (Figure 4-2b), that may be caused either by increased periarticular tissue stiffness or increased tonic baseline activity of the ankle musculature.

Increased periarticular tissue stiffness can occur through connective tissue remodelling of both ligamentous and capsular tissues, as well as perimuscular connective tissue, as a consequence of reduced movement
and/or habitual or sustained postures (Williams et al. 1988; Langevin and Sherman 2007). Individuals with chronic LBP have demonstrated movement reductions specifically at the hip and trunk (Mok et al. 2004; Shum et al. 2005a) and have been shown to stand with their COP in a more posterior position (Byl and Sinnott 1991; Mientjes and Frank 1999). A habitually posterior COP position could result from tonic increases in muscle tension in the ankle plantar flexor muscles that could alter the intrinsic stiffness about the ankle joint. Increased stiffness could result in increased ankle torque that would be most evident in the directions resulting in stretch of the ankle plantar flexors (i.e. forward sway elicited by backward perturbations), consistent with our findings.

Enhanced baseline tonic activity of the ankle muscles could also explain the increased ankle torque amplitude demonstrated during the passive period by individuals with LBP. Intrinsic stiffness has been shown to vary with the level of activation of the associated muscles such that ankle stiffness increased in parallel with voluntary ankle torque (Mirbagheri et al. 2000), created either by the ankle dorsi or plantar flexor muscles (Weiss et al. 1988). Although the magnitude of change in ankle stiffness for a given change in ankle torque remains controversial (Mirbagheri et al. 2000;
Loram and Lakie 2002) it is likely that even small levels of ankle stiffness
due to increased tonic activity could influence the destabilizing effect of
postural perturbation during this early period (Fitzpatrick et al. 1992).
Individuals with LBP may increase the ankle stiffness in a feedforward
manner (Gatev et al. 1999) to prevent potential destabilizing postural sway
given the impending perturbation. Our findings of a reduced COP sway in
the AP direction would be consistent with this argument.

In the frontal plane, no differences in torque during the initial period were
detected at the ankles, knees or hips, although there was a trend towards
reduced trunk side bending torque following rightward perturbations
(inducing leftward body sway) by individuals with LBP (Figure 4-2a; group
X dir: $P = 0.087$). It is unclear why trunk side-bending torque would be
reduced by individuals with LBP only in response to rightward
perturbations. It is possible that the LBP cohort under study may have
asymmetries in the location of their pain or, in the musculoskeletal
impairments associated with their pain, that could limit torque production
on one side of the body. Based on the data obtained in the current study
we cannot definitely attribute the observed torque asymmetries to this
explanation.
4.5.2 Altered active torque contributions to APRs occur at proximal joints

In general, individuals with a history of chronic LBP demonstrated active torque responses to multi-directional surface translations that were similar in pattern and in magnitude across lower extremity joints to those of healthy controls (Figure 4-3b-d) (Allum and Honegger 1992; Runge et al. 1999; Meyer et al. 2004; Park et al. 2004; Jones et al. 2008). However, individuals with LBP demonstrated reduced sagittal and frontal plane peak trunk torques regardless of perturbation direction, compared to those of the control group (Figure 4-3a).

Reductions in active torque production albeit at the ankle joint following unexpected perturbations have been identified in individuals with Parkinson’s disease (Horak et al. 1996; Carpenter et al. 2004a) and dorsal root ganglionopathy (Bloem et al. 2002) that were attributed to heightened background agonist-antagonist activity. Since these studies did not quantify trunk torques it is unknown whether these populations of individuals demonstrated similar torque reductions at the proximal joints, however in both populations increased baseline co-contraction of the proximal musculature is reported, that could lead to reduced proximal net
trunk torques. Nevertheless, the reduced net trunk torques demonstrated by individuals with LBP in our study are likely not due to increased baseline co-contraction given that we computed the torque relative to their baseline levels during quiet standing. Rather, these reductions in net peak torque following unexpected support surface perturbations may be due either to a strategy of generating increased antagonist relative to agonist torque or by reducing the agonist torque relative to the level of antagonist torque produced. Both of these strategies would result in a decrease in the net peak torque computed in response to the perturbation.

A strategy of generating relatively similar magnitudes of agonist and antagonist torques could require less specific knowledge of the nature of the perturbation, leading to reduced trunk torque regardless of perturbation direction, as was demonstrated in the current study. Alteration of the corrective torque strategy elicited by postural perturbation in this way could be mediated through changes in central set. Central set has been defined as “a central preparatory state within the nervous system related to higher-level task-related intentions and expectations” that can influence postural responses, both expected and unexpected (Cacciatore et al. 2005). Horak, Diener and Nashner (1989) reported a
“non-specific enhanced [postural] response” with unexpected perturbations of balance suggesting that healthy individuals set a higher default response magnitude in the absence of accurate information about the perturbation. Given the impairment of proprioception demonstrated by individuals with LBP (Brumagne et al. 2000; Newcomer et al. 2000; Brumagne et al. 2004; Descarreaux et al. 2005; Ginanneschi et al. 2007) it is plausible that these individuals may increase the gain of their postural responses irrespective of perturbation direction in order to compensate for a lack of accurate information and to avoid potentially injurious losses of balance. In addition, individuals with LBP may have a higher-level task-related intention to restrict lumbar movement, perhaps due to fear of movement or pain secondary to chronic LBP (Thomas et al. 2007; Thomas and France 2008), which could influence the implementation of this corrective torque strategy.

Directionally-appropriate reduced agonist relative to antagonist torque (or increased antagonist relative to agonist production) could also explain the reduced net trunk torque demonstrated by individuals with LBP in the current study. This strategy could also be mediated through changes in central set. Consistent with the pain-adaptation model, (Lund et al. 1991),
a reduction in agonist and/or an increase in antagonist trunk torque could result in restricted trunk movement, but, given the random and multi-directional perturbations used in this study, would need to do so in a directionally-appropriate manner. This pattern of responses would require very precise information about the type and direction of the balance perturbation, which, given the evidence suggesting impaired proprioception demonstrated by individuals with LBP (Brumagne et al. 2000; Brumagne et al. 2004; Della Volpe et al. 2006; Popa et al. 2007), is unlikely. A strategy of directionally-appropriate alterations in the relative agonist-antagonist trunk torque contributions could also result from the reduced need for an active, direction-specific response given the enhanced ankle contribution to postural responses during the initial period. Edwards (Edwards 2007) reported that reduction of joint stiffness below some threshold level needed to maintain balance required compensation by increased torque at other joints. It is conceivable that as a result of increased stiffness at one joint (in this case, the ankle) there could be a reduction in the stiffness required at another joint (in this case, the trunk). Individuals with LBP have demonstrated this type of torque-sharing among joints during sit-to-stand manoeuvres, where instead of significantly reducing the trunk torques in an attempt to reduce the spinal load,
individuals with LBP distributed torque among all joints (Coghlin and McFadyen 1994).

Whether the enhanced ankle torque during the initial period and the reduced trunk torque during the active period demonstrated in our study were related (either as a strategy of reduced trunk torque that necessitated a greater initial ankle response or whether the greater ankle initial contribution required a smaller active trunk response) or are just coincident findings, cannot be determined. However, given that the initial ankle torque enhancement was largely in response to backwards perturbations and no such bias was identified in the trunk torque reductions, it seems unlikely that this is the cause of the altered active proximal torque contributions.

4.5.3 Persistence of motor control alterations in chronic low back pain

Among the most striking findings of the current study is that altered movement patterns, demonstrated by spatial changes in joint torque patterns, persisted even in the absence of an active, current episode of LBP. The individuals participating in the current study had a history of LBP but were not in an active pain episode nor were they seeking treatment at
the time of testing. These individuals had low pain ratings [NPRS value of 2.0 (1.4) out of a possible 10 and 3.5 (2.8) words out of 20 circled on the McGill Pain Questionnaire] and low LBP-related disability ratings [2.4 (2.3) out of 18 on the Roland Morris Disability Questionnaire], although these were significantly greater than individuals with no history of LBP (Table 4-1). Despite low pain levels and perhaps in spite of the assumed large variability among individuals given a wide range of pain durations and severity of injuries, changes in both initial and active torque contributions of the postural responses to support surface translation were still evident.

Persistence of altered torque patterns in the absence of significant pain could indicate that altered movement patterns are the genesis of the initial LBP episode or recurrent episodes of LBP. Repetitive movement patterns have been shown to induce pain and pain behaviours in rats that did not resolve even once the cellular markers of pain had diminished (Barr 2006). Both hypo- and hyper-mobility of the lumbar spine could lead to tissue alterations causing fibrosis and inflammation, which can lead to peripheral and central sensitization characteristic of augmented pain processing (Langevin and Sherman 2007). The failure of these altered movement patterns to return to normal could indicate that the resolution of
acute pain, either due to spontaneous tissue healing processes or therapeutic intervention, did not adequately address the underlying motor impairments’ contributions to lumbar pain (Maluf et al. 2000; Sahrmann 2002; Van Dillen et al. 2003a; Van Dillen et al. 2005). In fact, it is plausible that failure to return to normal movement patterns (i.e. failure to effectively treat the underlying motor control impairments) might be the reason for the recurrent episodes demonstrated by this group of individuals with chronic recurrent LBP.

4.6 Conclusions

In conclusion, these findings demonstrate that altered strategies for maintenance of posture persist following disturbances of equilibrium in a population of individuals with a history of LBP, who were not in an active pain episode. Altered torque patterns characterized by increased initial ankle torque and reduced active trunk torque may result from additional movement constraints, either anatomical or volitional, demonstrated by individuals with LBP. Increased initial ankle torque may result from sustained or habitual postures that have lead to increased fibrotic tissue or may be an adaptive mechanism to enhance ankle stiffness. Reduced active trunk torque may result from increased equivalence of agonist-
antagonist torque possibly due to changes in central set. These results suggest that altered movement patterns persist even in the absence of an active pain episode and may be linked to chronicity of the LBP. Therapeutic interventions that address the underlying motor control impairments responsible for these altered movement patterns may contribute to the reduction of the probability, duration, and/or severity of recurrent episodes of pain.

4.7 Acknowledgments

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4.8 Additional findings: muscle activation patterns

In addition to the quantification of joint torque strategies in individuals with LBP who were in a quiescent period of their pain, muscle activation patterns of the lower limb and trunk were recorded and analyzed, but were not included in the submitted manuscript due to journal-specified space
limitations. Quantification of the underlying muscle activation patterns elicited by support surface translations provides insight into some of the muscular contributions to the joint torques. These patterns can be used to investigate the cause of the reduced trunk torques that were demonstrated by individuals with LBP, implicating either reductions in agonist or increased agonist-antagonist co-contraction elicited by unexpected perturbation.

4.8.1 Methods used to obtain surface EMG signals

EMG data of the left lower limb and the dorsal and ventral trunk, bilaterally, were recorded (BTS, Milan, Italy) using silver-silver chloride surface electrodes (Norotrode 20 bipolar, Myotronics, Kent, WA, USA) placed over the muscle bellies of 12 muscles: Tibialis Anterior (TA), Medial Gastrocnemius (GA), Rectus Abdominus (LRA and RRA corresponding to left and right RA respectively), Internal Oblique (LIO and RIO), External Oblique (LEO and REO), Erector Spinae at the 3rd lumbar segment (LES3 and RES3), and first lumbar segment (LES1 and RES1). Two ground electrodes were placed on the olecranon processes of both arms. Prior to placement of the electrodes the skin beneath the electrode site was shaved, abraded with gauze and cleaned with alcohol in order to
reduce impedance. The EMG signals were sampled at 1000 Hz in synchrony with the force plate signals, amplified (2000–10000x), full-wave rectified and band-pass filtered from 35-200 Hz.

EMG integrals were computed for each of 12 muscles and each of 12 perturbation directions using the full-wave rectified, filtered EMG signals across four epochs, baseline (250-50 ms prior to perturbation) and three 75 ms epochs spanning from 25-250ms following perturbation onset. Each epoch was divided by its duration (baseline 200 ms, other epochs 75ms) to obtain an average value for each epoch. These averages were normalized across all 12 perturbation directions and the 4 epochs for each muscle, by subject, such that the epoch with the greatest integral, regardless of timing or direction was set equal to 100%. Although raw magnitude differences could not be compared in this manner between groups, the spatial patterns of each muscle could be discerned as well as the relative contribution of the muscle for a given direction and epoch.

Descriptive statistics were computed for all measures and the responses to the perturbation directions were analyzed using a repeated measures analysis of variance for each muscle, with perturbation direction as the
repeated factor (across 12 directions) and LBP vs. NLBP as the grouping factor (SPSS Inc., Chicago, Il, USA). An alpha level of $P < 0.05$ was considered statistically significant for main effect comparisons.

### 4.8.2 Influence of LBP on muscle activation patterns

In general, individuals with LBP demonstrated increased muscle activation patterns of the proximal muscles, both ventral and dorsal, with few modifications distally. GA activation was increased for the LBP group compared with the NLBP group in directions of muscle stretch (i.e. perturbation directions with combined backward and rightward components where the muscle would act as a prime mover; Figure 4-4, $P = 0.003$). These group differences were demonstrated early in the response (25-100 ms post-perturbation) and tended to be sustained throughout the 100-175 ms epoch ($P = 0.051$).

Individuals with LBP demonstrated increased activity of the ventral trunk muscles, although some group differences were limited only to one side. RA activation was increased in the LBP group at baseline across most directions, bilaterally (Left RA, $P = 0.001$; Right RA, $P < 0.001$), a
Figure 4-4: Average normalized gastrocnemius activity across three epochs (25-100 ms, 100-175 ms, 175-250 ms) following platform perturbations. Polar plots depict group means comparing individuals with LBP (black circles) to those without LBP (NLBP; grey circles). Significant group by direction interactions (P < 0.003) are indicated by * at the directions with significant post-hoc group differences.
Average Normalized Gastrocnemius Activity Across 3 Epochs

25-100 ms Epoch

100-175 ms Epoch

175-250 ms Epoch

- NLBP
- LBP
pattern that was sustained throughout the 25-100 ms post-perturbation epoch (Left RA, P = 0.015; Right RA, P = 0.036) and the 100-175 ms epoch (Figure 4-5, RA, P = 0.016; Right RA, P = 0.094). This increased RA activity continued through the 175-250 ms epoch although only following perturbation directions that had a largely lateral component (Left RA, P = 0.027; Right RA, P = 0.029). IO activity was increased in the LBP group during the final two epochs, for the left side only (Figure 4-5, 100-175 ms epoch, P = 0.033; 175-250 ms epoch, P = 0.023), with a trend during the 25-100 ms epoch on the left side (Left IO, P = 0.093) and on the right side only following 300° perturbations (Right IO, P = 0.017). Individuals with LBP demonstrated increased activity of the EO muscles on the left side for all epochs (baseline, P = 0.0060; 25-100 ms, P = 0.055; Figure 4-5 100-175 ms, P = 0.040; 175-250 ms, P = 0.027), which despite having similar patterns was only significant on the right side during the baseline epoch (Right EO, P = 0.040).

The dorsal trunk muscles, ES at lumber segments 3 and 1 demonstrated increased activity by the LBP group compared to the NLBP group, although this was largely confined to the left side muscles. ES at L3 activity was significantly increased during the 100-175 ms epoch only, by
individuals with LBP (Figure 4-5, Left ES3, $P = 0.033$), with a similar but non-significant pattern demonstrated by the right side muscle (Right ES3, $P = 0.120$). A similar increased activation of the ES at L1 was evident on the left side of individuals with LBP during the baseline epoch (Left ES1, $P = 0.038$) and during the 100-175 ms epoch (Figure 4-5, Left ES1, $P = 0.044$), with similar but non-significant patterns on the right side (Right ES1 baseline, $P = 0.140$; 100-175 ms, $P = 0.200$).

4.8.3 Enhanced trunk muscle activation supports trunk stiffening strategy

As reported in section 4.2, we hypothesized that individuals in a quiescent period of their LBP would respond to unexpected balance perturbations using a trunk stiffening strategy (Jones et al. Submitted). A reduced active trunk torque contribution to the response was demonstrated by the LBP group and it was suggested that this could be accomplished through relatively equivalent contributions of agonist/antagonist trunk torques in both the sagittal and frontal planes. The patterns of trunk muscle activation apparent in the LBP and NLBP cohorts support increased co-activation of agonist/antagonist musculature by individuals with LBP likely contributing to reduced net trunk torques (Figure 4-5), likely due to a
Figure 4-5: Average normalized muscle activities for the trunk muscles for the 100-175 ms epoch following platform perturbations. Polar plots depict group means of the muscles of the left ventral (left panel) and dorsal (right panel) trunk comparing individuals with LBP (black circles) to those without LBP (NLBP; grey circles). Significant group effects (P < 0.050) are denoted by #.
Average Normalized Trunk Muscle Activity
100-175 ms Epoch

External Oblique#

Internal Oblique#

ES at L1#

ES at L3#

Rectus Abdominus#

Ventral | Dorsal
strategy of trunk stiffening elicited by unexpected balance perturbation.

Co-activation of the agonist/antagonist trunk musculature has been documented following unexpected load release (Radebold et al. 2000; Lariviere et al. 2010) and loading (Stokes et al. 2005) in individuals with LBP, thought to be an attempt to stiffen the trunk.

One additional finding concerns the heightened activity of the GA muscle by individuals with qLBP during the early epoch (25-100 ms) following postural perturbation (Figure 4-4). Similar elevated ankle torque during the initial period (50-100 ms post-perturbation) was demonstrated by individuals with LBP, that was most evident following perturbation directions with a largely backwards component (Jones et al. Submitted). It was suggested that this alteration could reflect either an increase in passive tissue stiffness or an increase in feedforward ankle stiffness by increased tonic activity (Gatev et al. 1999). Although enhanced baseline activity of the GA was not evident in the current study, GA activation during this early period (25-100 ms post-perturbation) was increased following perturbations with a backward component. This suggests that instead of baseline activity increases in the GA in this population perhaps these individuals demonstrate a heightened stretch reflex response in the
GA muscle, given that the perturbation directions of heightened activity correspond to those in which the muscle would be stretched.
Chapter 5: The influence of chronic recurrent LBP (during an active pain period) on corrective postural responses to multi-directional support surface translations

Based on our previous work we have established the postural strategies used to respond to multi-directional support surface translations in healthy controls and a cohort of individuals with chronic, recurrent LBP who were in a quiescent pain period (i.e. not in an active pain episode). To further delineate the complex relationship between LBP and altered motor control, the responses of individuals with LBP in an active pain episode must be quantified. Automatic postural responses (APRs) elicited by multi-directional surface translation perturbations were quantified in individuals with chronic, recurrent LBP who were in an active episode of their pain, by characterizing sagittal and frontal plane joint torque strategies as well as muscle activation profiles of the lower limb and trunk.

This manuscript will be submitted to the journal, Experimental Brain Research.
5.1 Abstract

**Objective:** Altered APRs have been demonstrated by individuals with a history of chronic, recurrent LBP while in a quiescent pain period consisting of enhanced initial ankle torque contributions and reduced active trunk torque contributions to the balance-righting response (Jones, submitted). In order to further delineate the influence of the cycles of pain exacerbation and quiescence on postural responses, this study aimed to quantify the APRs following multi-directional support surface translations, in a cohort of individuals with chronic, recurrent LBP, who were experiencing an active episode of their pain. **Methods:** Sixteen subjects with and 16 subjects without chronic, recurrent LBP stood on two force plates that were translated unexpectedly in 12 directions. Three-dimensional kinematics were combined with anthropometric equations and ground reaction forces to calculate net joint torques of the ankles, knees (sagittal only), hips and trunk, in the frontal and sagittal planes. Activation of 12 muscles of the lower limb unilaterally and the dorsal and ventral trunk, bilaterally, were recorded using surface electromyography (EMG). Peak and latencies to peak joint torques as well as integrated EMGs of windows characterizing the baseline and active muscle contributions were analysed for group by perturbation direction interaction effects. **Results:** In
general the APRs of the active LBP cohort were of similar torque magnitude but reached their peak earlier compared to individuals without LBP. Individuals with LBP demonstrated increased muscle activity in response to perturbation directions in which the muscle was acting as a prime mover and reduced muscle activity in the opposing directions in which the muscle would likely act as a stabilizer. These differences were evident at both proximal and distal muscles, although with some asymmetries proximally. **Conclusions:** The corrective responses demonstrated by individuals with LBP in an active pain episode are characterized by earlier but not altered peak torque magnitudes, and both distal and proximal direction-specific increased muscle activation. These alterations might indicate enhanced fusimotor drive leading to increased muscle spindle sensitivity or increased postural gain as a result of altered central set. Given that these motor alterations are demonstrated both proximally (near the site of pain) and distally (away from the site of pain), it is likely that these changes are mediated by descending input that may reflect the influence of central nervous system processing in this cohort.
5.2 Introduction

Despite the prevalence of low back pain experienced in our society, there is still no consensus on the etiology of the most common type of LBP, namely chronic, recurrent (Von Korff 1994) LBP. Although individuals with this type of LBP demonstrate altered movement patterns (Al-Obaidi et al. 2003; Henry et al. 2006; Lamoth et al. 2006b; MacDonald et al. 2010), it is still unknown whether these patterns developed subsequent to or preceded the first bout of pain experienced by these individuals. However, it is clear that some altered movement patterns remain between the recurrent episodes in this population (Henry et al. 2006; MacDonald et al. 2010) and it is plausible that these persistent patterns could contribute to the failure of complete resolution of symptoms of LBP and recurrent future episodes.

To further delineate the complex relationship between LBP and altered motor control, the responses of individuals who have a history of LBP but are in a quiescent period of their pain as well as individuals who are in an active period of their pain must be determined. Individuals with chronic, recurrent LBP in a quiescent period have been shown to have altered anticipatory and automatic postural coordination during predictable and
unpredictable trunk loading (MacDonald et al. 2010) and following unexpected, multi-directional translation perturbations (Henry et al. 2006; Jones et al. Submitted). These alterations involve reduced or earlier lumbar muscle activation in response to predictable and unpredictable loading (MacDonald et al. 2010) as well as reduced trunk contributions to the active frontal and sagittal plane corrective responses following unexpected translation (Jones et al. Submitted), that may influence the ability of this cohort to respond in an appropriate manner. Indeed, individuals with LBP during a quiescent period demonstrated reduced COP and increased CM responses in the sagittal plane leading to reduced stability as evidenced by a reduced margin of stability [COP-CM; (Henry et al. 2006)], as a consequence of these impaired corrective responses. These differences are notable as they likely represent alterations in motor coordination that persist even between recurrent episodes of LBP.

Furthermore, the postural responses of individuals with LBP in an active episode must also be quantified to determine whether these same altered responses are present or whether they have been modified in some fashion. This is important because understanding the influence of the cyclic patterns of quiescence and active pain on postural coordination can
lead to insights into the contributions of underlying motor control impairments to persistent pain cycles. Pain exerts a significant effect on motor coordination, altering muscle recruitment patterns that often impair an individual’s ability to respond to both planned (Hodges 2001; Hubley-Kozey and Vezina 2002) and unexpected balance perturbations by either delaying preparatory responses or eliciting inadequate or inappropriate muscle activation (co-activation) following unexpected perturbation (Radebold et al. 2000; Stokes et al. 2005). These pain effects may act via cognitive effects due either to stress (Moseley et al. 2003) or the anticipation of pain (Moseley et al. 2004b) influencing descending pathways, or by direct influence of inflammatory mediators that may result in peripheral and/or central sensitization of primary afferent fibres and dorsal horn neurons (Sluka 1996). An important step in clarifying the effects of pain on the motor coordination of postural control is to first determine whether active pain episodes exert different effects on movement patterns than those observed to persist between pain episodes. Given that APRs are not initiated by the motor cortex and are obligatory responses, the effects of pain behaviours would be minimized and the peripheral and descending influences on postural coordination can be examined. For instance, if pain exerts its effects on postural control
by interfering only with cognitive processes than little change in postural response strategies would be expected.

Therefore, the purpose of this study was to compare the APRs of individuals with and without LBP, to multi-directional support surface translations using net joint torques computed at the trunk, hips, knees (sagittal only) and ankles in the sagittal and frontal planes, as well as the myoelectric responses of muscles about the lower limb and trunk, in order to characterize the movement strategy used to govern corrective responses. A cohort of individuals with LBP was studied who reported a history of chronic LBP and were actively seeking treatment for their pain, and are therefore defined as being in an active episode (aLBP). It was hypothesized that individuals with aLBP would demonstrate altered APRs following support surface translations compared to individuals with no history of LBP. Given the redistribution of proximal vs. distal strategies identified in individuals without a history of LBP (Jones et al. 2008), the decreased trunk contributions characteristic of the corrective responses by individuals with quiescent LBP (Jones et al. Submitted) and the potential inability or reluctance of individuals with LBP to move and/or create forces at the trunk (McGill 2002), it is expected that individuals with LBP will
adopt a strategy of stiffening at the trunk that aims to reduce the net amount of trunk torque used to respond to unexpected balance perturbations.

5.3 Methods

5.3.1 Subjects

Sixteen subjects with chronic, recurrent (Von Korff 1994) LBP (aLBP; 8 female, 8 male) were recruited from local physical therapy clinics after an initial evaluation determined that they met the inclusion/exclusion criteria [mean (SD): age 33.9 (6.2) yrs, height 1.75 (0.09) m, mass 74.0 (8.9) kg]. Subjects with LBP were excluded by clinical exam if they had pain below the knee consistent with a disc herniation, presence of any neurological signs, serious spinal complications (e.g. vertebral fracture, tumour or infection), spinal stenosis, previous spinal surgery, systemic infection, balance or cardiovascular disorders, current pregnancy, history of any surgery in the 3 months prior to testing, uncorrected vision problems, or a severe musculoskeletal deformity (scoliosis or kyphosis) or injury to the lower extremity that would interfere with testing. Subjects were also excluded if they were receiving worker’s or disability compensation for their LBP, or if they were in litigation because of their LBP problem.
Subjects were tested when they were in a recurrence of their LBP and seeking treatment for their pain, and reported pain ratings of 3 (0-7) [median (range)] on the Numeric Pain Rating Scale (NPRS) the day of testing.

Sixteen subjects with no history of LBP (NLBP; 8 female, 8 male) were recruited from the local community through posted advertisements and word of mouth [mean (SD): age 33.5 (9.0) yrs, height 1.71 (0.07) m, mass 68.9 (12.3) kg]. NLBP subjects were excluded if they had a neurological disease or balance disorder, uncorrected vision problems, cardiovascular disorders, severe musculoskeletal injuries, back pain during the past week, or back pain during the prior 12 months that required medical attention or resulted in missed work.

All subjects in both groups were currently employed at the time of testing or participating fully in their usual role (e.g. full-time student, homemaker). All subjects participating in this study signed an informed consent document in accordance with University of Vermont Institutional Review Board policy and the rights of the subjects were protected.
5.3.2 Procedures

Subjects were instructed to stand on a moveable platform, looking forward, at self-selected stance width and toe-out angle (facilitated by marching in place), and with arms hanging comfortably at their sides. The foot positions were traced and marked on the platform to ensure correct repositioning of the feet following resting or stepping. All subjects were asked to maintain their balance following perturbation but were not given any instruction about how to respond. Any trials in which the subjects used a stepping strategy were discarded and repeated randomly at a later point in the protocol. Subjects were given 2-3 practice trials in each of two directions (180° translation, directly left; 90° translation, directly forward) prior to which they were told the direction of impending platform movement. Following these practice trials, linear translations of the support surface in the transverse plane were presented in 12 directions of 30° increments (Figure 5-1c) with three trials in each direction. Perturbations were presented in four blocks of trials, randomized among these 12 directions, and subjects were given several minutes of rest (3 rest periods in all) between each block of trials.
Figure 5-1: Experimental setup for support surface translations. Panel A depicts the kinematic marker setup, black dots represent reflective marker placement and the grey bars represent two force plates. Panel B depicts a displacement (cm) vs. time (ms) trace for platform movement during a rightward (0°) perturbation, as determined by the force plate kinematic markers. Panel C depicts the directions of platform perturbations with the induced body sway resulting from perturbations in the cardinal directions (i.e. left, forward, right and backward perturbations). Schematic stick figures are depicted with the subject facing to the right for the sagittal plane views and are viewed from the back for the frontal plane views.
5.3.3 Data recordings

Subjects stood with one foot on each of two force plates (Figure 5-1A; AMTI, Watertown, MA, USA) that were mounted within the moveable platform that was driven by electromechanical motors controlled by commercially available software (Compumotor, Parker Hannifin Corp., Rohnert Park, CA, USA). The platform was translated 9 cm using a ramp and hold waveform (duration of the waveform was approximately 400 ms) with a peak velocity of 43 cm/s and a peak acceleration of 127 cm/s² (Figure 5-1B). The output of the force plates was sampled at 1000 Hz and filtered with a low pass filter of 1050 Hz. The COP of each foot was calculated from the ground reaction forces (GRF) and moments recorded at the force plates.

A 3-camera, passive marker motion analysis system (BTS, Milan, Italy) was used to collect 3-dimensional spatial coordinate data for calculation of the body kinematics (Figure 5-1A). Kinematic data were sampled at 50 Hz and filtered using a dual pass 2nd order Butterworth low pass filter with a cut-off frequency of 2 Hz (a cut-off frequency of 5 Hz was used for the force plate markers assuming that these markers had the greatest accelerations given their proximity to the moving platform). An eight-
segment, rigid link model was constructed for each subject, with segment CMs determined using regression equations from the literature based on the subject’s height and mass (Zatsiorsky and Seluyanov 1983). The model represented the feet, shanks, and thighs bilaterally as well as the pelvis and trunk segments. Due to limitations in the resolution of the camera setup, markers delineating the pelvis could not be used. To compensate for this limitation, a “virtual pelvis” was computed as the line that intersected the marker over the 4th lumbar vertebra (distal trunk marker) and was perpendicular to a second line that connected the left and right proximal thigh markers. The virtual pelvis was a separate segment with mass and inertial properties assigned using the above anthropometric model and could move independently from the trunk and thigh segments. Because stepping trials were discarded, it was assumed that the feet remained stationary on the platform allowing calculation of the location of the foot CM in a fixed position.

EMG data of the left lower limb and the dorsal and ventral trunk, bilaterally, were recorded (BTS, Milan, Italy) using silver-silver chloride surface electrodes (Norotrode 20 bipolar, Myotronics, Kent, WA, USA) placed over the muscle bellies of 12 muscles: Tibialis Anterior (TA),
Medial Gastrocnemius (GA), Rectus Abdominus (LRA and RRA corresponding to left and right RA, respectively), Internal Oblique (LIO and RIO), External Oblique (LEO and REO), Erector Spinae at L3 (LES3 and RES3), and L1 (LES1 and RES1). Two ground electrodes were placed on the olecranon processes of both arms. Prior to placement of the electrodes the skin beneath the electrode site was shaved, abraded with gauze and cleaned with alcohol in order to reduce impedance. The EMG signals were sampled at 1000 Hz in synchrony with the force plate signals, amplified (2000 – 10000x), full-wave rectified and band-pass filtered from 35-200 Hz.

5.3.4 Data analysis

Marker position data were used to calculate joint angles, and first and second time derivatives of the joint angles were calculated with a 4-point weighted least-squares approach to provide angular velocity and acceleration data. Force data were low pass filtered at 10 Hz (2nd order, dual pass, Butterworth filter) and re-sampled at 50 Hz in synchrony with the kinematic data. Kinematic and force data were used to compute ankle, knee (sagittal only), hip and trunk (relative to pelvis) net joint torques in the
frontal and sagittal planes (SD/Fast, Needham, MA, USA) through inverse
dynamics techniques.

Three dependent variables for torque responses were derived. One torque
integral (defined as the area underneath a given torque vs. time curve)
was calculated to quantify the early part of the response during an initial
period (50-100 ms following perturbation onset) and this integral reflected
the passive contributions and the initial conditions prior to perturbation,
including torque due to baseline tonic muscle activity. Additionally, peak
torques (relative to baseline prior to the perturbation) and latencies to
peak torques were computed for each joint using custom software as a
means of quantifying the corrective torque responses (Matlab, Natick, MA,
USA). All torque values were normalized to height (m) and weight (N). The
absolute values of the peak torques were computed to analyze whether
the torque magnitude varied with perturbation direction, regardless of
whether the torque polarity was negative or positive. Statistical
comparisons of the peaks, latencies to peak, and passive integrals were
computed for perturbation directions of 60°, 90°, 120°, 240°, 270° and
300° for sagittal plane torques and 0°, 30°, 150°, 180°, 210° and 330° for
frontal plane torques.
EMG integrals were computed for each of 12 muscles and each of 12 perturbation directions using the full-wave rectified, filtered EMG signals across four epochs, baseline (250-50 ms prior to perturbation) and three 75 ms epochs spanning from 25-250 ms following perturbation. Each epoch was divided by its duration (baseline 200 ms, other epochs 75 ms) to obtain an average value for each epoch. These averages were normalized across all 12 perturbation directions and the 4 epochs for each muscle, by subject, such that the epoch with the greatest integral, regardless of timing or direction was set equal to 100%. Although raw magnitude differences could not be compared in this manner between groups, the spatial patterns of each muscle could be discerned as well as the relative contribution of the muscle for a given direction and epoch.

Descriptive statistics were computed for all measures and the responses to the perturbations were analyzed using a repeated measures analysis of variance for each joint torque and muscle, with perturbation direction as the repeated factor (across 6 directions for joint torques and across 12 directions for muscle activities) and LBP vs. NLBP as the grouping factor (SPSS Inc., Chicago, IL, USA). Demographic measures (age, height,
weight, BMI, stance width, foot angle) were compared between groups using independent samples T-tests. Pain measures were compared using a Wilcoxon Ranked Sum Test. An alpha level of $P < 0.05$ was considered statistically significant for main effect comparisons.

### 5.4 Results

During the initial period, individuals with aLBP demonstrated altered torques distally, in both the frontal and sagittal planes, with proximal joints influenced only in the frontal plane. Individuals with aLBP demonstrated joint torque responses that were similar in magnitude but reached their peak torque earlier than individuals without LBP at both proximal and distal joints. In general, individuals with aLBP demonstrated altered muscle activities both distally and proximally, dependent upon direction of perturbation, with enhanced activation in directions when the muscles act as prime movers and reduced activation generally in opposing perturbation directions.

#### 5.4.1 Subject demographics

The aLBP and NLBP groups did not differ on measures of age, height, weight, BMI, stance width or toe-out angle ($P$-values ranged from 0.193 to
Individuals with aLBP demonstrated significantly higher pain ratings (NPRS, P < 0.001; McGill Number of Words Score, P < 0.001, Table 5-1) than the NLBP cohort. Most individuals with aLBP reported bilateral pain (13 of 16), with two individuals reporting unilateral pain (1 reported left side pain, 1 reported right side pain) and one participant who reported no pain at the time of testing.

Table 5-1: Subject demographic information

<table>
<thead>
<tr>
<th>Parameter [Mean ( SD)]</th>
<th>aLBP (n = 16)</th>
<th>NLBP (n = 16)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>33.9 (6.2)</td>
<td>33.5 (9.0)</td>
<td>0.892</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.75 (0.09)</td>
<td>1.71 (0.07)</td>
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<tr>
<td>Weight (kg)</td>
<td>74.0 (8.9)</td>
<td>68.9 (12.3)</td>
<td>0.193</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.3 (2.6)</td>
<td>23.4 (3.2)</td>
<td>0.382</td>
</tr>
<tr>
<td>Gender (# Male/ # Female)</td>
<td>8 / 8</td>
<td>8 / 8</td>
<td>N/A</td>
</tr>
<tr>
<td>Stance Width (cm)</td>
<td>21.9 (3.0)</td>
<td>22.2 (5.3)</td>
<td>0.849</td>
</tr>
<tr>
<td>Toe-out Angle (°)</td>
<td>105.0 (11.1)</td>
<td>102.8 (5.2)</td>
<td>0.473</td>
</tr>
<tr>
<td>McGill Pain Questionnaire</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(# of Words Score)</td>
<td>5 (2-11)</td>
<td>0 (0-3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>[Median (Range)]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Numeric Pain Rating (/10)</td>
<td>3 (0-7)</td>
<td>0 (0-1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>[Median (Range)]</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oswestry Disability Index (/18)</td>
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<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Lumbar Flexion Range (°)</td>
<td>41.6 (17.2)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Duration of Symptoms (yrs)</td>
<td>8.4 (7.5)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>
5.4.2 Influence of an active episode of LBP on joint torque responses

During the initial period (50-100ms following the onset of platform movement), both groups demonstrated modulation of torque responses in a direction-dependent manner (Figure 5-2, all joints, P < 0.001). Sagittal plane initial torque responses were generally larger following forward perturbations than backward perturbations, while frontal plane torques during the initial period were generally greater in perturbation directions that induced limb loading (e.g. left Hip Abduction-Adduction torque was greater following rightward perturbations than leftward ones).

Torque responses during the initial period differed between the aLBP and NLBP groups, dependent upon perturbation direction, distally in both the sagittal and frontal planes and proximally in the frontal plane. Individuals with aLBP demonstrated greater Ankle Plantar-Dorsiflexion torques following forward and backward perturbations (Figure 5-2, left, P < 0.001; right, P=0.007). In the frontal plane, Ankle Eversion-Inversion torque responses were shifted to be less negative or slightly positive in the aLBP cohort, only for the left ankle in directions of limb unloading (i.e. 150°, 180°
Figure 5-2: Normalized joint torque integrals of the trunk, left hip and ankle in the frontal plane and the left ankle in the sagittal plane during the initial period (50-100 ms following perturbation). Group means (±SD) comparing individuals with LBP (aLBP; black circles) to those without LBP (NLBP; grey circles) for the perturbation directions that were analyzed (frontal plane, 330°, 0°, 30°, 150°, 180°, 210°; sagittal plane, 60°, 90°, 120°, 240°, 270°, 300°). Shaded directions indicate perturbation directions that would result in loading of the left leg. Significant group by direction interactions (P < 0.050) are denoted by * at the directions with significant post-hoc group differences. Non-significant post-hoc between group trends (P < 0.080) are denoted by +.
and 210°; Figure 5-2, left, P = 0.0030), although the pattern of responses was the same for the right ankle (unloading directions, 0°, 30° and 330°, P = 0.12). Some sporadic direction-specific group differences were evident for left Hip Abduction-Adduction (30° only, Figure 5-2, P = 0.039) and for Trunk Left-Right Side Bending (210° only, Figure 5-2, P = 0.043), such that individuals with aLBP demonstrated initial period torque responses that were less negative than the NLBP group.

Both individuals with and without aLBP demonstrated peak joint torque responses in the sagittal and frontal planes that varied with direction (all joints, P < 0.001, with the exception of Trunk Extension-Flexion P = 0.043), that reflected the use of both ankle and hip/trunk strategies in response to all perturbation directions. Individuals with aLBP demonstrated no differences in peak torque magnitude in either the frontal or sagittal planes (P-values ranged from 0.250 to 0.933 for group main effects) relative to the NLBP group. Only one group by direction effect was significant, namely Trunk Left-Right Side Bending torque (group X direction, P = 0.041), although post-hoc analysis detected only a trend to reduced torque by the aLBP group following 330° perturbations (post-hoc group effect, P = 0.078).
Temporal responses (latencies to peak torque) were similarly modulated with direction in both groups (0.001 < P = 0.023, with the exception of right Hip Extension-Flexion, P = 0.593). Despite the lack of magnitude differences between the two groups, temporal differences were significantly different between the aLBP and NLBP groups, demonstrated most consistently in the sagittal plane. Specifically, individuals with aLBP demonstrated earlier sagittal peak joint torques at the ankles (Figure 5-3, left, P = 0.042 right, P = 0.060), knees (Figure 5-3, left, P = 0.012; right, P = 0.003), hips (Figure 5-3, left, P = 0.033; right, P = 0.037) and for the initial trunk response (Figure 5-3, P = 0.003) but not the larger, later trunk response (Figure 5-3, P = 0.385) in the sagittal plane.

Some temporal group differences were evident in the frontal plane at the ankle (Figure 5-4, right, P = 0.020; left, P = 0.058) but not at the hips. Both the initial and later trunk side-bending responses demonstrated direction-specific group differences such that the aLBP group’s peak torques occurred earlier in left and rightward perturbations with either a forward or backward component; that is, early Trunk Left-Right Side Bending torques
Figure 5-3: Peak joint torque latencies in the sagittal plane for trunk (early and late responses), and left hip, knee and ankle. Polar plots depict group means comparing individuals with LBP (aLBP; black circles) to those without LBP (NLBP; grey circles). Significant group effects (P < 0.050) are denoted by #.
Peak Joint Torque Latencies - Sagittal Plane

- Trunk Extension-Flexion (Early)
- Trunk Extension-Flexion (Late)
- Left Hip Extension-Flexion
- Left Knee Flexion-Extension
- Left Ankle Plantar-Dorsiflexion

- alBP
- NLBP
Figure 5-4: Peak joint torque latencies in the frontal plane for trunk (early and late responses), and left and right hips and ankles. Polar plots depict group means comparing individuals with LBP (aLBP; black circles) to those without LBP (NLBP; grey circles). Significant group effects (P < 0.020) are denoted by #, while significant group by direction interactions (P < 0.040) are denoted by * at the directions with significant post-hoc group differences.
Peak Joint Torque Latencies - Frontal Plane

Early Trunk

Late Trunk

Left Hip

Right Hip

Left Ankle

Right Ankle

- aLBP
- NLBP
had significant group effects for 30° and 330° perturbations (Figure 5-4, P
= 0.032), whereas late Trunk Left-Right Side Bending torques
demonstrated significant group effects at 30°, 150°, 210° and 330°
perturbation directions (P < 0.001).

5.4.3 Influence of an active episode of LBP on muscle activation
responses
Baseline activities (representing activity during quiet standing from 250-50
ms prior to perturbation onset) did not vary with direction with the
exception of right Erector Spinae at L3 (P = 0.0090) and the right External
Oblique (P = 0.011). For both the RES3 and the right EO, the baseline
prior to leftward perturbations was slightly less than prior to rightward
perturbations, and the baseline prior to 270° (backward) perturbations was
slightly higher than the activity prior to forward perturbations. However,
these direction-specific differences represent alterations of less than 2% of
maximum activity and are likely not physiologically significant. Individuals
with aLBP demonstrated diminished baseline muscle activity levels
distally, for both the TA (P = 0.029) and GA (group X dir: P = 0.031, for
30°, 240° and 270° with non-significant trends across all other directions
except 120° and 150°), and no group differences for proximal muscle activation levels.

Individuals with and without aLBP demonstrated active muscular responses across all epochs (25-100 ms, 100-175 ms and 175-250 ms post-perturbation onset) that varied with direction, with the exception of the left IO (P = 0.281) which demonstrated no direction effect during the 25-100 ms epoch. The majority of the active muscle responses to perturbation occurred during the 100-175 ms and 175-250 ms epochs across all muscles for both groups, with only slight changes in muscle activity relative to baseline during the 25-100 ms period for all muscles.

In general, individuals with aLBP demonstrated corrective responses that were heightened in perturbation directions in which a given muscle would act as a prime mover and dampened responses in directions in which the muscle would be presumed to act as a stabilizer relative to the NLBP group. This was most evident distally as a greater relative contribution of the TA was used by the aLBP in response to forward perturbations in which the limb was loaded (30°, 60°) during the 25-100ms and 100-175ms epochs and a reduced relative contribution of the TA was evident following
backward and lateral perturbations (150°, 180°, 210°, 240°, 270°) during the 25-100 ms epoch (P < 0.001). GA activity acted in opposition to the TA with the aLBP group having an augmented relative contribution during the 100-175 ms epoch following backwards perturbations (270°, 300°, 330°) and a diminished relative contribution to forward and lateral perturbations (Figure 5-5, P < 0.001), compared to the NLBP group. Similar patterns were evident proximally for the left IOs and EOs, which demonstrated heightened relative responses by the aLBP group following perturbations with a forward/rightward component (30°, trend at 60°) during the 100-175ms epoch in which both muscles could contribute to a hip/trunk strategy and reduced relative contributions by the aLBP group to corrective responses to backwards and/or leftwards perturbations (Figure 5-5, left IO, P<0.001, left EO, P<0.001). This pattern of group differences was not evident on the right side.

The Erector Spinae muscles also demonstrated group differences dependent upon perturbation direction; however individuals with aLBP demonstrated augmented relative contributions by the ES at L3 and L1, with no diminished responses in opposing directions. Specifically, during the 25-100ms epochs the LES3 were heightened for forward, backward
and rightward perturbations for the left side (P = 0.002) and following forward perturbations only for the right side (RES3, P = 0.001). These group differences were expanded during the 100-175ms as LES3 demonstrated significant or non-significant trends to greater relative activity in the aLBP group across all directions with the exception of 240° (Figure 5-5, P = 0.042). For the LES1, group differences were evident during the 25-100 ms epoch only following 30° and 60° perturbations (P < 0.001 for both left and right muscles). This is consistent with the expectation that the ES could contribute to a reverse hip strategy following perturbations with a forward component.

5.5 Discussion

Individuals who were experiencing an active bout of their LBP demonstrated joint torque responses in both the sagittal and frontal planes that were of similar magnitude but peaked earlier relative to individuals without LBP. Group differences in initial period joint torques were most evident at the ankle with the aLBP group demonstrating increased torques in the sagittal plane and a shift towards reduced ankle inversion or increased eversion torque in the frontal plane. In general, individuals with aLBP demonstrated heightened muscle activity, both proximally and
Figure 5-5: Average normalized muscle activities for the left proximal and distal muscles for the 100-175 ms epoch. Polar plots depict group means of the left External and Internal oblique muscles, left Erector Spinae at L1 and L3, Tibialis Anterior and Gastrocnemius muscles comparing individuals with LBP (aLBP; black circles) and those without LBP (NLBP; grey circles). Significant group by direction interactions (P < 0.050) are denoted by * at the directions with significant post-hoc group differences.
Normalized Muscle Activity - 100 to 175ms Epoch

- **External Oblique**
- **ES at L1**
- **Internal Oblique**
- **ES at L3**
- **Tibialis Anterior**
- **Gastrocnemius**
distally, in directions of muscle stretch (also directions in which muscle
would act as a prime mover), although there were group side-specific
differences of the trunk muscle activity.

5.5.1 Individuals with aLBP demonstrate temporal not spatial alterations in
joint torque strategies

Individuals with aLBP demonstrated earlier peak torque responses across
proximal and distal joints, regardless of the perturbation direction, that
were most consistent across all joints in the sagittal plane (Figure 5-2).
These earlier peak torque response latencies could be explained either by
earlier onsets of the torque responses or similar onsets but with an
increased rate of torque development, thus reaching their peak along an
earlier time course. These two potential explanations have different
implications for the underlying postural control mechanisms that may be
altered when individuals with chronic pain are experiencing heightened
pain.

In general, individuals with LBP demonstrate increased muscle latencies,
particularly at the trunk, demonstrating delays in both anticipatory (Hodges
and Richardson 1996; Hodges 2001; Jacobs et al. 2009) and automatic
postural responses (Magnusson et al. 1996; Radebold et al. 2000; Cholewicki et al. 2002). However, earlier ES muscle onset latencies have been reported in individuals with LBP who were in remission from unilateral back pain in response to predictable loading, specifically on the previously painful side (Moseley et al. 2004b; MacDonald et al. 2010), and in the superficial abdominal muscles of healthy individuals who were anticipating induced back pain (Moseley et al. 2004b). Earlier muscle onset latencies in response to postural perturbation could be due to enhanced muscle spindle sensitivity as a result of increased fusimotor drive. While changes of muscle spindle activity have not been demonstrated with experimentally induced muscle or skin pain (Birznieks et al. 2008), increased fusimotor drive has been demonstrated in humans under conditions of increased arousal (Ribot-Ciscar et al. 2000). Given that earlier onset latencies have been demonstrated prior to predictable loading (MacDonald et al. 2010) and in anticipation of pain (Moseley et al. 2004b), it is possible that the early peak torque latencies we have demonstrated could be related to increased arousal in our aLBP cohort, perhaps due to pain-related fear or fear of movement. Pain-related fear has been identified as a more potent stimulus for movement restrictions than pain intensity (Thomas and France 2008) and has been associated
with trunk movement restrictions by individuals who had recently experienced an episode of LBP (Thomas and France 2007). Interestingly, this same strategy of trunk restriction was demonstrated by healthy individuals in anticipation of back pain (Moseley et al. 2004b), further implicating the state of arousal in reducing response latencies in the anticipation of pain.

In our current data set the onset of corrective torque responses were not quantified because the kinematic signals were filtered significantly to minimize the influence of noise on the torque calculation. Filtering in this way renders onset determination difficult and likely inaccurate, thus it was not performed. Onsets of muscle activation were not determined in the current study but epochs characterizing the EMG responses could indicate an earlier onset of muscle activation if the average activity across an early epoch was greater between the two groups. There were few group differences during the 25-100 ms epoch, occurring only at the TA, GA and Left ES3. Distally, these early responses in the aLBP were less than those of the NLBP group, except in directions that would stretch the muscle (e.g. a 30° or 60° perturbation would stretch the Left TA). At the Left ES3 individuals with aLBP demonstrated increased activity across all
directions, although these were only significant following perturbation
directions with a largely rightward or backward component. Although
greater muscle activation in perturbation directions that create muscle
stretch would point to increased muscle spindle sensitivity, we cannot
definitely report that shorter muscle onset latencies were the cause of
the early peak torque responses in the current study. Despite this
limitation, the observed enhanced ankle torque demonstrated by the aLBP
group during the initial period (50-100 ms post-perturbation) could also
reflect an early heightened response as a result of enhanced muscle
spindle sensitivity.

The shorter peak torque latencies demonstrated in the current study could
have also resulted from an increased rate of torque development (i.e.
slope of the torque vs. time curve), possibly reflecting an increased gain of
the postural response in the aLBP cohort. Horak, Diener and Nashner
(1989) reported a default rate of ankle torque response in healthy
individuals when the characteristics of the support surface translations
were unknown. This rate of torque development could be scaled either up
or down dependent upon prior knowledge of the perturbation, suggesting
that postural response gain can be manipulated through changes in
central set (Horak et al. 1989). Central set has been defined as “a central preparatory state within the nervous system related to higher-level task-related intentions and expectations” that can influence postural responses, both expected and unexpected (Cacciatore et al. 2005). It is possible that changes in central set either secondary to pain-related fear or fear of movement could influence the corrective torque responses to unexpected perturbations.

Individuals with high pain-related fear have demonstrated reduced performance on lifting tasks (Vlaeyen et al. 1995; Swinkels-Meewisse et al. 2006) and a leg strength test (Verbunt et al. 2005), restricted spinal motion (Thomas and France 2007), and reduced preferred and fast walking velocities compared to individuals with low fear (Al-Obaidi et al. 2003). In addition, individuals with LBP that had high pain-related fear also demonstrated an increased influence of anticipated pain on their movement patterns (Pfingsten et al. 2001; Al-Obaidi et al. 2003). These altered movement patterns may reflect changes in central set acting to restrict either the magnitude or the speed of the movement. Indeed, healthy individuals who experienced a postural threat (quiet standing at the edge of a high surface) reported greater conscious control of posture
that was related to an altered postural strategy (Huffman et al. 2009). Individuals with chronic LBP demonstrated decreased variability of trunk movements during walking while performing an attention-demanding task (Lamoth et al. 2008) and altered cerebrocortical activity during an arm raise task (Jacobs et al. 2010) that suggest a greater involvement of the higher brain centers on postural control in both volitional and anticipatory postural coordination. It is not known whether a similar reliance on the higher brain centers is present in individuals with LBP for automatic postural coordination. However, postural anxiety has been shown to increase the amplitude of corrective responses to unexpected surface rotation in healthy individuals, suggesting that fear-related constructs can impact automatic postural coordination (Carpenter et al. 2004b).

The shorter peak torque latencies demonstrated by the aLBP group in the current study were pervasive among sagittal plane joint torques regardless of perturbation direction, but less so in the frontal plane. In fact, frontal plane peak torque latencies demonstrated group differences only at the trunk in directions that included a diagonal component (e.g. rightward and backward or rightward and forward perturbations), and at the right ankle (trend in the left ankle). This is consistent with previous studies that have
demonstrated more robust sagittal but not frontal plane group differences in postural responses (Radebold et al. 2000; Henry et al. 2006). The explanations for these plane-specific differences could be threefold. First, sagittal plane responses require a greater active contribution than do frontal plane responses given the wide base of support (Rietdyk et al. 1999; Henry et al. 2001) and the coupling between ankle and hip motions in the frontal plane (Day et al. 1993). Therefore, if the cause of the decreased torque latencies demonstrated by individuals with aLBP is primarily neuromuscular in origin then it would be expected that group differences would likely be more pronounced in the sagittal plane. In addition, loss of balance may be more threatening in the frontal plane since loss of stability would require a stepping strategy across the body to prevent falling (Winter et al. 1996; Zettel et al. 2002). Thus, there may be stricter control of balance in the frontal plane and therefore less variability permitted in this plane. Finally, the perturbation velocity used in the current study was the same regardless of direction, thus perhaps the relative difficulty of the perturbation in the frontal plane was not equal to sagittal plane perturbations. Due to the potential for an increased passive contribution in the frontal plane as well as the larger base of support in this
plane, a given perturbation magnitude may be easier to respond to and therefore may be insufficient to elicit underlying group-specific differences.

5.5.2 Side-specific muscular responses to unexpected perturbations

Group comparisons between the aLBP and NLBP cohorts demonstrated side-specific differences in trunk muscle activation patterns, such that group differences were statistically significant on the left side with few differences detected on the right (Figure 5-6). These were not simply statistical similarities between groups but often asymmetric patterns of activation between the left and right muscles for both groups. Individuals with aLBP demonstrated reduced activity of the right RA, although the left and right patterns were similar. The left IO muscle followed the pattern of the distal muscles, with heightened activity by the aLBP cohort in directions of muscle stretch, with a more or less direction-independent response from the right IO in both groups. The EO and ES at L3 muscles demonstrated activities that were heightened in the directions in which the muscle could contribute to an early hip strategy (i.e. left EO had a greater relative response to rightward perturbations) but the aLBP demonstrated larger responses than the NLBP on the left side only, with no differences on the right side.
Figure 5-6: Average normalized muscle activities for the left (left panel) and right (right panel) trunk muscles for the 100-175 ms epoch, depicting side-specific group differences. Polar plots depict group means comparing individuals with LBP (aLBP; black circles) to those without LBP (NLBP; grey circles). Significant group by direction interactions (P < 0.050) are denoted by * at the directions with significant post-hoc group differences.
Side-Specific Group Differences in Muscle Activation
100 to 175 ms Epoch

Left

Erector Spinae at L3

Right

External Oblique

Internal Oblique

Rectus Abdominus
Asymmetries in trunk muscle activation, either by magnitude or frequency of firing, have been reported previously, elicited by unexpected forward support surface translations (Newcomer et al. 2002), during volitional trunk bending movements (Larivière C. 2000) and during isometric trunk extension (Alexiev 1994). Lariviere et al. (2000) report that these asymmetries were not related to anatomical or kinematic differences in trunk movement patterns between groups. None of these studies reported the pain location or the chronicity of pain in these individuals. Lariviere et al. (2000) suggested that asymmetries could result from hand-dominance or muscle wasting in individuals that may be related to pain. It is not surprising that activation asymmetries exist, particularly given the heterogeneous nature of the chronic LBP population who may have pain locations and duration, severity and frequency of pain episodes that may vary widely. In addition, the etiology of LBP is often unknown, as little as 20% of individuals with LBP can be given a structurally-based diagnosis (Nordin et al. 1998). In the current aLBP cohort, all individuals with the exception of two reported bilateral or central pain locations, did not have specific diagnoses and had experienced pain for 8.4 years on average (range: 1.5-25 yrs).
Group differences in muscle activation patterns could result from asymmetries that are present within the aLBP cohort due to the effects of pain on neuromuscular control, including reduced strength (Verbunt et al. 2005), peripheral feedback alterations (Brumagne et al. 2000; Van Dieen et al. 2003), tissue modifications (Gombatto et al. 2008) or may be the result of habitual movement patterns that have reinforced asymmetric muscle activation patterns. Although muscle activation patterns were not reported, Van Dillen et al. (Van Dillen et al. 2006) reported significantly more asymmetric movement impairments in individuals with LBP who routinely participated in asymmetric leisure activities (e.g. tennis, golf). Individuals with LBP who participated in rotation-related sports demonstrated more asymmetries of passive hip range of motion than individuals who participated in the same sports but did not have LBP (Van Dillen et al. 2008). Gombatto (2008) reported asymmetries in passive tissue resistance in both individuals with and without a history of LBP, although the LBP cohort demonstrated greater asymmetries, despite no group differences in passive tissue extensibility. Thus, both asymmetric movement patterns and passive tissue characteristics been linked to repetitive movement patterns.
Asymmetries in both individuals with and without LBP seem logical given that individuals from each group will potentially have quite varied movement patterns that they use in their daily lives. Thus, while we have reported on the general group differences that are present, further investigation of smaller, more homogenous groups with similar movement patterns might unmask specific pain-related deficits. Whether these muscle activation differences reported in the current study are related to underlying differences in repetitive movement patterns is unknown. Future attempts at classifying individuals with chronic LBP to provide more homogeneous subgroups (Van Dillen et al. 2003b) may be beneficial in understanding the whether the asymmetries in muscle activation patterns elicited by unexpected postural perturbations reflect altered, habitual movement patterns that are related to pain.

5.5.3 Do enhanced APRs in the aLBP group represent a short-term adaptation to a heightened pain state?

The decreased peak torque latencies and increased muscle activation patterns demonstrated by the aLBP cohort both proximally and distally may be related to the recent pain exacerbations experienced by individuals in this cohort. These heightened responses may represent an
adaptation to increased pain or may be secondary to pain-related fear, brought on by their recent active pain episode. Although the individuals in the current study had had a recent pain exacerbation they reported relatively low pain intensity, on average, [although this was greater than a cohort who was in a quiescent period; (Jones et al. 2008)], and measures of pain-related fear were not quantified.

The postural response strategies elicited by unexpected support surface translations of the aLBP cohort differed from those of individuals with chronic LBP who were in a quiescent period of their pain. Individuals in a quiescent period demonstrated altered active contributions to their corrective responses following unexpected support surface translation characterized by reduced trunk torques, mediated through increased co-activation of the dorsal and ventral musculature about the trunk (Jones et al. Submitted). In the current cohort who had recently experienced a pain exacerbation, there was a more global set of alterations characterized by no changes in peak torque magnitude but shorter latencies to peak torque. These alterations are noteworthy because they are present at both proximal joints and also distal joints remote from the site of pain, and may represent a reduction in the variability of the postural responses.
Reduced variability of postural strategy following a self-induced arm raise perturbation has been demonstrated in individuals experiencing induced back pain, that did not resolve once the pain had subsided in a subset of participants (Moseley and Hodges 2006). This subset of “non-resolvers” had significantly higher additive scores on the Back Beliefs Questionnaire, the Survey of Pain Attitudes and Pain Catastrophizing Scale questionnaires in addition to the loss of variability in muscle onset times. In the anticipation of back pain in healthy control subjects there was a shift to a strategy of earlier abdominal muscle onsets likely contributing to a stiffening strategy (Moseley et al. 2004a), potentially similar to our current findings. Thus it appears that even after pain resolution or in the anticipation of pain, altered movement strategies are evident. However, in the case of APRs elicited by support surface translation, it appears that the movement strategies demonstrated in close temporal proximity to a pain exacerbation are not the same altered strategies that persist over the longer term between pain exacerbations (Jones et al. Submitted).

It is plausible that APRs in individuals with chronic LBP are characterized by a pervasive heightened response temporally close to a pain episode,
which may give way to proximal restrictions that persist between pain episodes. However, the time course of this speculative shift is unknown. Thomas and France (2007) demonstrated that group differences in trunk restrictions during volitional reaching tasks between individuals with high and low pain-related fear that were present at 3 and 6 weeks post-pain episode, were diminished 12 weeks after their acute pain episode. Further study of APRs closely linked to a pain episode with intermittent follow-up, might provide a more specific time course that could be used to target appropriate interventions to mitigate symptoms and recurrence over the long-term.

5.6 Conclusions

Individuals with LBP who were in an active period of their pain demonstrated altered APRs to unexpected support surface translations that were characterized by shorter peak torque latencies evident at proximal and distal joints, and muscle activation levels that were increased in directions that would result in sway-induced muscle stretch. Shorter peak torque latencies could be the result of enhanced muscle spindle sensitivity leading to earlier torque onsets or an increased rate of torque development reflecting increased postural gain mediated by altered
central set. These altered automatic postural responses by individuals with LBP may represent a global, short-term adaptation to a recent heightened pain episode that diminishes overtime given that individuals with a history of LBP without a recent pain episode demonstrate alterations only at the trunk. Further characterization of the time course of this pain response might identify therapeutic interventions that could help to alleviate short-term pain adaptations that could potentially lead to recurrent pain episodes.

5.7 Acknowledgments

The authors wish to thank Kerry A. McCarthy, Tori Deschanneaux and Natalie Moore for their assistance in data reduction. This work was funded by the University of Vermont College of Nursing and Health Sciences Dean’s Research Incentive Award, and NIH/NCMRR/R01 HD04099.
Chapter 6: The influence of an active episode of chronic, recurrent LBP on sit-to-stand movements

One of the challenges in studying populations of individuals with chronic pain is the confounding influence of pain on behaviour. In order to provide some insight into the potentially confounding influence of pain and its impact on the higher brain centres on postural control, it is important to characterize a volitional task, one initiated by the motor cortex, to determine what, if any, volitional influences may be impacting movement patterns. In this study, individuals with and without chronic, recurrent LBP, during an active pain episode, were asked to perform self-paced sit-to-stand (STS) movements. Kinetic, kinematic and muscle activation patterns were quantified to determine the influence of pain behaviour on movement patterns during this volitional task.

This manuscript will be submitted to the journal, Clinical Biomechanics.

6.1 Abstract

Objective: Altered postural responses have been demonstrated by individuals with chronic, recurrent LBP reflecting changes in both automatic and voluntary postural control during some dynamic movements. In order to understand the
influence of pain behaviour on voluntary movement patterns, this study aimed to quantify STS movements in a cohort of individuals with chronic, recurrent LBP, who were experiencing an active episode of their pain. **Methods:** Thirteen subjects with and 13 subjects without chronic, recurrent LBP sat on a height-adjusted instrumented chair with their buttocks on one force plate and both feet on another. Three-dimensional kinematics and kinetics were used to calculate the centres of pressure and mass (COP and CM respectively), joint angular movements and net joint torques of the ankles, knees, hips and trunk in the sagittal plane. Activation of 16 muscles on the lower limb unilaterally and the dorsal and ventral trunk, bilaterally, were recorded using surface electromyography. Three analysis periods were computed based on the horizontal and vertical CM momenta representing the acceleration, transition and deceleration phases. Average net joint torques, ground reaction forces and muscle activities were computed across these three phases and compared using repeated measures analyses of variance. Peak COP and CM in the sagittal and frontal planes as well as joint angles in the sagittal plane were computed and compared using independent samples T-tests. **Results:** Individuals with LBP demonstrated similarly paced STS movements compared with the NLBP cohort, however, with an earlier seatoff and a shorter acceleration phase. In general, individuals with aLBP tended to restrict motion in their proximal joints while
increasing the movement of their distal joints. Individuals with aLBP also tended to use a greater relative contribution of their muscles both proximally and distally to achieve the STS movement. Consistent with these findings, during the transition phase, individuals with aLBP demonstrated a greater reliance on knee joint torque relative to hip torque than did the NLBP cohort. **Conclusions:** Individuals with aLBP performed the STS movement at the same speed but using a strategy that involved a redistribution of proximal to distal joint contributions. These alterations did not appear to impact the stability of the movement. It appears that individuals with aLBP may have a higher level intention to restrict trunk excursion through the STS movement which necessitates compensation by more distal joints.

**6.2 Introduction**

Individuals with chronic LBP have demonstrated movement patterns that differ from those of healthy controls and suggest alterations in automatic (Henry et al. 2006), anticipatory (Hodges 2001; Jacobs et al. 2010) and volitional (Coghlin and McFadyen 1994; Al-Obaidi et al. 2003; Shum et al. 2005a; Lamoth et al. 2006b) postural control. While it is important to quantify the more automatic postural tasks, it is also vital to understand how individuals with LBP may choose to move when performing a volitional task, one that might provide a window into the
higher level task-related intentions that may drive a given movement pattern. In general, individuals with LBP tend to move more slowly when performing a task, such as walking at a self-selected pace (Al-Obaidi et al. 2003; Lamoth et al. 2006b), despite having the ability to attain higher speeds such as those performed by healthy individuals (Lamoth et al. 2006b). Given that individuals have chosen to move in a slower manner, by examining movement patterns it may be possible to determine the underlying reason for this tendency if individuals are required to perform a task that challenges the motor control impairments that may be present in this group.

The STS task is a volitional activity of daily living that may require significant involvement of the trunk and pelvis in order to accomplish the movement. Although it may be accomplished primarily using the knee with little trunk contribution (Schenkman et al. 1990; Roebroeck et al. 1994), this strategy may compromise the stability of the movement by resulting in a more posterior location of the body’s CM as the base of support (BOS) is transferred to the feet only. A reduction in the stability of motion may be undesirable for individuals with LBP if their proprioceptive system is compromised, potentially making them less able to accurately plan their movements and to detect instability (Brumagne et al. 2000; Newcomer et al. 2000; Brumagne et al. 2004; Descarreaux et al. 2005;
Ginanneschi et al. 2007). Indeed, Coghlin and McFadyen (1994) reported that individuals with LBP tended to move more slowly during the ascension phase of the STS movement, potentially as a means to prevent loss or near loss of stability as the body CM was moved to its final upright position.

An alternate STS strategy (the hip/trunk strategy) involves primarily trunk and hip flexion (Doorenbosch et al. 1994), resulting in a more stable movement pattern that serves to move the body CM close to the BOS of the feet prior to seatoff. This pattern may be undesirable for a population of individuals who are experiencing pain or restricted range of motion in the lower back and may wish to limit movement and/or forces applied to the lumbar spine (McGill 2002), or may be unable to produce sufficient lumbar muscle forces (McGill 2002) secondary to disuse (Mayer et al. 1985; Lee et al. 1995) or habitual patterns of movement. Shum et al. (2005) reported that individuals with chronic LBP did restrict their lumbar spine and hip movements during the STS movement, and minimized the torque applied about their lumbar spine (Shum et al. 2007) suggesting that this cohort may favour restricting trunk and hip motion and or forces, perhaps at the expense of movement stability.
The STS movement, therefore, represents a volitional movement that may present a conflict for the individual with LBP; that is, choosing a movement strategy that requires either significant trunk/pelvis excursion or one that may result in compromised postural stability. A successful STS requires control of both horizontal and vertical momenta as well as significant movement of the trunk and pelvis segments, which may prove challenging to individuals with LBP. Previous studies have not adequately reported joint torques in combination with muscle activation patterns of proximal and distal joints, nor whole body CM parameters in the STS movement in a cohort of individuals with chronic LBP (Coghlin and McFadyen 1994; Shum et al. 2007).

Therefore, the purpose of this study was to characterize the movement patterns used by individuals with a history of chronic LBP, who were actively seeking treatment for their pain (aLBP), during STS movements at preferred speed, and to compare these to those performed by individuals without a history of LBP. This study aims to quantify the influence of aLBP on a volitional movement and will provide insight into movements initiated by the motor cortex to understand the higher level task-related intentions of individuals with this impairment.
6.3 Methods

6.3.1 Subjects

Thirteen subjects with chronic, recurrent (Von Korff 1994) LBP (aLBP; 6 female, 7 male) were recruited from local physical therapy clinics after an initial evaluation determined that they met the inclusion/exclusion criteria [Table 6-1, mean (SD): age 34.6 (6.4) yrs, height 1.75 (0.09) m, mass 74.0 (9.1) kg]. Subjects with LBP were excluded (by clinical exam) if they had pain below the knee consistent with a disc herniation, presence of any neurological signs, serious spinal complications (e.g. vertebral fracture, tumour or infection), spinal stenosis, previous spinal surgery, systemic infection, balance or cardiovascular disorders, current pregnancy, history of any surgery in the 3 months prior to testing, uncorrected vision problems, or a severe musculoskeletal deformity (scoliosis or kyphosis) or injury to the lower extremity that would interfere with testing. Subjects were also excluded if they were receiving worker’s or disability compensation for their LBP, or if they were in litigation because of their LBP problem. Subjects were tested when they were in a recurrence of their LBP and seeking treatment for their pain and reported pain ratings of 3 (0-7) [median (range)] on the Numeric Pain Rating Scale (NPRS) the day of testing.
Thirteen subjects with no history of LBP (NLBP; 6 female, 7 male) were recruited from the local community through posted advertisements and word of mouth.

[Table 6-1, mean (SD): age 33.0 (5.9) yrs, height 1.71 (0.07) m, mass 69.2 (13.2) kg]. NLBP subjects were excluded if they had a neurological disease or balance

Table 6-1: Description of discrete time points for analysis of STS movements

<table>
<thead>
<tr>
<th>Timepoint</th>
<th>Description</th>
<th>Parameter for Calculation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Movement Initiation</td>
<td>Initiation of STS movement</td>
<td>Net force in the anterior/posterior</td>
</tr>
<tr>
<td>Time of Seatoff</td>
<td>Time at which all mass has been transferred to the force plate under the feet</td>
<td>Vertical force of the seat force plate</td>
</tr>
<tr>
<td>End of Movement</td>
<td>Time at which final standing posture is attained</td>
<td>Vertical CM velocity</td>
</tr>
<tr>
<td>Baseline</td>
<td>Time period used for determination of baseline parameter level</td>
<td>500-250 ms prior to movement initiation</td>
</tr>
<tr>
<td>Acceleration</td>
<td>Time period for generation of horizontal momentum</td>
<td>From movement initiation to the time of maximum horizontal CM velocity</td>
</tr>
<tr>
<td>Transition</td>
<td>Time period for transfer of horizontal to vertical momentum</td>
<td>From time of maximum horizontal CM velocity to the time of maximum vertical CM velocity</td>
</tr>
<tr>
<td>Deceleration</td>
<td>Time period for slowing of momentum to attain final standing posture</td>
<td>From time of maximum vertical CM velocity to the end of movement</td>
</tr>
</tbody>
</table>
disorder, uncorrected vision problems, cardiovascular disorders, severe musculoskeletal injuries, back pain during the past week, or back pain during the prior 12 months that required medical attention or resulted in missed work. All subjects in both groups were currently employed at the time of testing or participating fully in their usual role (e.g. full-time student, homemaker). All subjects participating in this study signed an informed consent document in accordance with University of Vermont Institutional Review Board policy and the rights of the subjects were protected.

6.3.2 Procedures

Subjects sat comfortably on an instrumented chair (force plate under the buttocks) with no back or arm rests, adjusted to 100% of knee height while both feet were placed on a second force plate at a self-selected width (Figure 6-1). Subjects were not given any specific instructions about how to sit but a 12 cm high flexible support was attached to the force plate surface and placed behind the individual’s sacrum such that it provided minimal tactile force but served to aid the participant in returning to their original seated position following a STS trial. Subjects performed 7 STS movements with arms crossed at a self-selected natural pace, in addition to two quiet sitting and quiet standing calibration trials (duration 5 s each). Only the last 5 STS movements were recorded in order to
Figure 6-1: Sit-to-Stand experimental setup depicting the kinematic marker setup. Black dots represent reflective marker placements and the grey bars represent two force plates, one embedded in the surface of a chair adjusted to 100% of knee height (chair not shown) and the second under the feet. Schematic figure does not depict the individual’s arms, which were crossed across the chest.
STS Experimental Setup

Seat Forceplate

100% Knee Height

Feet Forceplate
provide two practice trials to acclimate the subjects to the testing apparatus, although the subjects were not aware that the trials were not being recorded.

6.3.3 Data recordings

A 3-camera passive marker system (BTS, Milan, Italy) was used to collect 3-dimensional spatial coordinate data for calculation of the body kinematics. Kinematic data were sampled at 50 Hz and low-pass filtered (dual pass, 2nd order Butterworth filter) with a cut-off frequency of 5 Hz. Retro-reflective markers were placed on the acromion process, the superior aspect of the iliac crest lying along the mid-axillary line, the greater trochanter, the lateral femoral condyle, the lateral malleolus and the 5th metatarsal head, all on the left side of the body to delineate the trunk, pelvis, thigh, leg and foot segments in the sagittal plane (Figure 6-1). Two markers each were placed on the top surface of the force plate under the feet and the lateral surface (at most superior position) of the force plate under the buttocks to delineate the locations of the force plates in space (Figure 6-1). First and second derivatives of marker coordinates were computed using a central difference technique to calculate both linear and angular velocity and acceleration data.
Kinetic data were measured using the force plates (AMTI, Watertown, MA, USA), one embedded within the surface of the moveable platform (which was held in constant position) and the second securely attached to the chair (Figure 6-1). Force plate signals were amplified (4000x), low-pass filtered at 1050 Hz and sampled at 1000 Hz. Kinetic data were subsequently filtered (2\textsuperscript{nd} order, dual pass, Butterworth filter) at 10 Hz and re-sampled at 50 Hz in synchrony with the kinematic data.

EMG data of the left lower limb and the dorsal and ventral trunk, bilaterally, were recorded (BTS, Milan, Italy) using silver-silver chloride surface electrodes (Norotrode 20 bipolar, Myotronics, Kent, WA, USA) placed over the muscle bellies of the 16 muscles: Tibialis Anterior (TA), Medial Gastrocnemius (GA), Rectus Femoris (RF), Medial Hamstrings (HA), Rectus Abdominus (LRA and RRA corresponding to left and right RA respectively), Internal Oblique (LIO and RIO), External Oblique (LEO and REO), Erector Spinae at the third lumbar segment (LES3 and RES3), the first lumbar segment (LES1 and RES1) and the eighth thoracic segment (LES8 and RES8). Two ground electrodes were placed on the olecranon processes of both arms. Prior to placement of the electrodes the skin beneath the electrode site was shaved, abraded with gauze and cleaned with alcohol in order to reduce impedance. The EMG signals were sampled at
1000 Hz in synchrony with the force plate signals, amplified (2000-10000x), full-wave rectified and band-pass filtered from 35-200 Hz.

6.3.4 Data analysis

A four-segment, anthropometric model [Figure 6-1; foot, leg, thigh and head/arms/trunk; (Winter 1990)] was used to determine segment mass and inertial parameters, with which to calculate the location of the whole body CM. A five-segment model [foot, leg, thigh, pelvis, HAT; (Zatsiorsky and Seluyanov 1983)] was used in conjunction with the force plate and kinematic data to determine the net joint torques at the trunk, hip, knee and ankle joints, using inverse dynamics.

Kinematic, kinetic and EMG data were analyzed by computing both spatial and temporal variables. The motion of the STS movement was characterized by computing the total movement duration, the relative proportions of the acceleration, transition and deceleration phases (Roebroeck et al. 1994), as well as the time of seatoff. These discrete time points were computed as indicated in Table 6-1. Peak magnitudes and latencies relative to the initiation of movement were computed for the COP, CM, CM velocities (horizontal and vertical components), segment and joint angles, forces, net joint torques and EMG
traces. In addition, COP and CM excursion measures (total excursion during the STS movement) were computed. The peak forward excursion of the shoulder marker relative to the ankle position was computed to represent the maximum forward displacement of the trunk relative to the base of support.

Integrals of the net joint torques (ankle, knee, hip, trunk), the GRFs (anterior-posterior, lateral and vertical) and all muscle activation profiles were computed across the acceleration, transition and deceleration phases and then divided by the individual trial durations of each of these phases to compute the average torque, force or muscle activity across the phase. In addition, a baseline integral computed from 500 to 250 ms prior to the initiation of movement was computed for all muscles, divided by the duration of this epoch to obtain the average baseline activity for each individual muscle. All muscle activation profiles were normalized to the maximal average activity over a 2000 ms window obtained during maximal, voluntary standardized contractions (MVC; Table 6-2). In addition, the relative joint torque contributions of the ankle, knee, hip and trunk during the acceleration, transition and deceleration phases were computed by summing the absolute values of the torque integrals across the individual movement phases to compute the total torque generated across a given phase.
and then the relative contributions of each joint to the given phase were computed as a percentage of this total torque.

Table 6-2: Muscles recorded by surface EMG

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Maximal Voluntary Contraction Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Erector Spinae at T8</td>
<td>Back extension with pelvis supported on a bolster</td>
</tr>
<tr>
<td>Right Erector Spinae at T8</td>
<td></td>
</tr>
<tr>
<td>Left Erector Spinae at L1</td>
<td></td>
</tr>
<tr>
<td>Right Erector Spinae at L1</td>
<td></td>
</tr>
<tr>
<td>Left Erector Spinae at L3</td>
<td></td>
</tr>
<tr>
<td>Right Erector Spinae at L3</td>
<td></td>
</tr>
<tr>
<td>Left External Oblique</td>
<td>Maximum elicited in 1 of 4 activities:</td>
</tr>
<tr>
<td>Right External Oblique</td>
<td>Resisted, seated left trunk twist</td>
</tr>
<tr>
<td>Left Internal Oblique</td>
<td>Resisted, seated right trunk twist</td>
</tr>
<tr>
<td>Right Internal Oblique</td>
<td>Trunk curl</td>
</tr>
<tr>
<td>Left Rectus Abdominus</td>
<td>Prone bilateral leg lift (knee at 110°, raised 5 cm)</td>
</tr>
<tr>
<td>Right Rectus Abdominus</td>
<td></td>
</tr>
<tr>
<td>Left Rectus Femoris</td>
<td>Resisted, seated knee extension</td>
</tr>
<tr>
<td>Left Medial Hamstrings</td>
<td>Resisted, seated knee flexion</td>
</tr>
<tr>
<td>Left Medial Gastrocnemius</td>
<td>Resisted, seated plantarfexion</td>
</tr>
<tr>
<td>Left Tibialis Anterior</td>
<td>Resisted, seated dorsiflexion</td>
</tr>
</tbody>
</table>

All temporal and spatial variables were compared using independent samples T-tests to detect group differences. GRF, joint torque and muscle activity integrals across the three movement phases were compared using a repeated measures analysis of variance (ANOVA), with phase as the repeated factor and aLBP vs.
NLBP as the grouping factor. For these comparisons the force and torque integrals were computed relative to baseline and thus the phase effect had three levels (acceleration, transition and deceleration). The muscle activity variables were compared using EMG averages normalized to average MVC values, thus average EMG during the baseline will be included as the fourth level of the phase effect. The relative contributions of the joint torques to the movement phases were computed for each individual phase (i.e. acceleration, transition, and deceleration) using a repeated measures ANOVA with joint as the repeated factor and aLBP vs. NLBP as the grouping factor. An alpha level of $P < 0.05$ was considered statistically significant for main effect comparisons.

6.4 Results

Individuals with aLBP demonstrated some alterations to their STS movement patterns that may reflect impairments at or near the site of their low back pain. These alterations were characterized by slight timing changes in the acceleration phase and time of seatoff, restriction of trunk movement, augmented distal joint contributions and increased co-activation of the proximal muscles during the STS movement.

6.4.1 Subject demographics
The aLBP and NLBP groups did not differ on measures of age, height, weight, BMI, stance width or toe-out angle (P-values ranged from 0.180 to 0.900, Table 6-3). Individuals with aLBP demonstrated significantly higher pain ratings (NPRS, P < 0.001; McGill Number of Words Score, P < 0.001, Table 6-3) than the NLBP cohort. Most individuals with aLBP reported bilateral pain at the time of testing (11 of 13), with one individual reporting unilateral pain (left side), and one participant reporting no pain.

Table 6-3: Subject demographic information

<table>
<thead>
<tr>
<th>Parameter [Mean (SD)]</th>
<th>aLBP (n = 13)</th>
<th>NLBP (n = 13)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>34.6 (6.4)</td>
<td>33.0 (5.9)</td>
<td>0.51</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.75 (0.09)</td>
<td>1.71 (0.07)</td>
<td>0.21</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>74.0 (9.1)</td>
<td>69.2 (13.3)</td>
<td>0.29</td>
</tr>
<tr>
<td>BMI (kg/m(^2))</td>
<td>24.0 (2.3)</td>
<td>23.3 (2.8)</td>
<td>0.49</td>
</tr>
<tr>
<td>Gender (# Male/ # Female)</td>
<td>7 / 6</td>
<td>7 / 6</td>
<td>N/A</td>
</tr>
<tr>
<td>Stance Width (cm)</td>
<td>20.2 (3.9)</td>
<td>22.2 (3.3)</td>
<td>0.18</td>
</tr>
<tr>
<td>Toe-out Angle (°)</td>
<td>96.8 (6.0)</td>
<td>96.5 (4.3)</td>
<td>0.90</td>
</tr>
<tr>
<td>McGill Pain Questionnaire (# of Words Score) [Median (range)]</td>
<td>5 (2-11)</td>
<td>0 (0-3)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Numeric Pain Rating Scale (/10) [Median (range)]</td>
<td>3 (0-7)</td>
<td>0 (0-1)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Oswestry Disability Index (/100)</td>
<td>19.4 (9.1)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Lumbar Flexion Range (°)</td>
<td>42.7 (18.4)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Duration of Symptoms (yrs)</td>
<td>8.2 (8.0)</td>
<td>N/A</td>
<td>N/A</td>
</tr>
</tbody>
</table>
6.4.2 Influence of aLBP on temporal parameters of the STS movement

Individuals with aLBP demonstrated no differences in overall STS movement time compared to the NLBP group (Figure 6-1, P = 0.570), nor in the relative proportions of the movement spent either in the transition (Figure 6-2, P = 0.130) or deceleration phases (Figure 6-2, P = 0.510). However, individuals with aLBP demonstrated a trend towards a shorter acceleration phase (Figure 6-2, P = 0.067) and had an earlier time of seatoff (Figure 6-2, P = 0.036) compared to individuals without LBP.

6.4.3 Influence of aLBP on CM and COP during STS movement

Height-normalized peak CM velocities, of the horizontal and vertical components, did not differ between the aLBP and NLBP groups during the STS movement (Horizontal, P = 0.510; Vertical, P = 0.510), although individuals in the aLBP attained the minimum position of their CM (largely reflective of the peak trunk flexion) earlier than did the NLBP group (P = 0.037). Individuals with aLBP also demonstrated a higher normalized position of the CM at the start of the STS movement (P = 0.018), at seatoff (P = 0.029), at the end of the acceleration phase (P = 0.012) and at the end of the STS movement (P = 0.001). In addition, individuals with aLBP demonstrated a greater total path length of the CM throughout the STS movement in the sagittal plane compared to the NLBP group.
Figure 6-2: Ensemble averages of the total movement time, and the acceleration (black bar), transition (light grey bar) and deceleration (dark grey bar) phases of the STS movement. Relative contributions of each phase to the STS movement are reported in white (% of total movement time) and the relative times of seatoff are denoted by the vertical black lines and corresponding black numbers (% total movement time). Bar plots depict group means comparing individuals with LBP (aLBP; top bar) to those without LBP (NLBP; bottom bar). Significant group effects are denoted by * (P < 0.040) and non-significant between-group trends (P < 0.070) are denoted by †.
(P = 0.045). There were no group differences in the onsets, peaks or timing of peaks for the COP in either the sagittal or frontal planes during the STS movement.

6.4.4 Influence of aLBP on the angular kinematics of the STS movement

Individuals with aLBP demonstrated proximal and distal alterations to their joint movements during the STS compared to the NLBP group. Specifically, individuals with aLBP demonstrated a trend to reduced flexion of the trunk joint (Figure 6-3, P = 0.078) and increased ankle dorsiflexion (Figure 6-3, P = 0.046) relative to individuals without LBP. Consistent with these findings is a trend towards an earlier time of peak trunk segment flexion (P = 0.082) and a later time to attain maximum ankle position (P = 0.018) demonstrated by the aLBP group.

Individuals with aLBP also demonstrated altered trunk and hip positions at key time points throughout the STS movement compared to individuals with LBP. Individuals with aLBP demonstrated a trend towards a greater trunk angle (P = 0.094) and a reduced hip angle (P = 0.037) at the initiation of movement, likely due to a posterior pelvic tilt as evidenced by the trend towards a greater pelvis segment angle (Figure 6-3, P = 0.070) in this group compared to the NLBP
group. Trunk angle flexion at seatoff also tended to be reduced in the aLBP group (P = 0.067) consistent with the joint excursion findings reported above.

6.4.5 Influence of aLBP on the kinetics of the STS movement

During the STS movement, individuals with aLBP demonstrated similar lateral (P = 0.640) and vertical GRF (P = 0.290) patterns compared to individuals without LBP, although they demonstrated altered anterior-posterior forces, largely evident during the transition and deceleration phases. Individuals with aLBP demonstrated increased negative average forces (i.e. increased forces in the posterior directions across the movement phase) across both the transition (Figure 6-4, P = 0.012) and the deceleration (Figure 6-4, trend, P = 0.068) phases compared to individuals in the NLBP group. These findings indicate that individuals in the aLBP group applied a greater braking force during these phases than the NLBP group.

Both groups demonstrated similar joint torque patterns proceeding in a largely proximal to distal fashion to accomplish the STS movements, with group differences occurring only distally. The STS movement was accomplished through primarily trunk and hip torque during the acceleration phase, with a shift to hip and knee contributions during the transition phase and ankle contributions
Figure 6-3: Peak flexion angles of the trunk, hip and ankle during the STS movement. The inset depicts the actual angle of the pelvis angle relative to the right horizontal. Bar plots depict group means (±SD) comparing individuals with LBP (aLBP; black bars) to those without LBP (NLBP; grey bars). Significant group effects (P < 0.05) are denoted by *, whereas # denotes non-significant trends (P < 0.08).
Figure 6-4: Average normalized anterior/posterior ground reaction forces across the acceleration, transition and deceleration phases. Bar plots depict group means (±SD) comparing individuals with LBP (aLBP; black bars) to individuals without LBP (NLBP; grey bars). Significant group effects (P < 0.020) are denoted by *.
dominating during the deceleration phase with some additional trunk torque contributions. The joint torque contributions differed only at the knee during the transition phase (Figure 6-5, P = 0.023), such that the aLBP demonstrated greater knee torque relative to the NLBP group.

Relative joint contributions differed between the two groups only during the transition phase such that the individuals with aLBP used greater distal vs. proximal contributions to the STS movement than the NLBP group. Both groups demonstrated a relative reliance on proximal joints (hip, trunk) during the acceleration phase, shifting to the hip and knee as primary contributors during the transition phase to an ankle-dominant response during the deceleration phase. However, during the transition phase, individuals with aLBP demonstrated a greater contribution of knee and a lesser contribution of the hip to the STS movement (Figure 6-5, P = 0.006). No group differences between the relative contributions of the joints were found during the deceleration phase.

6.4.6 Influence of aLBP on the muscular contributions to the STS movement

The STS movement was accomplished in a largely similar manner between the aLBP and NLBP groups with respect to muscle activation patterns (Figure 6-6).
The movement was largely governed by the TA (P<0.001), GA (P<0.001) distally and the right RA (P<0.001; Left was not significant, P = 0.151, although the pattern was the same as the Right) and the EO (Left, P = 0.003; Right, P<0.001), during the acceleration phase. During the transition phase, TA activity peaked, with HA (P<0.001) and RF (P<0.001) also contributing to the distal response. Proximally, the ES at L3 (Left, P<0.001; Right, P<0.001), L1 (Left, P<0.001; Right, P<0.001) and T8 (Left, P<0.001; Right, P<0.001) governed the movement during the transition phase. The deceleration phase was largely controlled by the activity of the HA distally and the IO (Left, P<0.001; Right, P<0.001) proximally, although the EO and ES3 contributed bilaterally, albeit to a lesser degree than during the transition phase.

Group differences between the muscular contributions to the STS movement were evident, largely for the distal muscles, throughout the STS movement. Individuals with aLBP demonstrated greater TA (Figure 6-6, P = 0.048) and HA (Figure 6-6, P = 0.049) activation throughout the movement, with the most pronounced differences during the acceleration and transition phases for the TA and the transition and deceleration phases for the HA. Individuals with aLBP demonstrated greater RF activity during the transition and deceleration phases (Figure 6-6, P = 0.003) compared to the NLBP group.
Figure 6-5: Relative contributions of trunk, hip, knee and ankle torques to the STS movement across the acceleration, transition and deceleration phases. Stacked bar plots depict the group means of each joint torques contribution to the total torque across each phase comparing individuals with LBP (aLBP; right panel) to those without LBP (NLBP; left panel). Significant group by phase interaction effects (P < 0.025) are denoted by *, whereas # denotes a non-significant between groups post-hoc trend (P < 0.060). Inset diagrams depict the relative contribution of each joint torque (% total torque) to the total summed torque across each STS phase.
Figure 6-6: Average normalized muscle activities (% MVC) for the right trunk and left leg muscles across the baseline, acceleration, transition and deceleration phases. Bar plots depict group means (±SD) comparing individuals with LBP (aLBP; black bars) to those without LBP (NLBP; grey bars). Significant group effects (P < 0.05) are denoted by *, whereas significant group by phase interactions (P < 0.004) are denoted by # at the phases with significant post-hoc differences.
Average Normalized Muscle Activity

- **REO***: Shows a significant increase in muscle activity during acceleration compared to the baseline. The aLBP group (black bars) has a higher average EMG (% MVC) compared to the NLBP group (gray bars) across all phases.

- **RES1**: The aLBP group has a higher average EMG (% MVC) during acceleration, with a significant difference compared to the NLBP group.

- **RIO***: A similar pattern is observed with higher EMG for the aLBP group during acceleration.

- **RES3**: Significant increase in muscle activity during acceleration for the aLBP group.

- **RF**: High EMG for the aLBP group during acceleration.

- **HA***: Significant increase in muscle activity for the aLBP group during acceleration.

- **TA***: Significant increase in muscle activity for the aLBP group during acceleration.

- **GA**: All groups show minimal changes in muscle activity across different phases.

Note: The bars represent the average EMG (% MVC) with error bars indicating standard deviation.
Proximally, the aLBP group demonstrated increased activity of the IO (Figure 6-6, Left, $P = 0.019$; Right, $P = 0.046$) across all phases, indicating greater involvement of the abdominal muscles in this group throughout the movement compared to the NLBP group. The aLBP demonstrated increased activation of the right EO, (Figure 6-6, $P = 0.034$) but this was not evident on the left side ($P = 0.640$). A similar asymmetry was detected for the right ES1, which demonstrated increased activity in the aLBP compared to the NLBP group during the transition phase (Figure 6-6, $P = 0.004$).

6.5 Discussion

Individuals with aLBP performed the STS movement with an initial posterior pelvic tilt, restricted their trunk motion, used greater ankle motion and relied on the knee more throughout the transition and deceleration phases compared to the NLBP cohort. There were no group differences in the total movement time but the aLBP group demonstrated earlier seatoff times and shorter acceleration phases compared to the NLBP group. It appears that individuals with aLBP relied on an increased contribution of the knee during the transition from horizontal to vertical momentum that resulted in an increased anterior-posterior (AP) excursion of the CM, which may necessitate the greater braking forces demonstrated by this group.
6.5.1 Individuals in an active episode of pain may restrict their trunk movement

Consistent with previous findings (Coghlin and McFadyen 1994; Shum et al. 2005a), individuals with LBP tended to restrict their trunk motion (Figure 6-3), particularly lumbar flexion, during the STS movement. Shum et al. (2005a) demonstrated that individuals with LBP (tested 1-12 weeks following LBP episode) performed the STS with restricted lumbar and hip movements, and a reduced overall contribution of the lumbar spine to the movement. In the current study, individuals with aLBP did not demonstrate restricted hip motion, although they did perform the movement with similar reductions in peak trunk flexion (Figure 6-3). The discrepancies in the two studies may be due to the lower pain ratings of our aLBP cohort [median (range) 3 (0-7)] vs. those of Shum et al. [2005a; mean (sd), 5.7 (1.6)], such that perhaps trunk and hip restriction are related to the severity of current pain experienced. In addition, our cohort had a history of chronic LBP as opposed to the cohort under study in Shum et al. (2005a), in which all but one participant was experiencing their first episode of pain.

In addition to the trunk flexion restrictions demonstrated by individuals with aLBP in the current study during the STS movement, these individuals demonstrated
alterations that may be indicative of reluctance to extend their trunk during the
STS movement. Individuals with aLBP initiated the STS movement with their
pelvis posteriorly-tilted (Figure 6-3), which may reflect a reluctance or inability to
extend the trunk while seated. It is unknown whether individuals with LBP
demonstrate a posterior pelvic tilt during sitting prior to the STS, as this was not
reported in several groups under study (Coghlin and McFadyen 1994; Shum et
al. 2005a), however, individuals with LBP who demonstrated a flexion motor
control impairment [i.e. a tendency to flexion of the spine (Dankaerts et al.
2006b)] have been shown to sit with a more kyphotic lumbar spine than
individuals without LBP (Dankaerts et al. 2006a). Individuals with aLBP also
demonstrated a significant increase in bilateral IO activity (Figure 6-6), especially
during the deceleration phase of the STS movement, which through their action
as trunk flexors, may act to slow trunk extension during the later phase of the
movement. Individuals with LBP have demonstrated both reduced trunk flexion
and extension ranges of motion (ROM) during clinical testing (Waddell et al.
1992), as well as slower lumbar flexion and extension velocities during the STS
movement (Shum et al. 2005a) that may indicate reluctance or inability to move
through a normal range of motion. In the current cohort of individuals with aLBP
we demonstrated a reduced lumbar flexion ROM [mean (sd): 42.7 (18.4)°]
compared to the normative values for individuals without LBP (Keeley et al.
suggesting that the limited movement demonstrated by this cohort in performing the STS could be related to lumbar ROM restrictions.

Restriction or reluctance of trunk movement has been previously reported during STS (Shum et al. 2005a), bending to put on a sock (Shum et al. 2005b) as well as in response to self-generated balance perturbations (Mok et al. 2004), thus affecting not only volitional but anticipatory postural control. Restriction of trunk movement could be mediated either through active modification of movement patterns, perhaps secondary to pain-related fear, or through passive tissue adaptations that may restrict ROM. Individuals with LBP demonstrate reduced movement speed (Al-Obaidi et al. 2003), increased inhibition of muscle activity (Verbunt et al. 2005) and lumbar flexion restrictions (Thomas and France 2007) that were related more strongly to pain-related fear than reported pain levels. Therefore it is conceivable that individuals with LBP adapt their movement patterns with the intention of restricting trunk motion. Given that restriction of trunk movement is apparent not only during volitional movements but also during anticipatory postural adjustments (Mok et al. 2004), as well as during dual task walking with increased attentional demands (Lamoth et al. 2008), it is possible that restriction of trunk motion represents a higher-level task-related intention (i.e. central set) as opposed to merely a volitional movement adaptation.
Passive tissue alterations resulting in difficult or painful trunk movement provide another plausible contribution to the trunk motion restrictions demonstrated by the aLBP cohort under study, especially given the chronic nature of their pain. A similar cohort of individuals with chronic LBP demonstrated passive tissue alterations of the perimuscular connective tissue of the lumbar region (i.e. non-specialized connective tissue comprising the fascial planes) as measured by ultrasound (Langevin et al. 2009). These individuals demonstrated increased perimuscular tissue thickness and echogenicity of tissue that may indicate a greater number of levels of connective tissue within the superficial fascia (Langevin et al. 2009), that could lead to impaired movement. It has also been demonstrated that individuals with LBP demonstrate altered passive tissue characteristics in the lumbar spine that included increased side-specific asymmetry of passive muscle elastic energy, suggesting that there may be increased passive tissue resistance in this population (Gombatto et al. 2008). This finding suggests that not only are passive tissue alterations present in individuals with LBP but that they are related to altered movement patterns given that this population of individuals demonstrated asymmetry of trunk lateral bending during clinical exam. Although these changes were tested only in the frontal plane, it seems likely that similar passive tissue changes could be linked
to movement restrictions in the sagittal plane, such as those demonstrated by our population.

Although there is growing evidence of passive tissue alterations in individuals with LBP, it remains unknown whether these alterations are the cause or the result of altered movement patterns. Passive tissue adaptations may have developed secondary to tissue microinjury and inflammation, caused by some initial insult to the system (Langevin and Sherman 2007). However, there is increasing evidence that altered tissue stress secondary to changes in movement patterns may actually lead to passive tissue adaptation. It has been demonstrated that immobilized muscle sustains changes in the intramuscular connective tissue (i.e. perimysium and endomysium), including increased collagen formation and alteration of collagen fibre orientation, that acts to reduce compliance within the muscle (Williams and Goldspink 1984). Abnormal tissue stresses (either hyper- or hypo-mobility) have been shown to alter connective tissue properties resulting in fibrosis that can limit normal movement (Langevin and Sherman 2007). Thus, altered movement patterns (regardless of the cause) can lead to tissue modifications that can further limit movement, leading to a vicious cycle of decreased mobility and tissue maladaptation (Langevin and Sherman 2007).
There exists the possibility, then, that movement restrictions secondary to pain-related fear or habitual movement patterns, and passive tissue remodelling could be linked and may contribute to the chronic cycle of pain recurrence in this population. Pain-related fear has been linked to reduced trunk motions (Thomas and France 2007) and overall reduced physical activity levels in individuals with chronic LBP (Swinkels-Meewisse et al. 2006), although this remains controversial (Verbunt et al. 2001). It is plausible that reduced movement as a result of pain-related fear could result in the tissue adaptations demonstrated in this population (Gombatto et al. 2008; Langevin et al. 2009). However, equally plausible is that these individuals performed repetitive movement patterns that lead to passive tissue alterations, either from hyper- or hypo-mobility (Langevin and Sherman 2007), that increase injury susceptibility. Gombatto et al. (2008) report that asymmetries were evident in both individuals with and without LBP but that the degree of asymmetry was greater in those who had a history of LBP. A cohort of individuals with LBP who participated regularly in sports requiring asymmetric movements demonstrated greater asymmetries and rotation-related impairments than individuals who participated in sports requiring symmetric movement patterns (Van Dillen et al. 2006), thus relating the movement impairments to repeated movement patterns. While this does not provide
definitive evidence that the repeated movement patterns lead to pain, it does provide a link between the movement pattern and the movement impairment, which, given research on tissue adaptation, does provide support for this cascade of events as a plausible mechanism. Regardless of the initiating circumstances of the pain experienced, it remains clear that neither spontaneous recovery nor therapy seems to address the underlying tissue changes that may be linked to recurrent episodes. Until these underlying changes are resolved, it seems unlikely that recurrent episodes will be avoided.

6.5.2 Individuals with aLBP may favour a knee strategy

Individuals with aLBP demonstrated, in addition to restricted trunk movement, increased ankle movement and a reliance on knee torque (with reductions in hip torque) particularly during the transition phase of the STS movement (Figure 6-5). A knee strategy (Schenkman et al. 1990; Coghlin and McFadyen 1994) is typified by high knee torque relative to the other joints [the ankles, hips and trunk torques individually contribute between 35% and 53% of the contribution of the knee joints; (Coghlin and McFadyen 1994)], as well as use of the trunk and hip to control forward momentum. This strategy may be desirable in the aLBP cohort given the trunk restrictions demonstrated by this group. Coghlin and McFadyen (1994) reported that instead of preferentially choosing a knee strategy to perform
the STS, their cohort distributed the torques among their joints more evenly, which was not evident in our current cohort. It is possible that differences in the subject groups under study could have contributed to these differences but unfortunately, subject demographics and LBP history were not adequately reported by Coghlin & McFadyen (1994). Shum et al. (2007) reported reduced lumbar and hip extensor torques during the STS between asymptomatic and symptomatic participants but did not report the contributions of the distal joints to the movement, making it difficult to judge whether this cohort reported an overall reduction in joint torques [similar to (Coghlin and McFadyen 1994)] or decreased proximal torques in conjunction with increased knee torque, which may indicate the use of the knee strategy.

As mentioned, the knee strategy necessitates control of forward momentum, largely by the hip and trunk in order to prevent loss of balance during the STS movement (Coghlin and McFadyen 1994). It appears that forward momentum was controlled in the LBP cohort under study through a larger braking force that was apparent during the transition and deceleration phases of the STS movement, consistent with that reported by Roebroeck et al. (1994). It does not appear that increased trunk extensor muscle activity controlled this braking force given that there were no group differences in ES activity. Instead the increased
contribution of the RF and HA muscles during the later phases of the movement demonstrated by the aLBP group might have contributed a backward-directed GRF through their biarticular muscle action. Roebroeck et al. (1994) reported that the RF acted in a largely isometric fashion while HA was shortening throughout the later phases of the STS movement. Activation of the HA would have a greater mechanical advantage to extend the hip and in conjunction with the near isometric activity of the RF (i.e. acting similar to a tendon to redistribute the torques from hip to knee) would contribute to knee extension (Roebroeck et al. 1994). The relative balance between these two muscles, if favouring RF activation, could act to control a rearward directed force applied at the feet, acting to slow the body’s forward momentum. Given that individuals with aLBP demonstrated an increased movement of their CM in anterior/posterior direction, they may have required a larger braking force to prevent a forward loss of balance during the later phases of the STS movement. Thus it is possible that although these individuals spared their trunk movement through relying on a knee strategy, this choice may have reduced their stability, potentially increasing the chance that they could experience injurious balance loss.
6.5.3 Persistent altered volitional movement patterns or short-term response to heightened pain

Individuals with aLBP demonstrate altered movement patterns in performing the STS manoeuvre, indicating preference of a knee strategy that would reduce the reliance on proximal torques to complete the motion. However, given the heightened pain experienced by this cohort it is unknown whether this alteration represents a short-term modification to increased pain or changes that are sustained between pain flare-ups. Individuals with chronic pain demonstrate altered STS movements that are distinctive enough to be distinguished from healthy or malingering individuals using neural networks (Gioftsos and Grieve 1996), but may be smaller in magnitude than those demonstrated by individuals who are in a heightened period of pain.

Both our current findings and those of Coghlin and McFadyen (1994) detail slight modifications in STS strategy (either favouring a knee strategy or torque-sharing among joints) in cohorts with chronic pain. These findings are smaller in magnitude [approximately 5° reduction in absolute trunk flexion, (Coghlin and McFadyen 1994) and the current study] than those reported by Shum et al. (2005a) who demonstrated a 16° reduction in the LBP group compared to healthy controls. In fact, the aLBP cohort demonstrated hip and trunk motions
that fell somewhere between the chronic LBP and healthy control groups in both the Coghlin and McFadyen (1994) and Shum et al (2005a) studies. Some of the absolute angular differences may relate to slight differences in initial body position and methodology among the three studies but it is clear that the relative change was greater in the cohort described by Shum et al. (2005a).

The individuals with LBP studied by Shum et al. (2005a) had greater pain levels [mean (sd), 5.7 (1.6) vs. median (range), 3 (0-7) in the current study] and were experiencing their first episode of LBP, whereas the aLBP cohort in the current study had a history of chronic pain lasting on average 8.2 years since initial onset [Table 6-3, mean (sd), 8.2 (8.0) years]. Thus the greater restrictions reported by Shum et al. (2005a) could be related to the heightened pain levels reported or may be related to increased pain-related fear, given that this is a stronger predictor of reduced trunk flexion individuals with LBP (Thomas and France 2007). Unfortunately, pain-related fear was not assessed by Shum et al. (2005a) or in the current study. It is plausible, however, that the greater trunk restrictions in the cohort experiencing their first episode of LBP (Shum et al. 2005a) could lead to sustained alterations, such as those demonstrated by the more chronic LBP cohorts [current and (Coghlin and McFadyen 1994)]. Indeed the greater reliance on a knee strategy in the current aLBP cohort could be a short-term
adaptation to a recent onset of pain that is obscuring or exaggerating the altered movement patterns that persist between pain episodes. Further longitudinal study on the STS movement patterns over time in the current aLBP cohort might provide a more solid understanding of which patterns are related to a pain exacerbation and which are persistent over time.

6.6 Conclusions

Individuals with aLBP demonstrated movement restrictions at the trunk with compensation by the distal joints that indicate that this cohort favoured the use of a knee strategy to complete the STS movement. It is not known whether this alteration is due to a volitional adaptation or higher level intention to restrict movement possibly due to fear of movement or pain, or is a result of passive tissue adaptations that may have resulted from habitual movement reductions. In addition, it is unknown whether these movement restrictions represent a habitual movement pattern that may have led to altered motor control and tissue adaptations or whether these movement restrictions represent a short-term adaptation to a recent, painful “flare-up”.
6.7 Acknowledgments

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Chapter 7: Summary and conclusions

7.1 Summary of results

The objective of this thesis was to determine the impact of chronic LBP on neuromuscular control during unexpected balance perturbations and a volitional movement, among individuals with LBP during a quiescent period of pain vs. an exacerbation of pain compared to individuals with no history of LBP. By doing so, we hoped to delineate the movement patterns characteristic of the quiescent and active cycles of recurrent LBP in order to understand the underlying neuromuscular changes that may persist between exacerbations and may contribute to recurrence of pain.

In Chapter 3, the torque strategies used to respond to unexpected, multi-directional support surface translations by individuals with no history of LBP were quantified as a means of understanding the underlying automatic postural responses used by the nervous system to maintain standing posture. We hypothesized that the magnitudes of the joint torques in both the frontal and sagittal planes would vary with direction of perturbation. We determined that joint torque magnitudes varied in a more or less sinusoidal fashion with perturbation direction in both the frontal and sagittal planes but that there were some
similarities in the relative joint torque contributions to all perturbation directions. Namely, it appears that an early proximal contribution representing implementation of a hip/trunk strategy is present, regardless of perturbation direction and that the relative contributions of the distal joints depend on biomechanical or anatomical constraints of the body. It appears that when one joint is limited in its capability to respond (for example, the ankle responding to backward body sway), the next most proximal joint (in this example, the knee) contributes significantly to the response. Therefore, it appears that despite direction-specific differences in torque magnitude, the overall aim of the postural control system is to use an early proximal strategy with a second and greater distal contribution to the corrective response, regardless of perturbation direction. These results support a single neural control strategy to respond to unexpected balance perturbation regardless of the direction of induced balance disruption.

In order to understand the underlying neuromuscular changes that may persist between recurrent bouts of pain, the automatic postural responses to multi-directional support surface translations in individuals with a history of chronic LBP who were in a quiescent period of their pain, were compared to a healthy control group, by quantifying both joint torque strategies and muscular contributions to the corrective responses. Individuals with chronic LBP
demonstrated similar response strategies to those of healthy controls that varied sinusoidally with perturbation direction; however, these individuals demonstrated heightened sagittal plane ankle torques during the initial period (most evident following backwards perturbations inducing forward sway) and reduced sagittal and frontal plane trunk torques during the active phase of the response. The increased ankle torques demonstrated during the initial period may reflect altered periarticular tissue stiffness secondary to sustained or habitual postures, or changes in baseline tonic activity of the ankle musculature possibly mediated through feedforward stiffening of the ankle musculature. Increased activation of the Gastrocnemius but not the Tibialis Anterior primarily following backward perturbations might support neuromuscular alterations in ankle stiffness. Reduced active trunk torques, regardless of perturbation direction, could be mediated through equivalent agonist-antagonist torque production, thus implicating trunk stiffening as a potential response strategy employed by individuals with chronic LBP following unexpected balance perturbation. Indeed, increased relative contributions of the proximal musculature, both dorsal and ventral (RA, IO and EO, ES, albeit with some side-specific group differences) suggest that individuals with chronic LBP do use a strategy of co-contraction in preparation for the destabilizing response of the impending perturbation, regardless of perturbation direction. This strategy may be implemented through
altered central set given that these individuals may demonstrate a cognitive
decision to restrict trunk movement, potentially secondary to pain-related fear or
given the impairment of proprioception demonstrated by individuals with chronic
LBP.

Chapter 5 investigated the automatic postural responses in individuals with a
history of chronic LBP who were actively seeking treatment for their pain and
were therefore assumed to be in an active pain episode. We hypothesized that
individuals with aLBP would adopt a strategy of stiffening at the trunk in order to
minimize trunk excursion and/or forces applied to the trunk. Although trunk
restrictions as evidenced by reduced trunk torques were observed in the
quiescent cohort (Chapter 4), no alterations in the magnitude of the corrective
torque responses were found compared to individuals with no pain, following
postural perturbations. However, the peak latencies of the torque responses
were earlier in this group, both proximally and distally, suggesting that either the
response was initiated sooner or developed more quickly relative to the onset of
perturbation. Earlier peak latencies could be indicative of altered central set
perhaps acting to either increase the gain of the postural responses, which could
contribute to faster torque generation, or to increase the fusimotor drive resulting
in the spindles being closer to firing threshold, initiating an earlier response.
Support for the latter explanation is provided by the muscular responses in this cohort, which demonstrate increased activation of both proximal (ES, IO and EO) and distal muscles (TA and GA) in perturbation directions when a given muscle acts as a prime mover (also the direction of muscle stretch). This direction- and muscle-specific response may indicate increased spindle sensitivity to stretch. Regardless of the etiology, these earlier peak torque latencies indicate a pervasive response that is likely due to central influences given that joints both near to and remote from the site of pain are affected.

Given that the cohort experiencing an exacerbation of their pain (aLBP) would be expected to have greater volitional influences of pain on postural coordination, in Chapter 6 we quantified the movement patterns of these individuals during a volitional movement, namely the STS. The STS movement is an activity of daily living that may provide a conflict between movement stability and trunk motion restrictions that may be unique to individuals with LBP. The movement strategy implemented may provide a window into the intentions governing an individual’s volitional movement patterns. Although individuals with aLBP performed the STS movement with the same movement time as individuals without LBP, they demonstrated a shorter acceleration phase, reduced trunk flexion, increased ankle motion and an increased reliance on the knee for the transfer of horizontal
to vertical momentum. In addition, these individuals initiated the movement with a posterior pelvic tilt and used a greater relative contribution of the ventral, proximal musculature (IOs) during the deceleration phase of the movement, which suggests an inability or reluctance to move through trunk throughout its full extension range of motion. These findings suggest that individuals with aLBP favour a knee strategy to perform the STS, which enables restriction of trunk motion (both flexion and extension), and requires compensation by the distal joints. Thus, it may be that individuals who have had a recent “flare-up” of their pain move with the intention of restricting trunk motion, however, it is not known whether this pattern of movement persists between painful episodes. It is plausible that individuals in an exacerbation of their pain may reinforce movement patterns, such as trunk restriction, that may be detrimental to the prevention of future pain episodes. Alternatively, these individuals may have underlying passive tissue alterations that make it difficult for them to move their trunk throughout its normal range of motion and may require the use of alternate movement strategies to complete the required movement.

Collectively, these results confirm that altered movement strategies persist in the chronic, recurrent LBP population between painful episodes. These movement strategies may reflect an intention to restrict trunk movement through a strategy
of trunk stiffening and may result in reduced stability that could make these individuals susceptible to future injury. Individuals with chronic LBP who are in an exacerbation of their pain appear to have a generalized heightening (i.e. encompassing proximal and distal joints) of their postural responses to unexpected perturbation that may reflect a short-term modification to a heightened pain state. These individuals move to restrict their trunk during volitional movements such as the STS, which require the use of alternate strategies that may again negatively impact their postural stability. This type of movement pattern, if repeated, may form the basis of the persistent movement patterns, both volitional and automatic, demonstrated by individuals in a quiescent period of their pain cycle that may increase susceptibility to future LBP episodes.

7.2 Study limitations

Given the controlled nature of the proposed studies and the balance paradigms used in this thesis, our findings will be directly applicable only to other laboratory based balance studies of this type. However, it is hoped that the diversity of the samples obtained (which had more or less equal proportions of males and females across a wide age range per group) enabled us to learn about characteristics of motor coordination that are applicable to balance challenges in
the real world. By using the sit-to-stand motion (an activity of daily living) and the support surface translation paradigm, which simulates a balance perturbation that could be analogous to standing on a moving bus, train or escalator, it is hoped that our results are predictive of differences that might be present in the participants' daily lives. However, the responses obtained are only directly applicable to perturbations of the type reported in the methods section, generated using a mechanically-driven platform, given that the type of perturbation stimulus can influence the responses obtained (Brown et al. 2001).

The results obtained by these studies should be considered within the limitations that are standard to the calculation of net joint torques using inverse dynamics techniques. Errors may have resulted in the determination of joint centers and segmental parameters due to the necessity to filter kinematic signals significantly to minimize magnification of noise in the double differentiation calculation. In addition, the anthropometric models used have an associated error in predicting individual subject characteristics, which could be larger for females than males given that the model was developed using male subjects only (Zatsiorsky and Seluyanov 1983). Since the torques calculated represent the net of all forces and moments applied to a given joint, both active and passive, it is impossible to determine specific muscle contributions to the posture-righting response.
Because the joint torque responses extend beyond the time of the platform deceleration, it is not possible to separate the torque responses to the initial destabilizing perturbation from those of the secondary perturbation resulting from platform deceleration (Carpenter et al. 2005), however these effects should be the same for all individuals.

Surface electromyography has several limitations that must also be considered. Given that we recorded myoelectric signals during dynamic movements, the recording volume of the electrodes could have captured the electric signals of different muscle fibres throughout the movements. However, these errors were likely small in magnitude relative to the changes in activation elicited either by different perturbation directions or between groups and likely similar in relative magnitude across individuals. In addition, tissue differences between individuals could have provided different low-pass filtering of the underlying myoelectric signals, although these differences should be minimized since we used subject-specific amplitude normalization of EMG signals and the groups had similar BMI values.
7.3 Significance to rehabilitation and future directions

Given that the cost of care for LBP is escalating and that effective treatments to prevent recurrent episodes of LBP continue to elude health care providers (Pengel et al. 2003), it is imperative that we develop a greater understanding of the persistent impairments that plague individuals with chronic LBP. Understanding the cycle of pain and quiescence in individuals with chronic, recurrent LBP may be the key to developing effective treatment strategies to prevent future pain episodes. Determination of the persistent impairments evident between pain episodes may provide insight into the factors that are not being ameliorated either through spontaneous healing or through the many treatment options available, and may therefore contribute to pain recurrence.

Among the important findings presented in this thesis are that trunk restrictions are present in individuals with chronic LBP in a pain exacerbation when performing a volitional movement, and in individuals with chronic LBP in a quiescent period elicited by unexpected balance perturbation. Trunk restriction during the STS movement may reflect underlying tissue restrictions or habitual movement patterns that have developed over time, given the length of time that these individuals have been experiencing chronic pain. Alternatively, restriction
may be a volitional adaptation to the recent pain exacerbation that they have experienced that could lead to sustained movement patterns which, in turn, prevent return to normal functioning and increase susceptibility to future pain episodes.

The Kinesiopathological Model proposed by Sahrmann (2002) hypothesizes that repeated movements or sustained postures can lead to a loss of movement precision that induces changes in the neuromuscular system, including changes to tissue properties and/or altered muscle activation patterns that contribute to LBP symptoms and/or injury. It is possible that pattern of trunk restriction evident during the STS movement in this study represents a habitual movement pattern that has led to cumulative stress on the tissues, making the individual susceptible to injury and that further exacerbation of symptoms could result from sustaining these habitual patterns. Unfortunately, it is not possible based on the results of the current investigation to determine whether these patterns contributed to the initial episode of pain, but it does seem logical, given the repetitive patterns of recurrent pain, that these movement alterations are likely related to the cyclic nature of the LBP problem. Until these altered movement patterns are addressed, complete resolution of local tissue irritation/inflammation, symptoms and prevention of future episodes seems unlikely.
A second hypothesis presents the possibility that some initial trauma led to injury of the spinal ligaments causing a cascade of events that, in turn, resulted in impaired proprioception, altered movement patterns and, ultimately, chronic LBP (Panjabi 2006). While this hypothesis includes the possibility that repetitive, cumulative trauma [similar to that suggested by (Sahrmann 2002)] could have led to the initial low back injury, it also suggests that an initial traumatic event could have lead to pathology. Panjabi (2006) suggests that damage to local mechanoreceptors leads to aberrant feedback to the neuromuscular control unit, resulting in the generation of inappropriate muscular response patterns. Given that spinal stability is critically dependent upon sufficient muscle activity, if the pattern of required activity is mismatched to the task requirements, injury could occur. With regard to the current study, the trunk restriction patterns we have identified could be a response to an initial episode of pain and/or inaccurate proprioceptive feedback, resulting in altered movement patterns, perhaps influencing both volitional and automatic postural coordination. Panjabi (2006) suggests that inaccurate feedback would result in delayed muscular responses, which is inconsistent with our findings. It is perhaps more likely that the movement patterns we have quantified represent sustained altered patterns that have led to symptoms or injury (consistent with the Kinesiopathologic Model) or
perhaps developed in response to an initial pain episode (whatever the etiology), leading to persistent motor control impairments that are linked to the recurrent pain bouts experienced by this chronic LBP population.

One potential benefit of these findings is to understand the importance of treating the underlying and sustained motor control impairments that persist between pain episodes in this population. If altered movement patterns lead to the recurrent pain episodes then it is logical that by ameliorating these impaired movement patterns it may be possible to induce changes both at the tissue and neuromuscular levels; these changes could allow the individual to return to increased variability and distribution of movement and appropriate control of movement precision.

The Movement Systems Impairment-Based Classification system (MSI) provides a means of clinically characterizing abnormal and/or symptom-provoking movement patterns and attempts to modify functional activities and trunk movements in a direction-specific manner with the goal of normalization and redistribution of trunk movement and alignment (Maluf et al. 2000; Van Dillen et al. 2005). The system classifies individuals into one of five movement categories based on consistent faulty movement patterns and symptom reproduction.
(lumbar rotation, lumbar extension, lumbar flexion, lumbar rotation with extension and lumbar rotation with flexion); the proposed intervention targets movement re-education to specifically address these motor control impairments. This method has been shown to be effective over a six month period at decreasing pain, increasing function and reducing recurrences (Harris-Hayes et al. 2005), but further study is needed to determine whether these altered movement patterns lead to resolution of the underlying neuromuscular impairments and tissue alterations that may be present in individuals with chronic LBP. Testing of automatic postural responses and volitional movements prior to and following MSI therapy, with long-term post-therapy follow-up could provide further evidence of the link between movement impairment and pain episodes.

One final finding that may be important to understanding the recurrent pain cycle typical of chronic LBP is the global heightened postural response that was evident in response to unexpected balance perturbation in the aLBP population, which may represent a short-term adaptation to the recent exacerbation of their symptoms. This adaptation could be necessary if pain has rendered peripheral feedback inaccurate (Panjabi 2006), which is possible given the impaired proprioception demonstrated by individuals with LBP (Brumagne et al. 2000) (Brumagne et al. 2004; Descarreaux et al. 2005). Horak, Diener and Nashner
(1989) reported that even healthy individuals used a “non-specific enhanced postural response” when information about the perturbation was lacking.

Given the influence of pain as well as any underlying pathology that may be present in this chronic pain population that may impair peripheral feedback, it seems logical that a general heightened postural response could be an effective strategy to maintain balance. However, it is not known whether this heightened response is directly related to an increase in pain levels or tissue injury, or may appear secondary to pain-related fear. In addition, the time course of these modifications is unclear and the extent to which these changes persist or diminish over time is unknown, although it was not present in our quiescent cohort. In addition, it is not apparent whether a short-term adaptation of this type could lead to long-term adaptations that may increase the likelihood of recurrent pain episodes.

Further study of repeated postural assessments at the time of a pain exacerbation, with long-term follow-up, might provide information on the time course of the development of these heightened responses and on the sustained changes that are present once symptoms and/or fear have abated. In addition, characterizing the reported pain levels and pain-related fear may provide a link
between these constructs and the postural responses exhibited. By understanding the nature of these interrelationships, appropriate targets for treatment, either pharmacologically to reduce the influence of pain or behaviourally to reduce the influence of pain-related fear may shorten the duration of this heightened response and mitigate the long-term consequences of an altered pattern of this type.
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Appendix 1 – Informed consent document

Effects and Mechanisms of Specific Trunk Exercises in Low Back Pain

Lay Summary and Consent Form to Participate in a Research Study

Invitation
You are being invited to participate in a research study that is described below. The study is conducted through the Department of Physical Therapy at the University of Vermont and sponsored by the National Center for Medical Rehabilitation Research. The Principal Investigator (Sharon Henry, PhD, PT) or a member of the research team will further explain this study to you, and answer any questions you might have.

Aims of study
The purposes of this study are to learn more about: 1) how trunk muscles are affected by low back pain; 2) which exercises might be most beneficial for people with certain kinds of low back pain; and 3) how these exercises influence trunk muscle function. By having a better understanding of which muscles are affected by low back pain, rehabilitation specialists can design exercise programs and therapeutic interventions that are more specific and more effective.

Background
Low back pain affects 80% of Americans at some time during their lives. Although recovery usually occurs within 6 months, there is a 50% recurrence within one year’s time. It has long been thought that poor control of trunk muscles may lead to abnormal forces across the spine, which then damage local spinal structures, thus, leading to low back pain. However, we know little about the function of specific trunk muscles in healthy subjects during various activities of daily life. Furthermore, the precise muscle dysfunction associated with low back pain has not been well characterized at all. In addition, we know little about which exercise protocol is most beneficial for particular subgroups of people with low back pain.

Procedures for testing sessions (healthy and low back pain subjects)
If you do not have low back pain, you will be asked to come to the Human Motion Analysis Laboratory for 2-4 testing sessions, each lasting approximately 2-3 hours. If the muscle recordings that are taken during the first two sessions are stable and repeatable, you will not need to come back for the third and fourth testing sessions (this will most likely be the case). The first session is today; the second session will be in 10 weeks from today; the third session in 6 months from today; and the fourth session in one year. At the end of the series of testing sessions, you will be compensated $50.00.

If you do have low back pain, you will be asked to come to the Laboratory for four testing sessions and 10 treatment sessions. The testing sessions will last approximately 2-3 hours and each treatment session will last approximately 30-60 minutes, depending how you are progressing with your exercise program. The first testing session is today; the second testing session will be approximately 10 weeks from
today. If you complete the first and ten-week testing and the 10 treatment sessions, you will be compensated $30. The third testing session will be in 6 months from today, and the fourth testing session in one year; after you complete each of these two testing sessions, you will be compensated $25 for each session. At 18 and 24 months from today, you will receive some questionnaires in the mail, asking about your pain, functional and work status, as well as any low back pain recurrences. If you complete and mail in both sets of questionnaires, you will be compensated another $20, for a total of $100 compensation over the course of your participation in the study.

During the first testing session, we will first take measures of your body weight and height, take a brief history (if a subject with low back pain), record your current exercise and work status, all of which will take approximately 10 minutes. Then, we will ask you to fill out some questionnaires (McGill Pain questionnaire, Quality of Life, and the Oswestry Scale) which will take about 10-15 minutes. The questionnaires ask you information about the location and intensity of your pain and how your pain has affected your activities of daily life.

Next, you will have surface electrodes placed over certain muscles. Each electrode has two 10 mm disks separated by 20 mm, and the electrode is stuck to your skin with an adhesive similar to that of a band-aid. In order to have a good recording of your muscle activity, the appropriate skin preparations (shaving and rubbing with alcohol) must be done, and recording electrodes must be placed over the skin above your muscles. Surface electrodes will be placed over the following sites: over the muscles in the front and back of your shin; over the muscles in the front and back of your thigh; over the muscle in your hip; over the muscles in your lower and upper back; over the muscles in your abdominal region. Your body movements will be detected by special cameras that track the positions of spherical reflective markers that are placed on various parts of your body. Three markers will be placed on each of the following body segments: the shin, the thigh, the pelvis, and the trunk.

In order to gather information about muscle function and body movement, we will ask you to do three activities while the surface electrodes and spherical markers are in place: 1) a double leg raise test; 2) a sit to stand test; and 3) a balance test while standing on a platform that can move in the horizontal direction. For the double leg raise test, we will ask you to lie on your back with your hips and knees comfortably bent. You will be asked to lift your feet about 1 cm off the bed and hold the position for 5 seconds. This test will be repeated 3 times.

During the second activity, a sit to stand test, you will be asked to move from a sitting to a standing position at your own pace. The chair will be adjusted to your height such that your knees will be at a 90 degree angle while sitting, and an accelerometer, a small lightweight device (3 x 3 cm), will be attached to your trunk so we can determine when you started your movement. This test will be repeated 3 times.

Lastly, for the third activity, you will be asked to stand barefoot on a platform that can move horizontally in any of 12 different directions. The directions of movement will be unknown to you in advance. You will receive a total of 60 different trials during the testing session. You may rest between trials at any time, for as long as you need, if necessary. As a safety precaution, you will wear a modified parachute harness that is suspended from the ceiling, but which does not give you any weight support. The harness
consists of a series of straps that go around your waist and over your shoulders. The harness will be adjusted comfortably for your body size and height.

The way in which you automatically activate your muscles during each of these three activities will be measured. Disc electrodes that are placed on various surface locations of your skin will measure the electric fields emitted by your muscles as they move.

**Procedures for treatment sessions** (for low back pain subjects only)

If you agree to be a volunteer participant in this study, you will be randomly assigned to one of two exercise protocols (strengthening/conditioning or stabilization) which will be supervised by a licensed Physical Therapist. We will ask you to attend physical therapy 1 time per week for 10 weeks; each session will last 30-60 minutes which is a usual and customary treatment session.

If you are assigned to strengthening/conditioning protocol, you will be instructed in exercises designed to strengthening your trunk muscles and to improve your endurance. At each treatment session, you will review these exercises with your Physical Therapist and the exercises will be progressed accordingly. You will also be instructed in exercises, such as walking, swimming or biking, to improve your endurance. The physical therapist will review with you information about the anatomy of the back, lifting techniques, postures to avoid, and will review a home exercise program with you. We will ask you to record the number of times you complete your home program.

If you are assigned to the stabilization protocol, you will be instructed in exercises designed to contract specific deep trunk muscles simultaneously around your spine. In order to learn how to activate these muscles, you will start in position on your back and then progress to more functional positions such as sitting, kneeling, walking, and finally to positions/activities that you currently find painful. To facilitate your ability to perform the co-contraction of these deep trunk muscles, real-time ultrasound imaging of your abdominal wall and back muscles will be used to provide you with visual feedback about the muscles that you are trying to contract. This ultrasound is the same type that is used to image fetuses in pregnant women. The physical therapist will emphasize that the contractions should be very slow and low effort and should be coordinated with your breathing. You will also be instructed in exercises, such as walking, swimming or biking, to improve your endurance. The physical therapist will review with you information about the anatomy of the back, lifting techniques, postures to avoid, and will review a home exercise program with you. We will ask you to record the number of times you complete your home program.

Once you have completed the ten physical therapy sessions, your physical therapy chart will be reviewed by the PI or a research assistant, so that data about your physical therapy treatment and progress can be gathered. Only data pertinent to this study, such as your muscle strength and flexibility; pain location and intensity; exercise compliance; treatment progression; cardiovascular status; treatment and history for any previous low back pain episodes, will be gathered. No other personal identifying information will be taken from your chart.

At some point during your physical therapy treatment, another physical therapist who is participating in the administration of the exercise protocols may observe one of your treatment sessions. This is a way to insure quality and consistency of the exercise
protocols administered by different physical therapists at different clinical sites. You may choose not to be a part of this audit process and still continue to fully participate in all other aspects of the study.

**Risks/Discomforts**

1. There is a small risk that you may fall and injure yourself while standing on the platform that moves unexpectedly in the horizontal directions. The risk of injury is minimized by having you wear a modified parachute harness so you cannot actually hit the ground, and there is always a person nearby to guard you from falling.

2. There is a very remote risk that a minor electric shock (tingling) from one of the electrodes could occur if the equipment malfunctions. The risk and amount of potential shock has been minimized by the design of the equipment. Electronic equipment will be checked for safety by the clinical engineering department of FAHC every 12 months.

3. If your skin is very sensitive to adhesives, you may have a reddened area at the site of electrode placement which lasts several hours.

4. Diagnostic ultrasound will be used in this study and pose no risk to the subject. Ultrasound is used for a variety of patients, including pregnant women, without any undue side effects.

**Benefits**

There may be direct benefits to you as a volunteer participant in this study to the extent that the exercises provide you with relief of pain, increased mobility and strength, and improved function. Even if you do not experience these direct benefits, the information gained from these experiments will improve our scientific understanding of the control of trunk muscles in people with and without low back pain. With a better understanding of how the trunk muscles are controlled, rehabilitation specialists can better evaluate the problem of low back pain and design more specific treatments (for example, what exercises to prescribe) for people with certain kinds of low back pain.

**Alternatives**

There are many other alternatives available for the treatment of low back pain, and some of those include massage and soft tissue techniques, modalities (ultrasound, heat, cold, electrical stimulation), chiropractic care, osteopathic care, spinal mobilization, work hardening programs, and other forms of therapeutic exercise.

**Costs**

As a participant in this study, there are no costs to you.

**Compensation**

Upon completion of the study, you will receive a total of $100 (for volunteers with low back pain) or a total of $50 (for volunteers without low back pain) in compensation to cover your travel expenses. In addition volunteers with low back pain will receive 10 free physical therapy treatments. If you are unable to complete the study,
due to fatigue or any other reason, the compensation will be prorated based on the amount of time elapsed from the beginning of your participation in the study, as outlined under Procedures for Testing on page one.

**Confidentiality**

All records from this research will be kept in confidence and will not be given to any one unless you give us written permission to do so with the exception of representatives from the National Institutes of Health (the funding agency), the UVM Committee on Human Research, or other related research agencies who may request access to the research records. Your results may appear in publication, but they will not be identified with your name. All data is encoded with numbers and the codes are kept in a locked cabinet file.

**Withdrawal**

As a volunteer participant in this study, you are free to refuse to participate or to withdraw from participation in the study at any time. Withdrawal will in no way affect your relationship with or treatment at the Fletcher Allen Health Care or at the University of Vermont. If, during your participation in this study, we need to continue to test you because some new findings develop, we will ask you if you are willing to continue with the testing. If you are willing to continue, we will proceed, and if not, the study will terminate as scheduled.

**What Happens If I Am Injured?**

If you are injured or become ill as a direct result of participating in this research project, Fletcher Allen Health Care, the hospital affiliated with the University of Vermont, will provide reasonable and customary medical care for that injury or illness at no cost to you providing certain conditions are met. These conditions are:

1) The costs of the care are not covered by your insurance or other third party coverage, or you have no such coverage or insurance:
2) It is the opinion of the investigator and/or sponsoring agency that the injury or illness is a result of the research;
3) For studies which provide treatment of a specific condition or disease, it is the opinion of the investigator and/or sponsoring agency that the injury is not associated with your disease/condition or with the expected complications of the usual therapies for the disease/condition;
4) You have followed all of the directions of the investigator;
5) You have notified the investigator of the injury or illness in a timely manner after onset; and
6) You have followed medical advice regarding the injury or illness.

In instances where the Study Sponsor has agreed to pay some or all of the costs of this care, reimbursement will be sought by Fletcher Allen Health Care directly from the Sponsor. The fact that Fletcher Allen Health Care provides free treatment or care to you as a result of a research-related injury or illness as just described is not an admission by
Fletcher Allen Health Care or the University of Vermont that it is responsible for such injury or illness.

It is not the policy of the University of Vermont or Fletcher Allen Health Care to provide any further financial compensation in the event of an injury or illness. You should understand, however, that by acknowledging this you are not waiving or releasing any of your legal rights.

You may contact Dr. Sharon M. Henry, the Investigator in charge of this study, at (802) 656-8146 for more information about this study. If you have any questions about your rights as a participant in a research project or for more information on how to proceed should you believe that you have been injured as a result of your participation in this study you should contact Nancy Stalnaker, the Institutional Review Board Administrator at the University of Vermont at 802-656-4067.
Appendix 2 – Reprint of publisher

RESEARCH ARTICLE

Responses to multi-directional surface translations involve redistribution of proximal versus distal strategies to maintain upright posture

Stephanie L. Jones · Sharon M. Henry · Christine C. Raasch · Juvena R. Hitt · Janice Y. Bunn

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Abstract Evaluation of postural control in multiple planes is necessary to determine the movement strategies used to respond to unexpected perturbations. The present study quantified net joint torques of the lower limbs and trunk in the sagittal and frontal planes following multidirectional surface translations. Twenty-one healthy subjects stood with feet on separate force plates mounted on a moveable platform, translated unexpectedly in one of 12 directions. Peak net torque magnitudes and latencies following perturbation onset were determined as were the relative contributions of each joint to total torque production. Magnitude of net torque generated by each leg varied by perturbation direction, with the largest individual joint magnitude elicited in directions of limb loading. Relative contributions of individual joint torques to the total response were dependent upon perturbation direction. Results suggest that a redistribution of the relative contributions of hip/trunk versus ankle strategies occurs dependent on perturbation direction, with a significant contribution by the knee joint in response to forward perturbations. Direction-specific redistribution of proximal versus distal strategies appears to depend upon the biomechanical constraints imposed by a given perturbation direction. Thus, it appears that sagittal and frontal plane postural-righting responses may not be uniquely controlled, and may instead be governed similarly, with modulation of relative torque contributions among joints when necessary, given direction-specific anatomical constraints.

Keywords Posture · Automatic postural response · Joint torque · Ankle strategy · Hip strategy

Introduction

The translating platform paradigm has been an invaluable tool in elucidating the underlying mechanisms of postural control, leading to the understanding of medium latency or automatic postural responses to unexpected perturbations (Nashner 1976, 1977). Using the translating platform paradigm, these responses, though originally thought to be immutable, have been demonstrated to be robust yet modifiable synergistic responses that are influenced by many intrinsic and extrinsic factors (Horak et al. 1997). It has been well-documented that perturbation characteristics, such as amplitude, velocity (Horak and Dener 1993; Park et al. 2004), and acceleration (Szturm and Fallang 1994), as well as subject characteristics such as prior knowledge of perturbation and central set (Horak et al. 1989) can influence automatic postural responses, demonstrating the adaptability of these responses.

Despite the focus on characterizing postural responses to the translating surface perturbation, several issues remain unresolved. One issue involves the control of posture in response to multi-directional perturbations of the support
surface, and whether differences exist between control in the frontal and sagittal planes. This is an important question given that many of the unexpected perturbations that occur in vivo involve multiple planes of movement. One viewpoint asserts that responses to anterior/posterior (AP) and lateral perturbations are controlled independently (Matjačić et al. 2001; Allum et al. 2003; Graueberg et al. 2005). Matjačić et al. (2001) argued that control in the sagittal and frontal planes is decoupled based on the observation that the net joint torque sums in the sagittal (ankle only) and frontal (ankle + hip) planes, respectively, were identical to those elicited in combination forward/backward and lateral perturbations. However, this finding does not necessarily implicate different control mechanisms between the two planes and instead may lend support to the view that a common strategy is utilized regardless of perturbation direction (Henry et al. 1998a, b). Evidence of the onset of trunk velocity in the frontal plane preceding onset in the sagittal plane, following uni- and multi-planar perturbation directions has been used to support a separate organization of balance responses between the frontal and sagittal planes (Carpenter et al. 1990; Allum et al. 2003). However, differential timing of frontal and sagittal plane trunk movements could reflect the inherent differences in skeletal geometry that serve to transfer the perturbation to the hip and trunk without the damping effect of the knee or significant movement of the ankle in the frontal plane.

A second hypothesis regarding multi-directional control is that the mechanisms for controlling posture in multiple planes are the same yet must act within biomechanical constraints of the body that differ in each direction of movement (Moore et al. 1988; Henry et al. 1998a, b). Henry et al. (1998a) suggest that the fore/aft force couple used to respond to translations in the forward/backward directions is analogous to the load/unload force couple demonstrated in response to lateral translations. The muscle synergies used to generate these reaction forces differ depending upon the direction of perturbation, but the output (surface reaction forces) is similar regardless of direction, suggesting that the strategies used by the nervous system to respond to multi-directional perturbations are similar. Park et al. (2004) determined that scaling of postural response gain occurs in proportion to the biomechanical constraints induced by the perturbation magnitude, suggesting that a single feedback control scheme is responsible for a range of responses.

To address these conflicting views, analysis of the joint torques generated by the lower limbs and trunk to respond to multi-directional perturbations (including forward, backward, and lateral directions) may lead to the determination of a global strategy or set of strategies to maintain balance. Little effort has been devoted to quantification of joint torques of the lower limbs and trunk in response to multi-directional support surface translations, having been reported only in response to backward translations that induce forward body sway (Allum and Honegger 1992; Runge et al. 1999; Botterm and Jensen 2001; Park et al. 2004) and lateral translations inducing left/right body sway (Meyer et al. 2004). In posterior translations, a continuum of responses has been identified, characterized primarily by ankle responses to lower velocity perturbations with increasing contribution of hip responses as perturbation difficulty increases, either by increased velocity or decreased support surface size (Horké and Nashner 1986; Runge et al. 1999). Meyer et al. (2004) reported a combined ankle and hip response to lateral translations driven primarily by contralateral hip abduction and contralateral ankle inversion torques. Although some element of the hip strategy is thought to contribute to the response to anterior translations (Henry et al. 1998a), this has not, to our knowledge, been substantiated through analysis of joint torques.

Therefore, the purpose of this descriptive study was to characterize the joint torques at the ankles, knees, hips, and trunk, in both the frontal and sagittal planes (knee in sagittal only), in response to unexpected translations of the support surface. We hypothesized that the magnitude of the torque responses (both spatial and temporal parameters) in the frontal and sagittal planes would be modulated based on the specific direction of perturbation.

Methods

Twenty-one healthy subjects (13 female, 8 male) were recruited from the local community (mean ± SD: age 33.0 ± 10.5 years, height 1.71 ± 0.08 m, mass 68.1 ± 12.9 kg). All subjects signed an informed consent document in accordance with University of Vermont Institutional Review Board policy and the rights of the subjects were protected.

Subjects were instructed to stand on a moveable platform, looking forward and with arms hanging comfortably at their sides. Subjects self-selected their stance width and toe out angle (facilitated by having subjects march in place until comfortable). The foot positions were traced and marked on the force platform to ensure correct repositioning of the feet following resting or stepping. All subjects were asked to maintain their balance in response to the perturbation but were intentionally not given any guidance about how to respond. Any perturbation trials in which the subjects used a stepping strategy to maintain their balance were not included in the analysis. Additional trials in the perturbation directions that elicited steps were repeated randomly following completion of the initially planned trials. Subjects were given 2–3 practice trials in each of two directions (180° directly left, 90° directly forward) prior to which they were told the direction of impending platform movement.
Following these practice trials, linear translations of the support surface in the transverse plane were presented in 12 directions of 30° increments (Fig. 1) with three trials in each direction, and two stimulus profiles. Only the responses from one with nearly constant velocity will be presented here. The platform was translated 0 cm using a ramp and hold waveform (duration of the waveform was approximately 400 ms), Fig. 1, boxed inset) with a peak velocity of 25.5 cm/s and a peak platform acceleration of 127 cm/s². Perturbation trials were randomized for both velocity and direction to ensure that subjects could not predict the perturbation type and were presented in four blocks of approximately 18 trials separated by three mandatory rest periods of approximately 5 min each. Subjects were offered additional rest periods if needed but none were required.

Subjects stood with one foot on each of two force platforms (AMTI, Watertown, MA) that were mounted within the moveable platform that was driven by electromechanical motors controlled by commercially available software (Compumotor, Parker Hannifin Corp., Rohnert Park, CA). The output of the two force platforms was sampled at 1,000 Hz and filtered with a low pass filter of 10.5 Hz. The center of pressure for each foot was calculated from the recorded forces and moments.

A three-camera, passive marker system (BTS, Milan, Italy) was used to collect three-dimensional spatial coordinate data for calculation of the body kinematics. Three non-collinear reflective markers were placed on the head, trunk, both thighs, and both shanks in order to determine joint velocities and accelerations in the frontal and sagittal planes. Two markers were placed, one on each force platform, to mark the level of the platform. Because trials in which the subject stepped were omitted from analysis, it was assumed that the feet remained stationary on the surface allowing calculation of the location of the foot center of mass in a fixed position. Kinematic data were sampled at
50 Hz and filtered using a dual pass second order Butterworth low pass filter with a cut-off frequency of 2 Hz (a cut-off frequency of 5 Hz was used for the force plate markers, which were assumed to have the greatest acceleration due to their close proximity to the translating surface). A pilot study with kinematic data collected at 100 Hz at the fast perturbation velocity (peak velocity of 43 cm/s and peak acceleration of 127 cm/s²) was used to evaluate the frequency spectrum of all markers. It was determined that 90.4–94.8% of the signal power was demonstrated at frequencies of 10 Hz or less, while 94.7–97% of the signal power was demonstrated at 25 Hz or less. Therefore, a sampling frequency of 50 Hz was deemed sufficiently accurate to capture the kinematic data of perturbations of the type presented in this report.

An eight-segment, rigid link model was constructed for each subject, with segment centers of mass (CM) determined using regression equations from the literature based on the subject’s height and mass (Zatsiorsky and Seluyarov 1983). The model represented the feet, shanks, and thighs bilaterally as well as the pelvis and trunk segments. Due to limitations in the resolution of the camera setup, markers delineating the pelvis could not be used. To compensate for this limitation, a “virtual pelvis” was computed as the line that intersected the marker over the fourth lumbar vertebra (distal trunk marker) and was perpendicular to a second line that connected the left and right proximal hip markers. The virtual pelvis was a separate segment with mass and inertial properties assigned using the above anthropometric model and could move independently from the trunk and thigh segments.

Marker position data were used to calculate joint angles, and first and second time derivatives of the joint angle data were calculated numerically with a four-point weighted least-squares approach to provide angular velocity and acceleration data. This approach fits a quadratic to four data points, which can then be used to calculate the first and second derivatives at the second point in the series. Force data were low pass filtered at 10 Hz (second order, dual pass, Butterworth filter) and re-sampled at 50 Hz in synchrony with the kinematic data. Kinematics and force data were used to compute ankle, knee (sagittal only), hip and trunk (relative to pelvis) net joint torques in the frontal and sagittal planes (SDFast, Needham, MA, USA) through inverse dynamics techniques. SDFast prescribes joint motion to follow experimentally measured trajectories (joint angular displacement, velocity, acceleration) and applies measured ground reaction forces to the feet, calculating the net “fzuer” torques necessary to generate the required motions. Knee abduction/adduction torques were not computed given the negligible motion of the knee in the frontal plane. These net joint torques represent the sum of all torques acting at a given joint, including the gravitational torque (due to the action of gravity at each segment center of mass), inertial torques (due to intersegmental interactions and linear and angular accelerations of the segments), and the muscle torque (which includes both the passive and active forces of all muscles acting about a joint as well as passive forces within the joint (Hoy and Zemnicke 1986)), and thus represent the net torque output at a given joint.

Peak torque and latency to peak torque were computed for each joint using programmable software (Matlab, Natick, MA, USA). In addition, torque integrals (defined as the area under a given torque vs. time curve) were calculated across seven 75 ms epochs from 25–550 ms after perturbation onset. All torque values were normalized to individual subject height (m) and weight (N). The absolute values of the peak torques were computed to analyze whether the torque magnitude varied with direction, regardless of whether the torque polarity was negative or positive. However, when the direction of perturbation was largely in the plane orthogonal to the plane of the torque being calculated (e.g., a 90 or 30° perturbation with sagittal plane torques, or a 90 or 120° perturbation with frontal plane torques), the polarity of the peak torque responses often varied among trials, although the timing of the peaks across trials was consistent. To address these changes in polarity, statistical comparisons of peak torques included only perturbation directions of 60, 90, 120, 240, 270, and 300° for sagittal plane torques and 0, 30, 150, 180, 210, and 330° for frontal plane torques (see Fig. 1 for a definition of perturbation directions). By limiting the directions of analysis in this way, we are confident that we compared only peak torques with the same polarity and did not equate peak torques of opposite polarities. Descriptive statistics were computed for all measures and the responses to the perturbations were analyzed using a repeated measures analysis of variance for each joint torque with direction as the repeated factor (SAS System for Windows, Cary, NC, USA). An alpha level of $P < 0.05$ was considered statistically significant for main effect comparisons. Post-hoc tests were multiple comparisons of all pair-wise differences with a Bonferroni correction to preserve the overall significant $P$-value of 0.05. Therefore, the effective significance level for post-hoc tests was set at $P < 0.0033$. Given that the Bonferroni correction is a conservative adjustment, we considered trends in post-hoc comparisons with $P$-values ranging between 0.0033 and 0.05.

Results
Multi-directional support surface perturbations elicited joint torque responses that were dependent on direction, with changes in both spatial and temporal parameters.
Spatial parameters

Frontal plane torque responses to perturbation directions with a large lateral component (150, 180, 210, 330, 0, and 30°) were characterized by ankle inversion/hip abduction in the loaded leg (side to which body sway was induced, contralateral to the perturbation) and ankle eversion/hip adduction in the unloaded leg (ipsilateral to the perturbation). In addition, contralateral lateral bending torque (to the side opposite the perturbation) was initially generated at the trunk followed by a larger ipsilateral lateral bending torque at the trunk in response to lateral perturbations (Fig. 1). Sagittal plane torque responses to predominantly forward perturbations (60, 90, 120°) were characterized by ankle dorsiflexion, knee extension, hip flexion, and trunk flexion torques, whereas responses to predominantly backward perturbations (240, 270, 300°) demonstrated ankle plantarflexion, knee flexion, and hip and trunk extension torques (Fig. 1).

In general, peak frontal torque amplitudes differed according to perturbation direction (trunk, $F_{5,55} = 3.61$, $P = 0.0051$; all other joints, $F_{5,55} = 3.89-58.55$, $P < 0.0001$; Fig. 2a–c). The loaded leg demonstrated smaller ankle torque contributions in response to combined lateral and forward perturbation directions (30° for left leg depicted in Fig. 2b, c, dark shading; 150° for the right leg, not shown) compared to the combination backward/diagonal directions (210° for the left leg, Fig. 2b, c, light shading; 330° for the right leg, not shown). This was higher for the left leg (Fig. 2b, c, light shading; 0° for the right leg, not shown), although it did not reach significance for the right ankle. Trunk torque differed significantly only between directions 30 and 150° ($P = 0.0031$).

Peak sagittal torque amplitudes for the lower extremity joints varied significantly across directions ($F_{5,71} = 3.13-42.23$, $P$-values ranged from $P < 0.0001$ to $P = 0.013$; Fig. 3) and tended to be greater in diagonal perturbations (210° for the left leg, Fig. 2b, c, left leg, right leg, Fig. 2b, c, light shading; 0° for the right leg, not shown). Although this did not reach significance for the right ankle. Trunk torque differed significantly only between directions 30 and 150° ($P = 0.0031$).

Fig. 2 Influence of perturbation direction on joint torque responses in the frontal plane. Group means (±SD) of the absolute values of normalized peak torque magnitude for the trunk (a) and left lower limb joints (b hip, c ankle) in the frontal plane. Analyses of perturbation directions for frontal plane torques included 0, 30, 150, 180, 210, and 330°. Areas of light gray shading represent directions that result in unloading of the left lower limb, while areas of dark gray shading represent directions that result in loading of the left lower limb due to induced body sway. Inset diagrams depict the actual normalized peak torque group averages across all perturbation directions, with polarity included. Significant main effects for direction (*) are indicated by superscripts listed adjacent to the joint of interest.

towards perturbations (270°) were greater than all other perturbation directions ($P$-values ranged from 0.0003 to 0.044).

Temporal parameters

In response to lateral perturbations, the latency of peak torque production varied between the directions that resulted in leg loading versus leg unloading. In general, the hip abductor/hip extensor peak torque responses occurred later...
in directions where the leg was loaded than those in which the leg was unloaded (left hip $F_{5.84} = 7.27$, $P < 0.0001$, Fig. 4a; right hip $F_{3.8} = 0.78$, $P < 0.0001$, not shown). Conversely, at the ankle, eversion/inversion peak torque responses tended to occur earlier in the loaded leg than the unloaded leg (left ankle $F_{5.85} = 6.47$, $P < 0.0001$, Fig. 4a; right ankle $F_{5.85} = 11.56$, $P < 0.0001$, not shown), with the exception of the latencies to 30° perturbations in the left leg which occurred at approximately the same time as those of the unloaded leg. In contrast, responses to predominantly forward or backward perturbation directions elicited fewer differences in peak torque latencies. Peak latencies in the sagittal plane torque responses did not demonstrate a direction effect at the distal joints (left ankle $F_{5.81} = 1.86$, $P = 0.11$; left knee $F_{5.82} = 1.69$, $P = 0.15$, Fig. 4b); however, at the proximal joints, direction effects were demonstrated. Hip torque production peaked earlier in the forward directions (60, 90, 120°) compared to backward (left hip $F_{5.86} = 2.92$, $P = 0.018$, Fig. 4b; with the exception of 240° which was more similar to the forward directions in time) while the trunk demonstrated earlier peak latencies in the backward directions (240, 270, 300°) relative to forward (trunk $F_{5.77} = 3.29$, $P = 0.0096$, Fig. 4b; only 90° perturbations reached significance).

Pattern of torque development over time

In order to document the pattern of joint torque responses to multi-directional perturbations, the relative magnitude of joint torque integrals across 75 ms intervals spanning 25-550 ms following perturbation onset were determined (Fig. 5). In lateral perturbation directions, the earliest response (between 25 and 175 ms post-perturbation) was initiated largely by unloaded hip abduction (ipsilateral to the perturbation), with some initial contribution of lateral trunk bending contralateral to the perturbation (Fig. 5a). The largest contributor to the equilibrium response between 175 and 400 ms following perturbation onset was loaded hip abduction, with concurrent increases in loaded ankle inversion torque, both contralateral to the direction of perturbation. In the later phases of the posture-righting response (400-550 ms), the trunk, loaded hip, and loaded ankle continued to dominate, contributing ipsilateral side bending, abduction and inversion torques, respectively. The unloaded ankle demonstrated fairly constant eversion torque throughout the movement response.

In forward perturbation directions, the initial response was dominated by early trunk extension torque (from 25 to 175 ms post-perturbation), with development of a large ankle dorsiflexion torque between 175 and 400 ms following perturbation onset (Fig. 5b). Knee extension and hip flexion torques began to increase from 175 ms after
Fig. 4 Influence of perturbation direction on peak torque latencies in the frontal (a) and sagittal (b) planes. Group means (±SD) of the peak torque latencies are plotted for the frontal plane trunk, left hip, and ankle joints (a) and the sagittal plane trunk, left hip, knee and ankle joints (b). Significant main effects for direction (*) are indicated by superscripts listed adjacent to the joints of interest. Areas of light gray shading represent perturbation directions that result in unloading of the lower limb, whereas areas of dark gray shading represent directions that result in loading of the lower limb due to induced body sway. Unshaded areas represent purely anterior or posterior perturbations that result in symmetrical loading between the two lower limbs (sagittal plane only).

perturbation, the knee providing a particularly large contribution from 250 to 550 ms post-perturbation. Trunk flexion torque developed late (from 400 to 550 ms post-perturbation) and likely did not provide substantial contribution to recovery of balance given the timing of torque development (after platform deceleration) and its percentage relative contribution to the total torque response summed across joints (Fig. 5b, inset). In contrast, the early response to backward perturbations was dominated by hip flexion and knee extension torque (between 25 and 175 ms post-perturbation), followed by a large ankle plantarflexor torque (Fig. 5c). Ankle plantarflexor torque remained the dominant torque contribution throughout the movement response (175–550 ms post-perturbation) although some knee flexion, hip extension, and trunk extension torques increased slightly in the later portion of the movement response (250–550 ms and 325–550 ms after perturbation onset for the knee and hip/trunk, respectively).

Discussion

The present study described the net joint torque output of the trunk, hips, knees, and ankles in response to multidirectional support surface translations. Individual leg contributions varied with direction, with the largest torque
Fig. 5 Group means of the contributions of lower limb and trunk torques (shaded according to panel legends) to the summed, normalized torque responses across joints, calculated by quantifying torque integrals across seven 75 ms epochs from 2.5-350 ms after perturbation onset. Frontal plane torques are presented in response to purely lateral perturbations (in this case, 180°), a) whereas sagittal plane torques are presented in response to purely forward (000), b) and backward (270°, c) perturbations. Polarity of the torque responses for each joint (e.g., Ext vs. Flex) are listed in the epoch of greatest total torque production and extend across epochs unless otherwise indicated by bi-directional arrows across epochs where the opposite polarity was elicited. For example, in lateral perturbations (180°, a), the polarity of trunk torque is left side bending (L) from 250-350 ms following perturbation onset (as indicated by the bar during the 250-400 ms epoch) and right side bending (R bend) between 25 and 250 ms following perturbation onset (as indicated by the bi-directional arrows spanning the three epochs between 25 and 250 ms). Inset pies depict the relative contribution (%) of each joint to the summed torque response for each of the seven epochs, with no reference to torque polarity. Flx flexion, Ev extension, DF dorsiflexion, PF plantarflexion, L left side bending, Add adduction, Ev eversion, Abd abduction, Inv inversion

Strategy selection: sagittal plane responses

Directions of maximum torque contribution for the lower limbs occurred in diagonal perturbations (i.e., those combining forward or backward and lateral components; Fig. 3b, c, d). Although it has been suggested that the off-axis perturbation directions that elicited maximal responses represent a “directional sensitivity” of muscle and torque responses (Allum et al. 2003; Gruneberg et al. 2005), it is plausible that these responses (which may include both stretch reflex and balance-correcting responses) are a direct consequence of the perturbation-induced body sway that results in asymmetric loading between the lower limbs (Fig 3b, c, d). Therefore, it is possible that the directional variation in sagittal plane response magnitudes often attributed to specific lines of muscle action, insertion points or joint axes (Henry et al. 1998b; Carpenter et al. 1999) may instead reflect increased torque production and presumably, muscle force, in response to asymmetric limb loading due to induced body sway. Asymmetric loading in this manner may not only apply to the directions of body sway (i.e., inducing limb loading, muscles primarily active as extensors; but also directions of unloading for muscles that are primarily active as flexors. Jacobs and Macpherson (1996) reported directions of maximal activity for both flexor and extensor muscle groups that corresponded to directions of maximal loading (for extensors) or maximal unloading (for flexors) due to the direction of body sway following perturbation. These directions represent multi-directional perturbations and require asymmetric muscle activation regardless of the specific role of the muscle (i.e., extensor vs. flexor) in the corrective response.

Sagittal plane responses to forward and backward surface translations demonstrated patterns that may be
viewed as similar when considered in context of the assumed biomechanic constraints influencing postural responses dependent on the direction of perturbation. An early contribution of the trunk and/or hip was demonstrated (Fig. 5b, c) with the trunk providing a large contribution to the torque responses following forward perturbations and the hip providing a greater proportion of the response to backwards perturbations. It is possible that since hip extension is limited to only 20° (Reese and Bandy 2002) when the body sways posteriory (forward perturbation, 90°), the trunk may provide a larger contribution to the response in what could be termed a “reverse hip strategy”, although with a significant contribution of the knee, consistent with the findings of Henry et al. (1998a). The knee extension torque elicited following forward perturbations would likely aid transfer of energy from the distal segments to the trunk and pelvis, which would act to dampen the effects of the perturbation, due to their inertia (Hall and Jensen 2002). In the opposite perturbation direction (backward perturbation, 270°), the hip demonstrates an early flexion response characteristic of a “hip strategy”, with limited contribution of the trunk, which may reflect the position of the hip as the first link in the chain from distal to proximal joints that can exert a significant direct effect on the large trunk/pelvis complex. Further study is needed to evaluate whether these assumed mechanical limitations govern the pattern of torque production elicited.

The role of the knee has been largely ignored in response to surface translations with most assuming minimal contributions to the posture-righting response, particularly in slower perturbations (Matjasic et al. 2001; Park et al. 2004). Park et al. (2004) suggested that while inclusion of the knee into postural models enhanced the ability of the model to predict experimental data, it was deemed too computationally complex to include for the reduction in error gained. Knee flexion torques similar to those elicited in the current study have been reported following backward surface translations, both experimentally (Runge et al. 1999) and theoretically (Allum and Honegger 1992), although the relative contributions of this joint to the posture-righting responses were not directly compared to the other joints. Hall and Jensen (2002) reported significant contributions of the knee to the recovery of balance following forward perturbations, demonstrating the contribution of the knee to energy absorption used to minimize destabilization of the trunk. Our findings support an increased role for the knee in response to both forward and backward perturbations, such that the relative contribution of the knee to the corrective response parallels and may exceed that of the hip, especially in response to forward perturbations (Fig. 5b, c).

Strategy selection: frontal plane responses

Responses to predominantly lateral perturbations were controlled using the load/unload strategy described previously (Winter et al. 1993; Rietdyk et al. 1999; Henry et al. 2001) as demonstrated by increased torque production by the lower limbs in the direction of lateral body sway, and the decreased torque on the opposite side (Fig. 2b, c). Consistent with the arguments above describing the need for increased torque in directions of limb loading, the directions of purely lateral body sway (rightward, 0° and leftward, 180°) demonstrated increased frontal plane torque production, particularly at the hip and trunk (Fig. 2). Balance was recovered primarily through the use of contralateral hip abduction, ankle inversion, and ipsilateral trunk lateral bending torques (Fig. 5a), consistent with previous findings (Rietdyk et al. 1999; Meyer et al. 2004). However, a small but not insignificant proportion of the response was initiated by ipsilateral hip abduction and contralateral trunk lateral bending torques, a finding that has not previously been reported (Fig. 5a). Although these contributions, occurring between 25 and 175 ms post-perturbation, are small relative to those produced later in the response, the fact that they are maintained and are the dominant corrective torques during this period suggests that they may represent some combination of passive and active responses. Sagittal knee torque contributions to lateral perturbation directions could not be compared statistically (see “Methods” for explanation) and were small relative to the frontal plane corrective torques (inset of Fig. 5c, directions 0° and 180°). However, it is likely that the bilateral sagittal knee torques (of differing magnitudes or polarities) may have provided some additional contribution to the load/unload strategy of balance recovery following lateral perturbations. The early contralateral trunk lateral bending torque could represent initiation of what has been termed a “hip strategy” (Horak and Nashner 1986; Kujo and Zajac 1993; Runge et al. 1999) albeit in the frontal plane, reflecting the bending at the hip and trunk in the direction of body sway that aids to reduce the moment of inertia of the body to quickly reverse the direction of body CM motion (Kujo and Zajac 1993). Early ipsilateral hip abduction torque could be the result of muscle spindle discharge caused by sway-induced hip abduction, a motion that could act to facilitate the hip strategy in combination with the demonstrated trunk torque.

Control of automatic postural responses: sagittal versus frontal planes

In contrast to the peak latencies in the sagittal plane, which were independent of direction at the distal joints, frontal
plane torque latencies demonstrated modulation with perturbation direction at all joints (Fig. 4). The explanation for this observation could be three-fold. First, it is possible that control in the frontal plane is more highly regulated than the sagittal plane, due to the fact that loss of balance to the lateral side does not easily lend itself to a stepping strategy (Winter et al. 1996; Zettl et al. 2002). Alternatively, it may be that the perturbation velocity used in this study is less difficult to respond to in the frontal plane given the stability provided by the relatively wide base of support compared to the base of support in the sagittal plane that is dependent on foot length. Third, it is also plausible that the added passive contribution to lateral perturbations (Reddy et al. 1999; Henry et al. 2001), due to the relatively wide base of support in this plane (heel to heel distance in the current study, mean ± SD: 21.5 ± 6.6 cm), could diminish the need for a substantial active response by allowing the geometry of the skeletal system in the frontal plane to dictate the response.

Although it has been suggested that postural control in the sagittal versus frontal planes is de-coupled (Matiac et al. 2001; Gruneberg et al. 2005), we do not find compelling evidence to support this assertion; nor can we prove definitively that a single control schema is used to control both frontal and sagittal planes. However, our findings point to similarities in control across directions, in both the frontal and sagittal planes. While direction-specific differences in the elicited responses were demonstrated, they can be explained by a simple redistribution of distal and proximal strategies required by the biomechanical constraints influencing postural responses for each direction of perturbation. Both the hip/trunk and ankle strategies emerge in response to all directions of perturbation with the addition of the knee when it is most mechanically advantageous to contribute to the response (i.e., can provide substantial extensor torque in forward perturbations). In fact, it may be that the responses are ordered such that the most distal joint that is best-suited to respond to a given direction of perturbation (given anatomical limitations of joint ranges of motion; Reese and Bandy 2002) will provide the largest contribution to the postural response (Fig. 5). For example, in a forward perturbation (0°), the ankle is somewhat limited in its ability to generate sufficient dorsiflexor torque to resist backward body sway, requiring the addition of significant knee torque to prevent balance loss. In response to a backwards perturbation (270°), the ankle is well-suited to resist forward body sway due to the long lever arm of the foot in this direction; thus, the relative contribution of the knee to the posture-righting response is reduced and that of the ankle is enhanced. In addition, the hip and trunk demonstrate early contributions to the postural responses in all directions of perturbations, suggesting that regardless of the perturbation direction, an early proximal strategy may be used to rapidly initiate body CM movement over the base of support. Therefore, the direction-specific torque magnitudes elicited by multi-directional perturbations represent a range of responses generated through a relative redistribution of proximal and distal strategies based on the unique set of constraints imposed by skeletal anatomy in response to each direction of perturbation.

Conclusions

In conclusion, these findings emphasize the importance of considering trunk torques independently from those torques generated across the hip joint, given that in all perturbation directions, the trunk demonstrates an independent and significant contribution to the posture-righting response. Redistribution of the hip/trunk and ankle strategies appears to occur when biomechanical constraints render one joint less able to contribute to the postural response. The knee has been overlooked as a potential contributor to the automatic postural response, and appears particularly important in response to forward perturbations, where both the ankle and hip joints are limited in their ability to resist backward sway. Postural responses do not appear to be controlled separately in the sagittal and frontal planes, given that the responses to all perturbation directions are accomplished with an early contribution of the hip and trunk joints and significant corrective torque generated by the joints that are mechanically best-suited to respond, given direction-specific biomechanical constraints.

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