Alterations and Asymmetries in Trunk Mechanics and Neuromuscular Control among Persons with Lower-Limb Amputation: Exploring Potential Pathways of Low Back Pain

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ABSTRACT

Low back pain (LBP) is a substantial secondary disability among persons with lower-limb amputation (LLA). Abnormal mechanics of movement subsequent to LLA may increase the stability demands on the spinal column, and repetitive exposures to such abnormal movements may alter trunk passive properties and/or the coordination of surrounding trunk muscle responses. Further, preferential use of the sound limb may lead to asymmetries in these behaviors. Spine biomechanics (e.g., loading and stability) are substantially influenced by trunk passive properties and neuromuscular control, and alterations in these behaviors are associated with abnormal mechanics of the spinal column and an increased LBP risk. However, there is limited evidence regarding whether prolonged repeated exposures to abnormal gait and movement resulting from LLA and subsequent repeated use of a prosthetic device affect these trunk behaviors.

Eight males with unilateral LLA and a matched sample of non-amputation controls completed three studies in which several measures of trunk passive properties, neuromuscular control, and spine biomechanics were quantified using laboratory experiments and biomechanical analyses. Each study involved a distinct task to investigate potential alterations and/or asymmetries in trunk passive properties and neuromuscular control. The first study used a seated balance task to assess trunk postural control and stability. The second study used multidirectional trunk perturbations to assess trunk mechanical and neuromuscular behaviors. Finally, the third study

used controlled quasi-static trunk movements to assess load-sharing mechanisms between active and passive low back tissues.

Significant alterations and asymmetries in trunk passive properties and trunk neuromuscular responses were present among participants with LLA, specifically reduced and asymmetric trunk stiffness and reflex response; decreased and asymmetric passive contributions to trunk movements; and increased trunk muscle activities. Significant increases in trunk postural sway and trunk muscle activities were also present during seated stability measures. Such alterations in these behaviors may be a result of repetitive exposures to abnormal gait and movement subsequent to LLA and the use of a prosthetic device, and could play a contributing role in the development of LBP in this population. Future work should investigate the temporal relationship between altered trunk behaviors and repeated exposure to abnormal gait and movement subsequent to LLA, to better identify critical years for rehabilitation and preventative care.

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Chapter 1: Background and Motivation

1.1 Low Back Pain: General Population

Low back pain (LBP) is the most prevalent source of musculoskeletal disability in the United States and has been cited as one of the most common reasons to visit a physician (Deyo and Phillips, 1996; Katz, 2002; Liliedahl et al., 2010). It is estimated that nearly 80% of the population will experience LBP at some point in their lives (Manek and MacGregor, 2005; Roffey et al., 2010), many of whom will continue to report symptoms and/or disability after one year or more (Croft et al., 1998; Enthoven et al., 2004). As a result, the annual direct and indirect costs associated with LBP have been estimated at a substantial $19.6-$118.8 billion and have continued to rise over time (Dagenais et al., 2008; Frymoyer and Cats-Baril, 1991; Luo et al., 2004). Despite the high prevalence and economic impact of the disorder, the exact etiology of LBP remains unknown. However, mechanical risk factors appear to play a more important role in certain populations.

1.2 Low Back Pain: Persons with Lower-Limb Amputation

The prevalence of LBP is substantially higher among persons with lower-limb amputation (LLA; 52-71%) compared to the general population (6-33%; (Ehde et al., 2001; Smith et al., 1999). A cross-sectional survey of over 250 persons with LLA revealed that 52% experienced at least one back pain episode in the 4 weeks prior to the completion of the study, 25% described their LBP as constant, and 31% described their pain as severe (>7 on a 0-10 scale; Ehde et al., 2001). Evidence also suggests that LBP may even be more bothersome than residual limb and phantom pain, two sources of pain commonly thought of as the major causes of post-amputation morbidity (Ehde et al., 2001; Smith et al., 1999). The frequency and severity of LBP

in persons with LLA have been well documented, yet the underlying causes in this population remain unknown. Given the high prevalence and debilitating nature of LBP among persons with LLA, it is important to understand the mechanisms of LBP onset and recurrence in this population.

1.3 Spine Biomechanics

Abnormal mechanics of the spinal column, particularly higher loads and lower stability, are considered one of the predominant causes of LBP (Cholewicki and McGill, 1996; Panjabi, 2003; Radebold et al., 2000). An important mechanical function of the lumbar spine is to support the upper body while distributing loads during movement. To avoid pain or injury, mechanical equilibrium and stability need to be maintained throughout wide ranges of external loads and displacements while keeping internal spinal loads within physiologic tolerances (Panjabi, 1992b). The properties of trunk passive spinal tissues/structures and neuromuscular responses from surrounding musculature play an integral role in the maintenance of spine biomechanics (e.g., loading and stability). The neuromuscular system supports the passive structures in the maintenance of postural stability, for example, by altering muscle responses based on sensory orientation cues and external demands (Peterka, 2003). Therefore, preserving an effective synergy between trunk passive spinal structures and active neuromuscular responses is important for maintaining spinal stability and controlling spinal loads.

1.3.1 Trunk Passive Properties

The passive lumbar spine is composed of five vertebrae, facet articulations, intervertebral discs, spinal ligaments, and any passive mechanical properties of muscles. These structures collectively distribute forces and displacements in response to external loads and postural

movements. However, the load-carrying capacity of the passive spine is low around neutral postures and therefore fundamentally unstable (Panjabi, 1992b). *In vitro c*adaver studies demonstrate that an isolated passive lumbar/thoracolumbar spine can buckle under compressive loads as low as 90N/20N, respectively (Crisco et al., 1992), yet the passive spine is capable of providing substantial resistance to prevent damaging motions beyond the range of physiologic movement. This capacity is a result of the non-linearity of the load-displacement behavior of the passive structures (Edwards et al., 1987; Solomonow, 2009).

Preserving an effective synergy between trunk passive spinal structures and active neuromuscular responses is important for maintaining spinal stability and controlling spinal loads. While lumbar range-of-motion (ROM) can be used to assess passive spinal function and mechanical properties of the spine, the “flexion-relaxation phenomenon” has long been used as an additional means to assess the synergy of voluntary muscles activations and passive structures of the low back (McGill and Kippers, 1994; Schultz et al., 1985). With forward trunk flexion, myoelectric silence occurs in lumbar extensor muscles as the external gravitational moment is nearly fully supported by spinal ligaments, intervertebral discs, and passive components of muscle-tendon units (Adams et al., 1980; McGill and Kippers, 1994). The resistance to forward-flexion moments can vary dependent on the mechanical properties of passive tissues (Adams and Dolan, 1996; Gunning et al., 2001), which thereby alter the active-passive load sharing behavior of the passive spine and surrounding musculature. Substantial decreases in passive contributions to active-passive load sharing behavior may lead to an inability to turn off the active musculature, increasing loads distributed to the spine. Laxity in the passive spinal tissues and reduced resistance to flexion moments have also been related to spinal instability (Olson et al., 2004).

Spinal ligaments can also serve in a feedback role, providing information about the position and motions of the spinal segments (Panjabi, 1992a). A ligament-muscle reflex loop exists whereby mechanical stimuli applied to ligaments can either increase activity of surrounding musculature to help stabilize a joint or reduce loads on a ligament, or inhibit the activity of muscles that are acting to destabilize the joint (Solomonow, 2009). However, increased laxity in spinal ligaments can lead to reductions in the sensitivity of the stretch receptors and inhibit reflexive activations. The threshold level at which normal mechanoreceptors would be stimulated is likely to be increased in the presence of ligament laxity, leading to inhibition of the erector spinae or other posterior musculature (Claude et al., 2003). The mechanical properties of the passive spine are important for maintaining proper load distribution among passive structures to assure no neurologic deficits, major deformities, or pain occur within the spine, as well as preserving the synergy between the passive structures and the active neuromuscular system.

1.3.2 Trunk Neuromuscular Control

The active neuromuscular system represents both voluntary and reflexive control of skeletal muscles. Trunk muscles can be voluntarily activated to control vertebral motions and balance opposing muscle forces or external loads and moments placed on the spinal column. As previously mentioned, the passive spinal column is inherently unstable without responses from the active neuromuscular system. Therefore, muscle force magnitude and recruitment patterns are important for maintaining mechanical equilibrium and stability of the spine.

Active trunk muscle forces provide additional mechanical stability to the spinal column (Bergmark, 1989; Brown and McGill, 2009; Moorhouse and Granata, 2005; Panjabi, 2003). During active exertions, trunk stiffness is increased proportionally with steady-state coactivation and is believed to contribute to more trunk stability (Gardner-Morse and Stokes, 2001;

Moorhouse and Granata, 2007). However, coactivation of trunk flexor and extensor muscles, which has been shown to occur during various static and dynamic exertions, can increase compressive and shear loads on the spine by 45% and 70%, respectively (Granata and Marras, 1995; Granata et al., 1999; Hughes et al., 1995). Consequently, as additional active trunk muscle stiffness increases spinal stability (Gardner-Morse and Stokes, 1998; Granata and Marras, 1995; Thelen et al., 1995), it also paradoxically increases spinal loads and metabolic energy consumption (Hatze, 1975). Large muscle forces may also overload passive spinal structures (e.g., discs, ligaments, and tendons), and lead to errors in proprioceptive feedback (Panjabi, 1992a; Solomonow, 2009). Further, asymmetric muscle stiffness could act to destabilize the spine. Increases in voluntary muscle activations are therefore not the only appropriate contribution to spinal stability by the neuromuscular system.

Involuntary muscle reflexes complement active muscle stiffness and passive contributions in the maintenance of spinal stability. A recent study suggests that involuntary reflexes may account for up to 42% of the total stabilizing dynamics of the torso (Moorhouse and Granata, 2007). Perturbations applied to the trunk can result in the excitation of large mechanoreceptors within spinal ligaments to initiate reflexive responses of paraspinal muscles (Simon, 1994). These reflexive responses are more efficient than voluntary coactivation because they do not require steady-state energy consumption, but rather only brief muscle activation. The magnitude of the reflexive response (reflex gain) has been shown to be proportional to the pre-existing levels of voluntary activation (Moorhouse and Granata, 2007), and increases in reflex gain have been shown to decrease the amount of metabolic power required for maintenance of spinal stability (Franklin and Granata, 2007). However, the response is time-delayed by the latency of the reflex loop (Granata et al., 2004). Increases in the reflex latency can therefore adversely affect spinal stability.

1.4 Altered Trunk Behaviors and Low Back Pain

Alterations in trunk passive properties and/or inefficiencies in neuromuscular control are associated with abnormal mechanics of the spinal column, and have been identified in patients with LBP. Specifically, patients with LBP have altered passive mechanical properties (Hodges et al., 2009), reduced lumbar range of motion (Geisser et al., 2004), and the absence of muscle relaxation at full trunk flexion (Shirado et al., 1995; Watson et al., 1997). Further, LBP patients have increased muscle activity prior to external perturbations (Stokes et al., 2006) and altered recruitment patterns (Radebold et al., 2000), as well as delayed reflex responses (Radebold et al., 2001). Numerous experimental studies have similarly demonstrated disturbances in trunk behaviors of healthy persons following acute exposure to physical LBP risk factors, and epidemiologic evidence suggests an association between these risk factors and the occurrence of LBP (Hoogendoorn et al., 2000). Prolonged static or dynamic flexion reduces passive support of the spine (Hendershot et al., 2011; McGill and Brown, 1992; Olson et al., 2004; Shin and Mirka, 2007), repetitive lifting alters muscle recruitment patterns (Dickey et al., 2002), and whole-body vibration exposure can inhibit muscle response (Roll et al., 1980). Animal and human models also indicate alterations in the reflexive control of muscle responses following exposure to static and cyclic flexion (Hendershot et al., 2011; Rogers and Granata, 2006; Solomonow et al., 2003).

Mechanical and/or sensory deficits of the spine can lead to instability and pain. Abnormal motion, increased external loads or internal muscle forces, vibrations, repetitive loading, or any combination of these may lead to instability of the spinal column. These mechanical stimuli can also lead to local material and structural alterations that cause inflammation or stimulation of embedded pain receptors, which are sensitive to pressure and deformation, within the spinal column and surrounding musculature. Evidence also suggests that increased laxity in the

passive spine, caused by any of the aforementioned stimuli, can lead to a decreased stretch receptor reflex response (Solomonow, 2009). A less stable spine is thus more susceptible to larger-than-normal spinal motions or increased loading from active muscles to compensate for losses in passive contributions. Despite the well-documented alterations in trunk behavior (both due to exposure to risk factors and in response to LBP), is it presently unclear whether such changes in trunk behaviors are causes or effects of LBP (Roffey et al., 2010; Wai et al., 2010). However, chronic exposure to abnormal mechanics appears to play an important causal role in persons with LLA.

1.5 Abnormal Mechanics of Locomotion among Persons with Lower-Limb Amputation

Exposure to repetitive or cumulative trauma may play an important causal role in the development of LBP. Alterations in amputee gait and locomotion have been identified. Exposures to such alterations are thought to cause important comorbidities, including pain at other joints and osteoporosis (Norvell et al., 2005). Recent biomechanical evidence suggests an increased association between abnormal spine mechanics and LBP during gait among persons with LLA (e.g., Goujon-Pillet et al., 2008; Morgenroth et al., 2010). Specifically, alterations in tri-planar lumbar kinematics were found among persons with LLA compared to healthy controls, and are thought to be one of the reasons for LBP development in this population. Persons with LLA also appear to have substantial deficits in low-back extensor muscle strength and endurance (Friel et al., 2005). It is therefore theorized that prolonged or chronic exposure to abnormal and asymmetric gait and movement caused by LLA may lead to alterations in trunk behaviors and spine biomechanics, and thus the high prevalence of LBP in this population. Spine biomechanics are substantially influenced by trunk passive properties and neuromuscular control. Yet, the effects of LLA on trunk passive properties and surrounding musculature remain unknown. Therefore, a casual pathway between lower-limb amputation

and LBP will be explored. Central to this pathway is that coping with altered gait/mechanics can chronically disturb trunk passive properties and neuromuscular control, which in turn compromise spine biomechanics, leading to LBP.

Viscoelastic tissues of the passive spine can develop altered mechanical properties in response to loading patterns/history (Adams and Dolan, 1996; Gunning et al., 2001). While acute mechanical loading of spinal structures beyond their physiologic tolerances can lead to injury or pain, repeated sub-tolerance loads that accumulate over time can also lead to tissue fatigue and a reduction in failure tolerances (McGill, 1997). Creep of spinal ligaments in response to strains well below physiologic thresholds has been shown to cause microdamage in the collagen within ligaments and a degradation of its functional properties (i.e., the stress-strain relationship; Claude et al., 2003). Thousands of these small-load cycles occur every day, during normal walking for example, without insult in the healthy population. However, substantial alterations in gait and body mechanics, subsequent to LLA and repeated used of a prosthetic device, may result in altered loading histories (e.g., rates and magnitudes) in this population, thus compromising the ability of the passive spine and active neuromuscular responses to maintain effective load distribution, mechanical equilibrium, and stability.

Locomotion is more difficult for persons with LLA. For example, persons with LLA expend as much as 25-65% more metabolic energy than able-bodied individuals during gait and movement (Hoffman et al., 1997; Schmalz et al., 2002). Typically, vertical and lateral displacements of the body's center of mass are limited to reduce energy expenditure during gait (Kuo, 2007), yet notable alterations and asymmetries in gait mechanics among persons with LLA have been identified (Winter and Sienko, 1988). Several specific differences include weight-bearing (Mâaref et al., 2010; Nolan et al., 2003; Vrieling et al., 2008a) and stride-length asymmetries (Isakov et al., 1997; Isakov et al., 2000), increased joint kinetics (Sanderson and Martin, 1996)

and joint kinematics (Bateni and Olney, 2002), and increased postural sway (Buckley et al., 2002). Recent evidence also indicates alterations in the biomechanics during common tasks such as turning (Segal et al., 2011) or sit-to-stand transfers (Burger et al., 2005), and decreases in total-body balance and stability (Kendell et al., 2010; Vrieling et al., 2008b).

Detrimental alterations in trunk passive properties and neuromuscular control may be present among persons with LLA as a result of exposure to abnormal mechanics, and thereby contributing to the high prevalence of LBP in this population. Similarly, the equilibrium and stability demands of the spine may be increased during the exposure to abnormal mechanics during locomotion. However, there is very limited evidence regarding whether prolonged exposure to abnormal mechanics resulting from lower-limb amputation affect these trunk behaviors. It was therefore *hypothesized* that persons with LLA have altered trunk passive properties and neuromuscular control compared to healthy controls as a result of this chronic exposure. In addition, persons with LLA were expected to have asymmetries in trunk passive properties and neuromuscular control due to the inherent asymmetry in the amputation itself. These differences in trunk behaviors were in turn expected to compromise spine biomechanics (e.g., loading and stability) and thus provide support for a causal pathway leading to the high prevalence of LBP in this population.

1.6 Document Organization

Three laboratory studies were completed to address these hypotheses. The first study used a seated balance task to assess trunk postural control and stability. The second study used multidirectional trunk perturbations to assess trunk mechanical and neuromuscular behaviors. Finally, the third study used controlled quasi-static trunk movements to assess load sharing mechanisms between active and passive tissues of the low back.

This dissertation is organized such that one chapter describes each study. Chapter 2 describes trunk stability among persons with LLA during a seated balance task, Chapter 3 describes trunk mechanical and neuromuscular behaviors among persons with LLA using multidirectional trunk perturbations, and Chapter 4 describes load-sharing mechanisms between active and passive tissues of the low back during controlled flexion-extension movements. A summary of these studies is included in the final chapter (5), and also discusses their implications and suggestions for future research.

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Chapter 2: Persons with Lower-Limb Amputation have Impaired Trunk Postural Control While Maintaining Seated Balance

2.1 Abstract

Abnormal mechanics of movement resulting from lower-limb amputation (LLA) may increase stability demands on the spinal column and/or alter existing postural control mechanisms and neuromuscular responses. A seated balance task was used to investigate the effects of LLA on trunk postural control and stability, among eight males with unilateral LLA (4 trans-tibial, 4 trans-femoral), and eight healthy, non-amputation controls (matched by age, height, and weight). Both traditional measures derived from center of pressure (COP) time series, and measures obtained from non-linear stabilogram diffusion analyses, were used to characterize trunk postural control. All traditional measures of postural control (95% ellipse area, RMS distance, and mean velocity) were significantly larger among participants with LLA. Non-linear stabilogram diffusion analyses also revealed significant differences in postural control among persons with LLA, but only in the antero-posterior direction. Normalized trunk muscle activity was also larger among participants with LLA. Larger COP-based sway measures among participants with LLA during seated balance suggest an association between LLA and reduced trunk postural control. Reductions in postural control and spinal stability may be a result of adaptations in functional tissue properties and/or neuromuscular responses caused by repetitive exposure to abnormal gait and locomotion, which could lead to an increased risk for spinal instability, intervertebral motions beyond physiological limits, and pain.

Keywords: Amputation; Lower-Extremity; Postural Control; Low Back Pain; Biomechanics

2.2 Introduction

Low back pain (LBP) represents a substantial secondary disability among persons with lower-limb amputation (LLA; Gailey et al., 2008). Although some controversy remains regarding the exact definition, spinal instability is considered one of the predominant causes of LBP (Panjabi, 2003), since reductions in the control of spinal stability could lead to increased trunk displacement (increased tissue strains) and trunk moment (increased spinal loads). Experimental studies have demonstrated notable alterations and asymmetries in gait following LLA, as well as in the mechanics of movement during common daily tasks (Sagawa Jr et al., 2011). Such alterations have been associated with increased three-dimensional trunk kinematics compared to able-bodied controls (e.g., Goujon-Pillet et al., 2008), suggesting that abnormal mechanics of movement resulting from lower-limb amputation may increase the stability demands of the spinal column and/or alter existing postural control mechanisms and neuromuscular responses. Despite these alterations and adaptations in gait and movement, the effects of lower-limb amputation, and the subsequent use of a prosthetic device, on trunk postural control and spinal stability are not well understood.

Maintenance of spinal stability requires efficient and synergistic responses from passive structures and active neuromuscular control (Panjabi, 1992a). The aforementioned alterations in gait and movement among persons with LLA may result in new spinal loading patterns (e.g., rates and magnitudes), which over time could alter motor control strategies and functional properties of the passive spine. Numerous studies have demonstrated disturbances in passive and active trunk behaviors of healthy persons following acute exposure to mechanical loading and atypical postures, suggesting an association between altered trunk behaviors, reduced postural control, and the occurrence of LBP. For example, prolonged static or dynamic trunk flexion reduces passive support of the spine (McGill and Brown, 1992), repetitive lifting alters

trunk muscle recruitment patterns (Dickey et al., 2002), and whole-body vibration exposure can alter trunk proprioception (Li et al., 2008). Mechanical or sensory deficits in passive tissues can also lead to decreased muscle force output, reduced proprioception, and inhibition of stretch reflexes (Solomonow, 2009). Thus, repeated exposures to abnormal mechanics of motion could lead to similar, but chronic, alterations in trunk behaviors, and subsequent reductions in trunk postural control and spinal stability among persons with LLA.

Measures of the center of pressure (COP) during seated balance have been used to assess trunk postural control in both healthy individuals and LBP patients, and impaired trunk postural control has been associated with spinal instability and LBP (e.g., Radebold et al., 2001). While deficits in whole-body postural control during quiet standing have been associated with LLA (Rougier and Bergeau, 2009), these alterations are likely a result of lost musculature in the lower limb (e.g., at the ankle joint). During quiet upright stance, postural adjustments can be made using a variety of responses through the ankle, knee, hip, and lumbosacral joints. It was therefore anticipated that performing a task not requiring the lower limbs, where inherent differences and asymmetries are present among persons with LLA, a better understanding of the effects of LLA on trunk postural control and spinal stability could be obtained. Therefore, the goal of the present work was to use a seated balance task to investigate the effects of LLA on trunk postural control and stability. It was hypothesized that persons with LLA have impaired trunk postural control compared to non-amputation controls, suggesting a decrement in spinal stability and the potential for increased risk of low back injury.

2.3 Methods

2.3.1 Participants

Eight males with unilateral LLA (4 trans-tibial, 4 trans-femoral) and eight male, non-amputation controls participated (Table 1). Members of the control group were recruited to match participants with LLA. Inclusion criteria for participants with LLA, consistent with previous studies comparing biomechanical characteristics between participants with and without LLA (Segal et al., 2011; Vrieling et al., 2008), were: 1) adults with a unilateral above- or below-knee amputation; 2) regular/daily use of prosthesis (≥ 1 year post-amputation/rehabilitation); and, 3) moderately active without the use of walking aids. Potential participants (in both groups) were excluded if they had any recent history (6 months) of falls, neurologic deficits, or any underlying musculoskeletal disorders that could confound the results. In particular, none of the participants in the study had low back pain at the time of testing. Mean (SD) duration of prosthetic use among participants in the LLA group were 12.3 (10.1) yrs. Prior to data collection, each participant completed informed consent procedures approved by the Virginia Tech Institutional Review Board (Appendix A). Participants with LLA were wearing their prosthetic device during all testing procedures.

Table 1. Mean (SD) participant characteristics in the lower-limb amputation (LLA) and control groups (*p*-values from unpaired *t*-tests).

	LLA (*n*=*8*)	Control (*n*=*8*)	*p*-value
Age (yr)	41.1 (18.7)	36.9 (13.4)	0.61
Stature (cm)	175.0 (5.0)	174.2 (3.8)	0.74
Body mass (kg)	76.6 (10.2)	80.3 (11.4)	0.50

2.3.2 Experimental Design and Procedures

Seated balance was tested using an unstable chair (Figure 1A) that pivots on a low-friction ball-and-socket joint. Adjustments to the seat allow for the participant's center of mass to be centered over the ball-and-socket joint. Four springs are placed circumferentially, spaced 90˚ in each cardinal direction, to provide supplemental seat support (Slota et al., 2008). These springs can be adjusted inward/outward (7-22cm) from the center, thereby facilitating control of task difficulty by altering the resistance of the chair to rotation (Figure 1B). Following calibration procedures (Lee and Granata, 2008; Slota et al., 2008), the spring positions were converted to a percentage of the gravitation gradient (gradG) for each participant seated on the chair. The value of gradG determines the mass (or weight) distribution of the participant on the chair, with 100% gradG specifying spring positions that will fully equilibrate the gravitational gradient. In other words, 100% gradG can facilitate seated stability with no need for compensation by the participant. Here, the task difficulty (i.e., spring positions) was standardized to 60% gradG for all four springs and for all participants. This setting is similar to what has been used in previous studies, which have determined that it is a level of difficulty sufficient for discerning differences in seated sway measures between groups or exposure conditions (Slota et al., 2008; Tanaka et al., 2009). It is slightly conservative, however, since it was not known a priori to what degree postural control would be degraded among persons with LLA. Pelvic motions were minimized using a belt placed across the hips, and an adjustable footrest was used to limit motion of the lower-limbs and keep the knees and hips at ~ 90˚ angles.

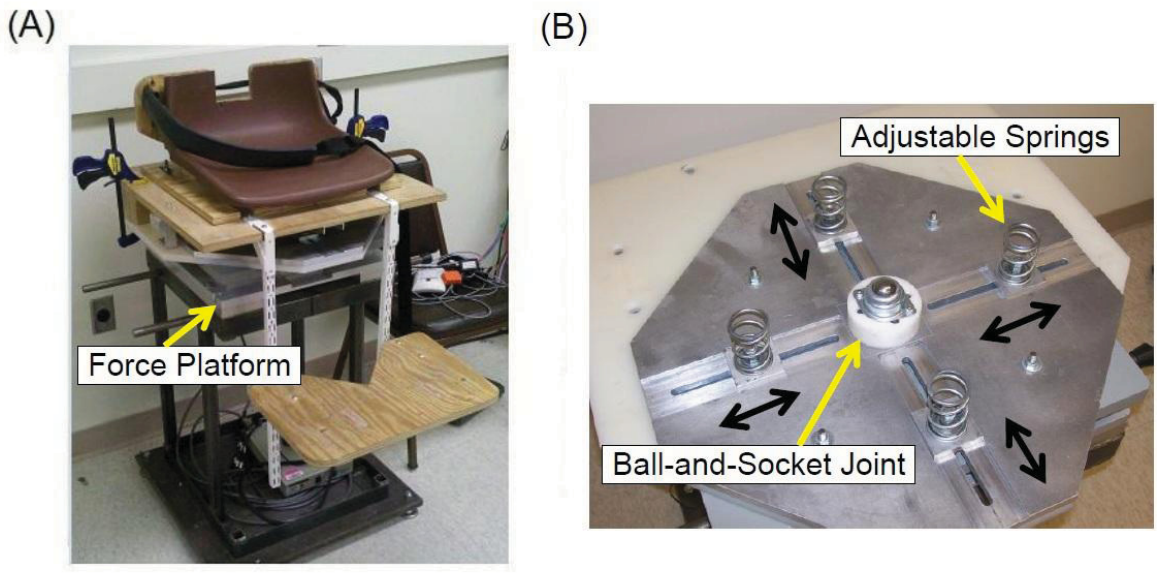

Figure 1. (A) Unstable chair with pelvic strap and adjustable foot rest, rigidly attached above a force platform. (B) Base of the unstable chair showing the central low-friction ball-and-socket joint and the adjustable A-P and M-L springs.

Initially, participants performed seated maximum voluntary contractions (MVCs) in trunk flexion, extension, and left/right lateral bending using a separate fixture. During these, electromyographic (EMG) activities of the bilateral lumbar (L3) erector spinae, rectus abdominis, and external oblique muscles were recorded using bipolar Ag/AgCl surface electrodes, and following existing electrode placement protocols (Granata and Wilson, 2001). Prior to applying electrodes, the skin was prepared using abrasion and cleaned with alcohol, and inter-electrode impedance was maintained below 10 KΩ. Raw EMG signals were preamplified (x100) near the collection site, bandpass filtered (10-500 Hz), amplified and converted to RMS in hardware (Measurement Systems Inc., Ann Arbor, MI, USA), then sampled at 1000 Hz. Peak EMG-RMS values were identified, and used subsequent for purposes of normalization (see below).

Participants were then given five practice trials to reduce learning effects and help participants acclimate to the task (Lee and Granata, 2008). The task involved maintaining seated balance on the chair using (primarily) motion of the lumbar spine. Participants completed three seated

balance trials. During these, participants were instructed to keep the chair surface as level as possible while sitting with an upright posture (no slouching), eyes open and looking straight ahead, and with their arms folded across their chest. Each trial lasted 65 s, and a minimum of two minutes of rest was provided between each to minimize fatigue. During trials, EMG was collected as during MVCs. In addition, triaxial reaction forces and moments were recorded (1000 Hz) using a force platform (AMTI, OR6-7-1000, Watertown, MA, USA) located beneath the chair/spring assembly. These were low-pass filtered (second-order, zero-phase-lag, Butterworth, 10 Hz cut-off frequency), and processed to obtain COP time series in the antero-posterior (A-P) and medio-lateral (M-L) directions.

2.3.3 Dependent Measures and Statistical Analysis

The initial 10 s and final 5 s of each seated balance trial were removed prior to analysis to avoid initial adjustments and anticipatory effects, respectively, and all COP data were de-meaned prior to analyses. Postural control during seated balance trials was then determined using several traditional measures derived from the COP time series (Prieto et al., 1996): 95% ellipse area (cm^2), RMS distance (cm), and mean velocity (cm/s). Among a large number of such measures, these were chosen based on evidence of relatively good within- and between-day reliability (e.g., Lee and Granata, 2008; Lin et al., 2008). Higher values of these measures are typically interpreted as indicating inferior (or deteriorated) postural control (Norris et al., 2005; Prieto et al., 1996). To supplement these traditional measures, local dynamic stability was assessed using non-linear stabilogram diffusion analyses of the COP time series. Stabilogram diffusion analyses allow for a control system to be characterized as two parts, involving both short-term open-loop control, and long-term closed-loop control (Collins and De Luca, 1993; Norris et al., 2005). A scaling exponent, H, evaluates correlations between past and future displacements (of COP), and is computed from the slope of the mean square COP

displacement versus time interval curve (log-log). A critical point, C_P, represents the transition from short-term open-loop control to long-term closed-loop control. However, only the short-term exponent (H_s) has been shown to differentiate between participant groups (Lee and Granata, 2008; Radebold et al., 2001), and long-term stability diffusion analysis parameters are not representative of a bounded system (Delignières et al., 2003); thus, long-term parameters were not used in the present study. The short-term scaling exponent (H_s) and the critical point coordinate [C_P; time (s) and amplitude (cm^2)] were calculated here, separately in the A-P and M-L directions.

All EMG responses were normalized to peak levels recorded during seated MVCs, and the mean EMG activity within each seated balance trial was obtained for each muscle. These were then combined for bilateral pairs of muscles, since initial analyses indicated no bilateral differences in normalized activity levels. To address the study hypothesis, one-way analyses of variance (ANOVA) were used to compare seated balance performance between participants with LLA and controls. Muscle activities from one participant with LLA were excluded from these analyses due to measurement errors. Note that initial analyses revealed no significant differences in any of the dependent measures between participants with trans-tibial and trans-femoral amputations, and thus all participants with LLA were analyzed as a single group. In addition, there were no significant or substantial differences (i.e., that might indicate learning effects) across the three replications, with one exception: mean rectus abdominis values were ~ 5% higher in the second of the three replications. All statistical analyses were performed using JMP (Version 9, SAS Institute, Cary, NC, USA) with statistical significance set at $p < 0.05$.

2.4 Results

All traditional measures of postural control (95% ellipse area, RMS distance, and mean velocity) were significantly larger among participants with LLA (Table 2). RMS distances were larger in the A-P direction for both groups. The short-term scaling exponent (H_s) was significantly larger among controls in the A-P direction, but similar in the M-L direction. Critical point time and amplitude were respectively longer and larger among participants with LLA in the A-P direction, but both were similar between groups in the M-L direction (Table 3). Mean normalized RMS muscle activities were larger among participants with LLA (Figure 2) in the L3 erector spinae ($p < 0.0001$), rectus abdominis ($p < 0.0001$), and external oblique ($p = 0.0045$).

Table 2. Mean (SD) center of pressure sway measures of seated balance among participants with LLA and matched controls. Significant differences between groups are indicated by bolded *p*-values.

Variable	LLA (*n*=8)	Control (*n*=8)	*p*-value
95% Ellipse area (cm^2)	6.50 (2.70)	3.94 (1.60)	**0.034**
RMS distance- AP (cm)	0.81 (0.25)	0.56 (0.12)	**0.015**
RMS distance- ML (cm)	0.51 (0.16)	0.37 (0.09)	**0.049**
Mean velocity- AP (cm/s)	0.88 (0.25)	0.62 (0.11)	**0.011**
Mean velocity- ML (cm/s)	0.95 (0.26)	0.60 (0.10)	**0.003**

Table 3. Mean (SD) non-linear stabilogram diffusion analysis parameters of seated balance among participants with LLA and matched controls. Significant differences between groups are indicated by bolded *p*-values.

Variable		LLA (*n*=8)	Control (*n*=8)	*p*-value
A-P	H_s	0.75 (0.04)	0.80 (0.04)	**0.003**
	C_P time (s)	1.46 (0.25)	1.20 (0.25)	**0.012**
	C_P amplitude (cm^2)	0.53 (0.26)	0.26 (0.09)	**0.009**
M-L	H_s	0.73 (0.07)	0.75 (0.06)	0.69
	C_P time (s)	1.32 (0.30)	1.16 (0.23)	0.22
	C_P amplitude (cm^2)	0.38 (0.21)	0.22 (0.12)	0.08

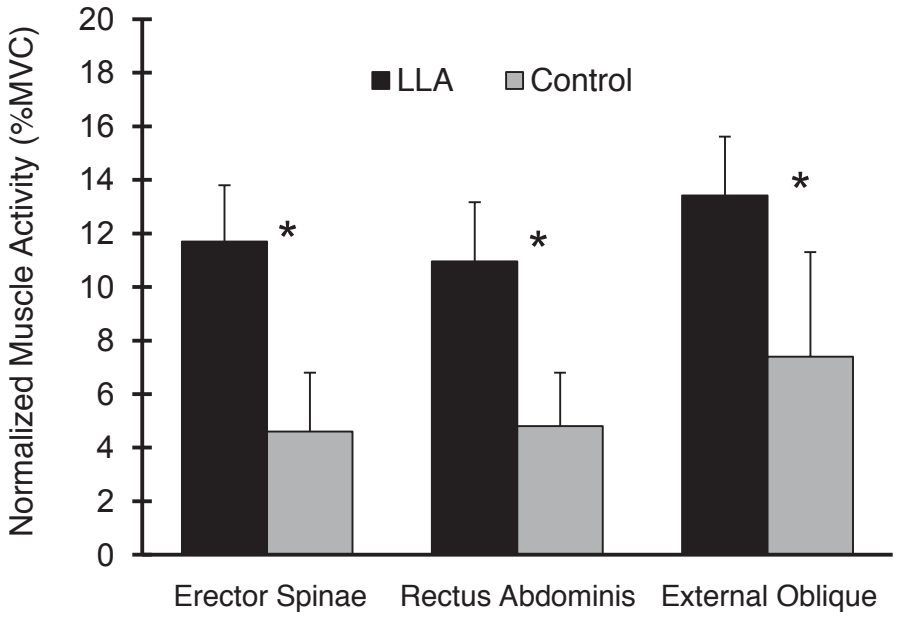

Figure 2. Normalized trunk muscle activities during seated balance tasks. Error bars indicate standard deviations, and asterisks represent a significant difference between groups.

2.5 Discussion

The primary goal of the present study was to investigate the effects of LLA on trunk postural control and spinal stability. It was hypothesized that participants with LLA have impaired trunk postural control compared to non-amputation controls, evidenced by increases in COP-based sway measures during seated balance. All traditional and non-linear COP sway measures were larger among participants with LLA compared to matched controls during seated balance (with the exception of H_s; see below), supporting our hypothesis and suggesting an association between LLA and reduced trunk postural control and spinal stability.

Increases in traditional COP-based sway measures among participants with LLA suggest a less tightly controlled postural control system in both the A-P and M-L directions (Table 2). Non-linear stabilogram diffusion analyses revealed similar differences in postural control in the A-P

direction among persons with LLA compared to controls, but similar control in the M-L direction. Comparable findings have been observed when comparing seated balance performance between patients with LBP and healthy controls, suggesting that the larger range of spinal motion in the sagittal plane better discriminates deficits in postural control between groups (Radebold et al., 2001). Although both groups here displayed persistent short-term movements ($H_s > 0.5$) in both directions, participants with LLA had significantly less persistence than controls in the A-P direction. Less persistence implies that deviations in a time series are less likely to be followed by subsequent deviations in the same direction (Dingwell and Cusumano, 2010). Given the increases in traditional COP-based sway measures, it can be argued that such reductions in persistence indicate a less healthy system (Peng et al., 1994).

Further, the C_P amplitudes for participants with LLA were nearly double in the A-P direction compared to controls, suggesting that the threshold for closed-loop corrective mechanisms is much larger among LLAs (Collins and De Luca, 1993; Collins et al., 1995; Norris et al., 2005). Likewise, longer C_P times among participants with LLA, and between the A-P and M-L directions, suggest increased delays in corrective responses to postural perturbations (i.e., transition from open-loop to closed-loop control). According to Collins and De Luca (1993), the C_P amplitude may be determined, physiologically, by a proprioceptive “dead zone” whereby slight variations in trunk position and orientation are left uncorrected. Similar to the interpretation provided by Radebold et al. (2001) with respect to LBP patients vs. healthy controls, it can be speculated that participants with LLA generated larger COP movements during short-term intervals to overcome larger sensory thresholds. This interpretation is consistent with calculated C_P amplitudes in the current work, and could indicate larger spinal neutral zones among participants with LLA. The spinal neutral zone is a region of intervertebral motion where the passive tissues provide little resistance to movement (Panjabi, 1992b). Increases in the neutral zone, which may occur with losses of passive stiffness, muscle

weakness, and/or inhibition of stabilizing musculature, have been associated with spinal instability and risk of injury (Panjabi, 1992b).

Increases in trunk muscle recruitment have been shown to decrease postural control during seated balance (Reeves et al., 2006), and to be related to decreased postural control and spinal stability among persons with LBP (Hodges et al., 2009). Here, participants with LLA had significantly higher levels of mean superficial trunk muscle activity during seated balance compared to controls (~12% MVC vs 5% MVC). Of note, the observed levels among controls are consistent with control data from an earlier study during seated balance (Reeves et al., 2006), as well as those among balancers and non-balancers (Preuss et al., 2005). When the spinal neutral zone is increased (as suggested above), the spinal column may become unstable, therefore requiring increasing compensations from active trunk muscles. Increases in trunk muscle activities among participants with LLA could therefore indicate an attempt to stiffen the trunk, perhaps to compensate for reductions in passive contributions to spinal stability. A stiffer trunk caused by increased trunk muscle activities could consequently result in overcorrections in posture during seated balance, and thus lead to the observed increases in traditional COP sway measures (most notably RMS distance and 95% ellipse area). Decreases in proprioception and/or reductions in reflexive muscle responses could similarly explain the need for increased muscle activation among persons with LLA, though future work is needed to determine if such decreases are indeed present.

2.5.1 Study Limitations

Challenges in recruiting persons with LLA made it difficult to obtain a homogeneous group with respect to age, reason for amputation, amputation level (e.g., trans-tibial vs. trans-femoral), and the duration of prosthetic use. Such differences could explain some of the variability in

responses among participants with LLA, and future work could explore potential dose-response relationships through more systematic recruitment criteria with better access to such a population. Nevertheless, non-amputation control participants were carefully matched, which should have controlled for important potential sources of confounding. We attributed changes in postural control directly to aspects related to spinal stability (i.e., passive trunk stiffness and active neuromuscular responses). However, participants with LLA could have inherent differences in other sensory systems (e.g., vestibular or visual) that affect seated balance performance. Finally, repetitive strain injuries in the lower back are possible with altered gait and movement biomechanics occurring with LLA, because of the linking between the pelvis, torso, and lower limbs (Dananberg, 1993). However, the current study could not discriminate if such injuries are causes or consequences of reduced postural control and spinal stability among persons with LLA.

2.6 Conclusions

All traditional and non-linear COP sway measures were larger among participants with LLA compared to matched controls during seated balance (with the exception of H_s), suggesting an association between LLA and reduced trunk postural control. Reductions in postural control and spinal stability may be a result of adaptations in functional tissue properties and/or neuromuscular responses caused by repetitive exposure to abnormal gait and locomotion, which could lead to an increased risk for spinal instability, intervertebral motions beyond physiological limits, and pain.

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Chapter 3: Persons with Unilateral Lower-Limb Amputation have Altered and Asymmetric Trunk Mechanical and Neuromuscular Behaviors Estimated using Multidirectional Trunk Perturbations.

3.1 Abstract

Among persons with unilateral lower-limb amputation (LLA), preferential use of the sound limb during gait and movement may lead to chronic alterations and/or asymmetries in trunk mechanical and neuromuscular behaviors. Trunk stiffness, the magnitude and timing of maximum reflex force, and EMG reflex delays of superficial trunk muscles, were estimated here using multidirectional (anteriorly- and laterally-directed) position perturbations. Potential alterations and asymmetries in these trunk behaviors were assessed among eight males with unilateral LLA, and eight healthy non-amputation controls. During anteriorly-directed perturbations, trunk stiffness and maximum reflex force were significantly lower, and the timing of maximum reflex force was significantly later, among participants with LLA. During lateral perturbations, trunk stiffness and maximum reflex force were also significantly lower among participants with LLA, and bilateral asymmetries were present in trunk stiffness and the timing of maximum reflex force in this group. Specifically, trunk stiffness was significantly lower and timing of maximum reflex force was significantly later during perturbations involving spinal tissues and muscles ipsilateral to the side of amputation. These differences in trunk stiffness and reflexive responses among persons with LLA suggest an association between low back pain risk and repeated exposure to abnormal and asymmetric gait and movement subsequent to LLA. Further, preferential use of the sound limb may result in bilateral asymmetries in trunk mechanical and neuromuscular behaviors, substantially reducing such responses on the same side of the amputation.

Keywords: Lower-Extremity; Amputation; Trunk Stiffness; Reflex; Position Perturbation

3.2 Introduction

Low back pain (LBP) is one of the most prevalent and costly musculoskeletal disorders (Dagenais et al., 2008), and while causal links between biomechanical factors and LBP have been investigated, these factors may play a more important role in certain populations. For example, there is a substantially higher prevalence of LBP among persons with lower-limb amputation (LLA; 52-71%) than among the general population (6-33%; Ehde et al., 2001; Smith et al., 1999). Asymmetries have been identified in the kinetics and kinematics of gait and other movements among persons with unilateral LLA (Sagawa Jr et al., 2011), often indicating a favoring of the sound limb and an association with altered trunk kinematics (Goujon-Pillet et al., 2008). Such alterations in trunk motion subsequent to unilateral LLA likely result in distinct spinal loading patterns (e.g., rates and magnitudes), due to increased external demands placed on the trunk neuromuscular system to achieve control of equilibrium and stability of the spine. Repeated exposures to such alterations could chronically alter trunk motor control strategies and/or functional properties of the passive spine. Despite the significant role of trunk mechanical and neuromuscular behaviors in the development of LBP, it is unknown whether there are changes in these due to lower-limb amputation and the subsequent repeated use of a prosthetic device.

The properties of passive spinal tissues/structures and reflexive neuromuscular responses from surrounding trunk musculature play an important role in the maintenance of spinal stability (Moorhouse and Granata, 2007; Panjabi, 1992). Several alterations in these properties and responses have been identified in patients with LBP, including increased trunk stiffness (Hodges et al., 2009), altered trunk muscle recruitment patterns (Radebold et al., 2000), and delayed/reduced reflex responses (Radebold et al., 2001). Numerous studies have also demonstrated similar disturbances in passive spinal tissues and trunk neuromuscular behaviors

among healthy persons following acute exposure to mechanical loading and atypical postures, suggesting an association between altered trunk behaviors and the occurrence of LBP. For example, prolonged static or dynamic forward trunk flexion reduces passive support of the spine (McGill and Brown, 1992), and mechanical/sensory deficits caused by repetitive passive loading of the lumbar viscoelastic tissues result in prolonged neuromuscular adaptation (Olson, 2011; Solomonow, 2012) and reduced reflex responses (Rogers and Granata, 2006). Preferential use of the sound limb in persons with LLA during walking alters kinematics of their trunk, thus exposing the spine and trunk neuromuscular system to repeated atypical mechanical loading and postures, and could lead to similar alterations and/or asymmetries in passive spinal properties and trunk reflexive responses.

Sudden-loading paradigms have been used to estimate trunk mechanical and neuromuscular responses to external perturbations (e.g., Brown and McGill, 2009; Gardner-Morse and Stokes, 2001). Recent work using position-controlled perturbations (vs. force perturbations) allow separate estimation of intrinsic (passive) mechanical properties and reflexive neuromuscular responses through the use of rapid and controlled perturbations (Bazrgari et al., 2011a; Hendershot et al., 2011; Moorhouse and Granata, 2007). Trunk perturbations are typically applied in the antero-posterior direction, though several studies have investigated the dynamic response of trunk muscles to multi-directional force perturbations (Gardner-Morse and Stokes, 2001; Masani et al., 2009; Stokes et al., 2006). While these latter studies demonstrated that the trunk dynamic response depends on perturbation direction, muscle responses were typically bilaterally symmetric when comparing left and right lateral perturbations among healthy individuals. However, due to the noted asymmetric tendencies among persons with LLA, multidirectional perturbations are expected to provide a better understanding of how repeated exposures to asymmetric movement could affect the symmetry in trunk mechanical and reflexive behaviors among persons with LLA. The goal of the present work was therefore to use

multidirectional sudden-loading position perturbations to investigate the effects of LLA on some aspects of trunk mechanical and neuromuscular behaviors. It was hypothesized that persons with LLA have altered and/or asymmetric trunk mechanical and neuromuscular behaviors compared to non-amputation controls.

3.3 Methods

3.3.1 Participants

Eight males with unilateral LLA – four trans-tibial (3 right leg, 1 left leg) and four trans-femoral (2 right leg, 2 left leg) – and eight male, non-amputation controls completed the study. Members of the control group were recruited to match participants with LLA, at the individual level, in terms of age, stature, and body mass (within <8 yrs, <5 cm, and <5 kg, respectively). Respective mean (SD) values of these for the participants with LLA were 41.1 (18.7) yr, 175.0 (5.0) cm, and 76.6 (10.2) kg. Corresponding values for the non-amputation control group were 36.9 (13.4) yr, 174.2 (3.8) cm, and 80.3 (11.4) kg, and there were no significant differences between groups ($p \geq 0.5$ from unpaired *t*-tests). Mean (SD) duration of prosthetic use among participants in the LLA group were 12.3 (10.1) yrs. Inclusion criteria for participants with LLA were consistent with previous studies comparing biomechanical characteristics between participants with and without LLA (Segal et al., 2011; Vrieling et al., 2008). The same criteria were also used in our prior study (Chapter 2), in which the same participants were involved. These criteria were: 1) adults with a unilateral above- or below-knee amputation; 2) regular/daily use of prosthesis (≥ 1 year post-amputation/rehabilitation); and, 3) moderately active without the use of walking aids. Potential participants (in both groups) were excluded if they had any recent history (6 months) of falls, neurologic deficits, or any underlying musculoskeletal disorders that may confound the results. In particular, none of the participants in the study had low back pain at the time of

testing. Experimental procedures were approved by the Virginia Tech Institutional Review Board, and participants provided informed consent prior to data collection (Appendix A). Participants with LLA were wearing their prosthetic device during all testing procedures.

3.3.2 Experimental Design and Procedures

A sudden-perturbation paradigm was used to quantify trunk mechanical and neuromuscular behaviors, as previously described (Bazrgari et al., 2011a; Hendershot et al., 2011). Here, participants were randomly exposed to a sequence of trunk perturbations in each of three configurations (Figure 3), with each configuration conducted in a random order across participants. During these, participants maintained a relaxed, upright posture in a rigid metal frame, and adjustable straps were used to restrain the pelvis and lower limbs. Each sequence consisted of 12 position perturbations (±5 mm) generated by a servomotor (Kollmorgen AKM53K, Radford, VA, USA), and that were applied to the trunk through a custom chest harness and rigid rod (Figure 3). A perturbation sequence was completed in ~45 s, and each perturbation was completed within 40 ms. Pseudorandom delays were included between each perturbation to minimize anticipation of perturbation timing by participants and thereby reduce potential confounding from variations in anticipatory muscle activation (Grondin and Potvin, 2009).

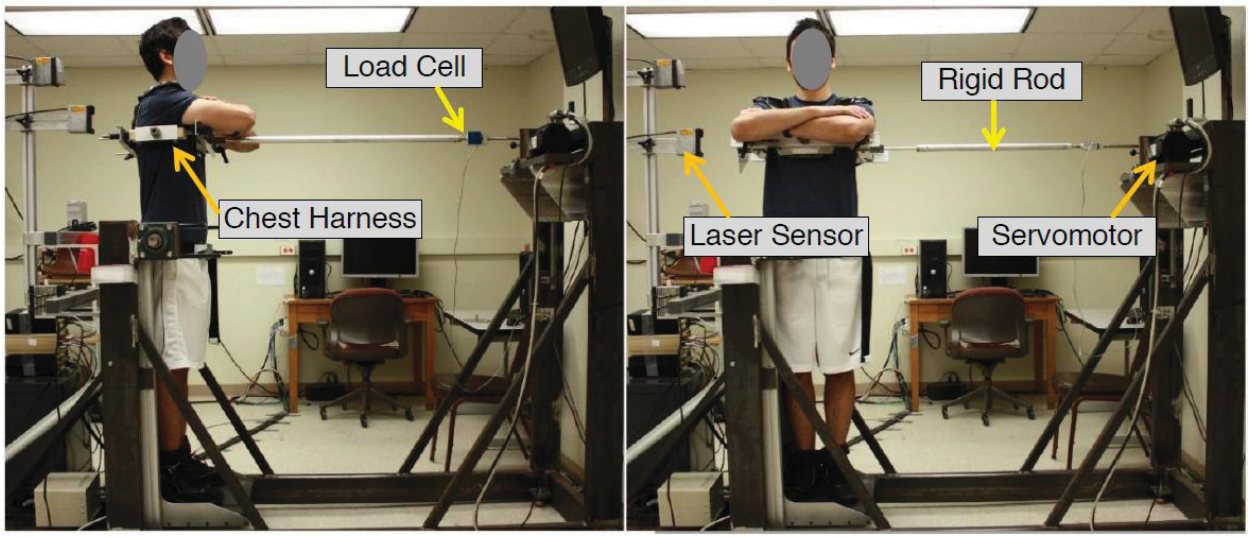

Figure 3. Experimental set-up demonstrating a control participant in an ANT (left) and LEFT (right) position. Participants were turned 180° and reconnected to the motor for RIGHT perturbations. Participants' arms were similarly folded across the chest and rested on the harness for all three perturbation directions.

Postural displacements were measured with a high-accuracy laser displacement sensor (Keyence LK-G 150, Osaka, Japan) and the servomotor encoder. The laser sensor was directed at the dorsal aspect of the trunk harness for anterior perturbations, and a rigid plate attached to the side(s) of the harness during lateral perturbations. Applied loads were measured with a load cell in-line with the rigid connecting rod (Interface SM2000, Scottsdale, AZ, USA). Load cell, motor encoder, and laser sensor data were collected at 1000 Hz, and filtered with a 10 Hz, bidirectional, low-pass filter (Bazrgari et al., 2011b). Electromyographic (EMG) activities of the bilateral lumbar (L3) erector spinae (ES) and external oblique muscles (EO) were recorded using bipolar Ag/AgCl surface electrodes, and following existing electrode placement protocols (Granata and Wilson, 2001). Prior to applying electrodes, the skin was prepared using abrasion and cleaned with alcohol, and inter-electrode impedance was maintained below 10 KΩ. Raw EMG signals were preamplified (x100) near the collection site, bandpass filtered (10-500Hz) and amplified in hardware (Measurement Systems Inc., Ann Arbor, MI, USA), and then sampled at 1000 Hz.

A subset of data was extracted from the perturbation sequences in each configuration. Specifically, mechanical and reflexive behaviors were determined (see below) from perturbations directed toward the motor. For example, when the motor was on the left, perturbations directed to the left were analyzed, and which thereby assessed stiffness of the trunk in left lateral bending and reflexive responses of the contralateral muscles (on the right, that were "stretched").

3.3.3 Dependent Measures and Statistical Analysis

Initially, the latencies of reflexive muscle responses following each perturbation were determined as the time delays between displacement onset and the onsets of muscle reflex responses, the latter identified as EMG peaks exceeding two standard deviations above mean activity prior to the perturbations (Granata et al., 2004; Zhang et al., 1999). Reflex latencies from the bilateral ES were determined and averaged bilaterally following anteriorly-directed perturbations. These latencies were only analyzed unilaterally from the contralateral ES and EO during right- and left-oriented lateral perturbations.

Trunk mechanical properties were then estimated by quantifying the trunk dynamic response in two time windows. The first window was restricted to the period of movement (~40 ms) to ensure that there were no contributions from muscle reflexes. During this window, the trunk was modeled as a single degree of freedom mass-spring-damper system, and an extra mass-spring-damper element was included to account for the mechanical properties of the connecting elements between the motor and the trunk (*cf.* Bazrgari et al., 2011a; Hendershot et al., 2011). Trunk damping was set to zero, as in our previous work (Bazrgari et al., 2011a; Hendershot et al., 2011) and consistent with prior evidence suggesting negligible contributions from trunk

damping (Cholewicki et al., 2000), so that alterations in trunk mechanical behaviors could be represented by changes in trunk stiffness. In the model, the mass of the connecting elements was 3.1 kg (vs. 1.5 kg in our prior work, to reflect increases in the mass of the new multidirectional harness design). Model parameters were estimated using a least-squares curve fit in MATLAB (Mathworks, Natick, MA, USA), minimizing the error between measured (load cell) and model-predicted driving forces (Hendershot et al. 2011).

Reflexive force was estimated in the second time window (i.e., after reflex delays), by subtracting the model-estimated intrinsic force contribution from the total measured trunk response (i.e., reaction force measured by the load cell). Magnitude and timing (with respect to perturbation onset) of the maximum reflex force represent the overall reflexive behavior of the trunk, differing from EMG-driven estimates that rely on measured EMG responses of select superficial trunk muscles. Reflexive analyses were limited to a window size of 150 ms following reflex onset to avoid subsequent voluntary muscle responses.

Perturbations yielding the highest correlation between measured and model-predicted force responses within a given sequence (of 12) were used for these analyses. Model correlations were similar between groups and directions (all $p > 0.24$). Results were pooled for all participants with LLA, given the small sample sizes and results from preliminary analyses that indicated no significant or substantial differences depending on the side or level of amputation. To address the study hypothesis, mixed-factor analyses of variance (ANOVAs) were used to compare our measures of trunk mechanical and reflexive behaviors between and within groups. One set of ANOVAs was used to compare responses between groups for anteriorly-directed (ANT) perturbations. A second set of ANOVAs was used to assess the results of laterally-directed perturbations. These included left- and right-directed (LEFT/RIGHT) perturbations for the control group, and ipsilaterally- and contralaterally-directed (IPSI/CONTRA) for the group

with LLA. For the latter, IPSI or CONTRA categorizations were based on the side of amputation; a perturbation to the left was IPSI if the amputation was on the right, given that reflexive responses on the right were assessed. Where the effect of direction was significant, a set of orthogonal *post hoc* contrasts was used to determine: 1) overall differences between groups; 2) bilateral differences among controls; and 3) bilateral differences among participants with LLA. All statistical analyses were performed using JMP (Version 9, SAS Institute, Cary, NC, USA), with statistical significance determined when $p < 0.05$. Summary statistics are presented as means (SD).

3.4 Results

During ANT perturbations, all measures of trunk mechanical and reflexive behaviors (except for ES reflex delay) were significantly different between groups (Table 4).

Table 4. Mean (SD) trunk mechanical and reflexive behaviors during anteriorly-directed perturbations. Significant differences between groups are indicated by bolded *p*-values.

	LLA (*n*=*8*)	Control (*n*=*8*)	*p*-value
Trunk Stiffness (N/mm)	10.0 (2.1)	13.2 (2.5)	**0.017**
Max. Reflex Force (N)	55.3 (11.8)	71.7 (12.3)	**0.017**
Timing of Max. Reflex Force (ms)	187.4 (4.6)	173.1 (11.4)	**0.005**
ES Reflex Delay (ms)	68.2 (3.8)	64.7 (3.6)	0.075

During lateral perturbations, there were substantial differences both between and within (bilateral) group. Trunk stiffness was significantly ($p = 0.021$) lower overall in participants with LLA, bilaterally similar ($p = 0.43$) among controls, but significantly ($p < 0.0001$) lower during IPSI perturbations among participants with LLA (Figure 4).

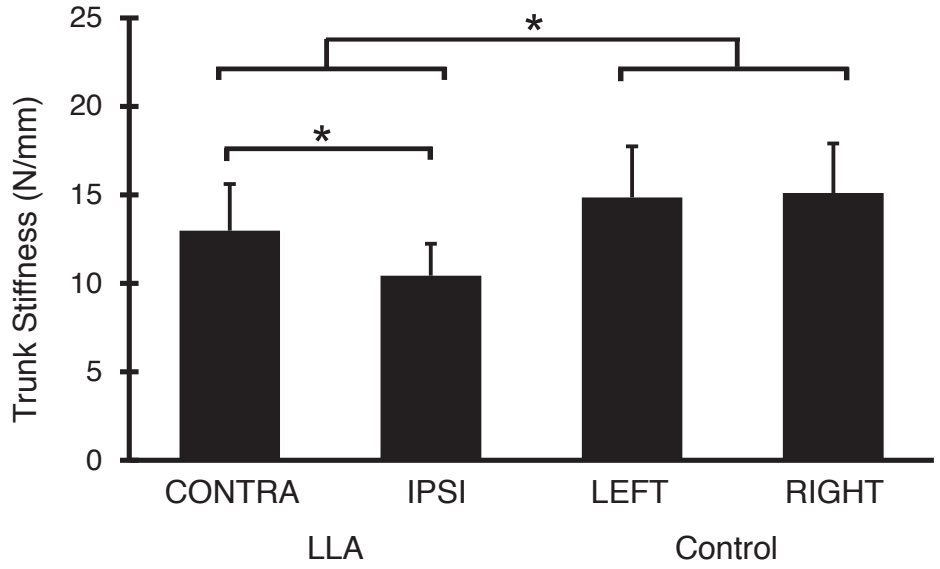

Figure 4. Between and within (bilateral) group differences in trunk stiffness during lateral perturbations. Error bars indicate standard deviations, and results from *post hoc* contrasts are indicated by brackets (* = significant difference).

Maximum reflex force was significantly ($p = 0.015$) lower overall in participants with LLA than controls, at 56.3 (18.3) and 77.7 (13.1) N, respectively, but bilaterally similar among both controls ($p = 0.95$) and participants with LLA ($p = 0.13$). Timing of maximum reflex force was significantly ($p = 0.002$) longer overall in participants with LLA, bilaterally similar ($p = 0.61$) among controls, but significantly ($p < 0.0001$) longer during IPSI perturbations among participants with LLA (Figure 5). ES reflex delays were similar ($p = 0.12$) between groups, but significantly ($p = 0.01$) shorter during left- vs. right-oriented perturbations among controls (62.8 (3.6) vs. 65.3 (3.3) ms, respectively). These delays were bilaterally similar ($p = 0.13$) among

participants with LLA. EO reflex delays were significantly (p = 0.002) longer in participants with LLA (68.2 (4.0) ms) vs. controls (60.7 (4.1) ms), but bilaterally similar among both controls (p = 0.78) and participants with LLA (p = 0.99).

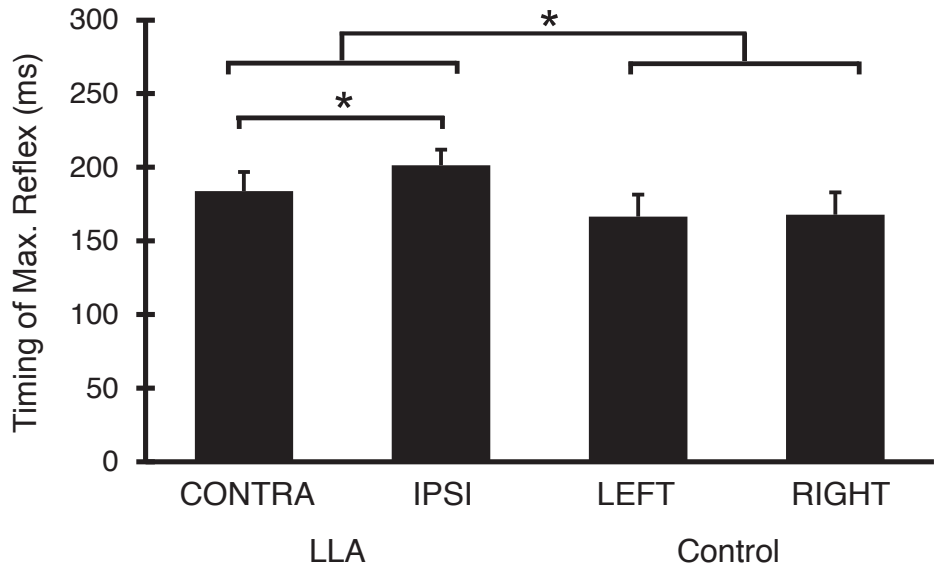

Figure 5. Between and within (bilateral) group differences in the timing of maximum reflex force during lateral perturbations. Error bars indicate standard deviations, and results from *post hoc* contrasts are indicated by brackets (* = significant difference).

3.5 Discussion

3.5.1 Overall Group-Level Differences

A relative decrease in trunk stiffness around neutral standing posture was found among participants with LLA. It has been suggested that such individuals adopt a neuromuscular/movement strategy that employs trunk weight/inertia to aid in stabilizing the entire body during gait and movement (Hof et al., 2007; Tura et al., 2010), and previous experimental studies have found increases in pelvic and trunk kinematics during gait compared

to able-bodied controls (Goujon-Pillet et al., 2008; Jaegers et al., 1995; Morgenroth et al., 2010). Therefore, increased (and repeated) deformation of soft tissues in/surrounding the spine, and resulting from such increases in kinematics, could cause reductions in the viscoelastic contributions of these tissues to trunk stiffness, and subsequent neuromuscular dysfunction (Avela et al., 2004; Magnusson et al., 1995; Sbriccoli et al., 2005). While increased trunk kinematics could potentially aid in stabilizing the whole body during movements, the resultant decrease in trunk stiffness reduces the stability of the spine and might simultaneously increase the risk for strain injuries within tissues surrounding the spinal column.

Reduced trunk stiffness in the current individuals with LLA, a group which more generally has a relatively higher prevalence of LBP, is contradictory to increases in trunk stiffness previously identified among persons with LBP (Hodges et al., 2009). As such, the present results suggest that a reduction in trunk stiffness around the standing posture may play a causal and/or indicator role for future occurrence of LBP. This argument is also supported by other findings that suggest increased trunk stiffness among patients with LBP is a result of increased muscle activations, not increases in contributions from passive tissues (Marras et al., 2001; van der Hulst et al., 2010; van Dieën et al., 2003). Of note, the magnitudes of trunk stiffness reported here are larger than those previously estimated using similar methods (Bazrgari et al., 2011a; Hendershot et al., 2011). However, differences in participant characteristics (increased age and body mass here) and methodologies (increased trunk harness mass and altered arm position here) may account for these increases.

The magnitude and timing of trunk reflex responses are important for maintaining spinal stability, since trunk stiffness alone cannot adequately stabilize the spine (Brown and McGill, 2009; Moorhouse and Granata, 2007). However, our results demonstrate that the efficiency of such reflex responses in persons with LLA is reduced, perhaps further increasing the risk of

LBP due to spinal instability. Reduced resistance from trunk viscoelastic connective tissues (reflected by overall decreases in trunk stiffness among persons with LLA) may be, in part, responsible for the alterations in reflexive muscle responses found in the LLA group. Acute exposure to passive stretch of posterior tissues has been shown to result in an immediate "hyper-excitability" in the reflex response (Bazrgari et al., 2011a; Hendershot et al., 2011; Solomonow, 2012). Repeated stretching, however, results in a depression in the magnitude of reflex response, and which often requires substantial time for full recovery (Gedalia et al., 1999; Rogers and Granata, 2006; Solomonow et al., 1999). Further, a delayed onset of reflexive muscle responses following trunk perturbations has been associated with LBP (Radebold et al., 2001). However, and consistent with an earlier study (Cholewicki et al., 2005), our results suggest that delayed reflexive responses may be a pre-existing risk factor for LBP, rather than an effect of LBP. The observed increases in EO reflex delays among persons with LLA during lateral perturbations are also notable here, since the obliques are important abdominal contributors to spinal stability in the standing posture (Arjmand et al., 2008; Gardner-Morse and Stokes, 1998).

3.5.2 Bilateral Differences

Preferential use of the sound limb among persons with unilateral LLA may contribute to bilateral differences in trunk mechanical and neuromuscular behaviors. Many alterations in gait and movement among persons with LLA indicate a favoring of the sound limb (Sagawa Jr et al., 2011), which can result in asymmetric distributions of joint loads in the musculoskeletal system. As a result, osteopenia and osteoporosis in the residual limb are major concerns that arise from reduced loading on the amputated side (Burke et al., 1978). Further, disuse atrophy decreases muscle size and strength, efficiency of muscle recruitment, and the tension-generating capacity of muscle (Nigam et al., 2009); all of these changes likely reduce trunk stiffness. Hypertrophy of

the psoas muscle on the non-amputated side, and a corresponding atrophy of the psoas muscle on the amputated side, have been identified among persons with LLA (Kulkarni et al., 2005); this muscle is an important trunk stabilizer that also contributes to hip flexion and trunk lateral flexion during unilateral contraction. In the present study, lateral trunk stiffness was greater than ANT trunk stiffness, and is likely due to the geometry of the trunk musculature in the standing posture (Brown and Potvin, 2007). Brown and McGill, (2009) also found that passive stiffness of muscle, even when relaxed, provides substantial stiffness to the spine. Reductions in IPSI trunk stiffness among persons with LLA in the present study may therefore reflect decreases in psoas (and other deep stabilizing muscles) contributions to trunk stability.

Bilateral asymmetries in the magnitude and latency of the reflexive response among participants with LLA may also arise from preferential use of the sound limb. Though not significant, maximum reflex force was 27% larger on the intact side of the body (CONTRA perturbations). Aruin et al., (1997) reported similar increases in background muscle activities in the sound side among persons with unilateral LLA, during anticipatory postural adjustments while standing. Additionally here, the timing of maximum reflex force occurred later on the side of amputation among persons with LLA, which could be related to decreases in the sensitivity of mechanoreceptors to detect muscle stretch due to decreases in trunk stiffness (Rozzi et al., 1999). While there was also a bilateral asymmetry in the ES reflex delay among controls, this difference [~2.5 ms (<5%)] may not be practically meaningful, and could be related to habitual activity and/or bilateral limb dominance. The EMG response of the ES also only represents one of many superficial and deep trunk muscles, while the mechanical estimate of the timing of maximum reflex force incorporates the overall trunk reflexive response. Further, bilateral differences in EMG measures have been shown to have poor reliability, and large sources of error may impair its use as a clinical measure (Larivière et al., 2005).

Higher background muscle activities have been suggested to increase the reflexive response of trunk muscles to sudden perturbations (Cholewicki et al., 2000; Granata et al., 2005). Though it might have increased the likelihood of detecting bilateral differences within groups, we did not include such a muscle pre-activation prior to perturbation in the present study to avoid confounding due to differences in muscle recruitment and neuromuscular behaviors between groups. As such, we set the goal of the present study to simply assess potential alterations and asymmetries in trunk mechanical and neuromuscular behaviors in the absence of voluntary trunk muscle pre-activation(s) prior to perturbations. It is possible that the relaxed state prior to perturbations in the present study was not capable of capturing bilateral differences in the reflex response among persons with LLA. Therefore, future work should investigate bilateral asymmetries in the reflex response among persons with LLA at varying magnitudes of pre-activation of the trunk musculature. It is also possible that the magnitude of the displacements previously found to sufficiently elicit a reflex response from posterior trunk muscles during ANT perturbations is too small during lateral perturbations. Specifically, larger displacements may be required to fully stretch contralateral muscles during lateral perturbations.

In summary, differences in trunk stiffness and reflexive responses among persons with LLA suggest an association between low back pain risk and repeated exposure to altered and asymmetric gait and movement subsequent to LLA. Further, preferential use of the sound limb may result in bilateral asymmetries in trunk mechanical and neuromuscular behaviors, substantially reducing such responses on the same side of the amputation.

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Chapter 4: Altered Flexion-Relaxation Responses during Asymmetric Trunk Flexion Movements among Persons with Unilateral Lower-Limb Amputation

4.1 Abstract

Repetitive exposure to alterations in gait and movement following lower-limb amputation (LLA) can lead to changes in passive tissue properties and neuromuscular control in/surrounding the lumbar spine, and which in turn may alter the synergy between passive and active tissues during trunk movements. Eight males with unilateral LLA and eight healthy controls completed quasi-static trunk flexion-extension movements in seven distinct conditions of rotation in the transverse plane: 0° (sagittally-symmetric), ±15°, ±30°, and ±45°. Electromyographic (EMG) activity of the bilateral lumbar erector spinae and lumbar kinematics were simultaneously recorded. Peak lumbar flexion and EMG-off angles were determined, as well as the difference ("DIFF") between these two angles. During sagittally-symmetric movements, peak lumbar flexion and EMG-off angles were similar between groups, but DIFF was significantly larger among controls. During sagittally-asymmetric movements, peak lumbar flexion significantly decreased with increasing transverse rotation. Among participants with LLA, peak lumbar flexion was significantly smaller when moving contralateral to the side of amputation at the higher rotation angles. EMG-off angles also significantly decreased with increasing transverse rotation, but this effect was similar between directions and groups. DIFF measures were bilaterally similar among controls, but significantly smaller during CONTRA movements at 30° transverse rotation among participants with LLA. Decreased and asymmetric passive contributions to trunk movements require increases in active trunk muscle responses, altering the load-sharing behavior between active and passive low back tissues.

Keywords: Amputation; Lower-Extremity; Lumbar Spine; Flexion-Relaxation Phenomenon

4.2 Introduction

An estimated 623,000 people were living with a major lower-limb amputation (LLA) in the United States in 2005, a number that is projected to more than double by the year 2050 (Ziegler-Graham et al., 2008). Chronic secondary physical conditions and pain are common among persons with LLA (Gailey et al., 2008). Low back pain (LBP) in particular, which has been rated as more bothersome than residual and phantom limb pain (Ehde et al., 2001; Smith et al., 1999), represents a substantial secondary disability among persons with LLA. Due to the coupling of joints in the musculoskeletal system (Dananberg, 1993), alterations in gait and movement patterns among persons with LLA may influence the loading in tissues in/surrounding the spine and thereby contribute to chronic alterations in passive spinal tissues and neuromuscular control. Further, experimental studies indicate increases in trunk and pelvic kinematics during gait compared to able-bodied controls (Goujon-Pillet et al., 2008; Jaegers et al., 1995; Morgenroth et al., 2010); these may lead to (or be consequences of) increases in the demands on the trunk musculature to maintain equilibrium and stability of the spine. Though it appears that repetitive exposures to altered gait and movement among persons with LLA may play an important role in the development of LBP in this population, it is currently unclear whether adaptations in trunk passive properties and/or neuromuscular control occur as a result of LLA and subsequent use of a prosthetic device.

Preserving an effective synergy between passive spinal structures and active trunk neuromuscular responses is important for maintaining spinal stability and controlling spinal loads (Panjabi, 1992). As such, differences in the mechanical properties of spinal tissues can alter the active-passive load sharing behavior of the spine and surrounding musculature. Reductions in the passive resistance to forward flexion movements, for example, have been related to spinal instability (Graham and Brown, 2012; Olson et al., 2004; Solomonow et al.,

2000). Prolonged static trunk flexion reduces passive support of the spine (Bazrgari et al., 2011; Hendershot et al., 2011; McGill and Brown, 1992), delays the onset of muscle relaxation during flexion movements (Shin and Mirka, 2007), and increases the magnitude of muscle activation during flexion/extension movements (Olson et al., 2004). Repeated exposures to abnormal mechanics of movement and increased trunk kinematics with LLA could lead to similar alterations in the active and passive contributions to flexion-extension movements.

The myoelectric silence that develops in the posterior musculature as the passive spine resists forward flexion – aka the "flexion-relaxation phenomenon" – has long been used as a means for assessing the noted synergy (e.g., Schultz et al., 1985). The extent of load transfer from active muscles to passive tissues during forward-flexion movements can vary depending on the mechanical properties of passive tissues (Adams and Dolan, 1996; Gunning et al., 2001). While reduced and asymmetric intrinsic (passive) mechanical properties have been identified around the neutral spine posture among persons with LLA (Chapter 3), it is presently unclear how these changes in passive properties influence the load-sharing behavior (synergy) between active and passive responses during forward-flexion movements. Therefore, the goal of the present work was to use controlled trunk flexion-extension movements to investigate the effects of LLA on active-passive load sharing mechanisms of the low back. It was hypothesized that persons with LLA have decreased and/or asymmetric passive contributions to load sharing behaviors compared to non-amputation controls.

4.3 Methods

4.3.1 Participants

Eight males with unilateral LLA (4 trans-tibial, 4 trans-femoral) and eight male, non-amputation controls completed the study. Members of the control group were recruited to match participants with LLA. Participant characteristics, and inclusion and exclusion criteria for all participants, were the same as our prior studies (Chapters 2 and 3), and in which the same participants were involved. In particular, none of the participants in the study had low back pain at the time of testing. Prior to data collection, each participant completed informed consent procedures approved by the Virginia Tech Institutional Review Board (Appendix A). Participants with LLA were wearing their prosthetic device during all testing procedures.

4.3.2 Experimental Design and Procedures

Quasi-static trunk flexion-extension movements were performed in seven distinct conditions of rotation in the transverse plane: 0° (sagittally symmetric), ±15°, ±30°, and ±45°. The latter three were performed bilaterally, directed to the left and right (Figure 6A), and this set of movements is similar to that described earlier by Ning et al. (2011). Participants stood in a rigid metal frame, and movement of the pelvis and lower limbs was minimized with a rigid pelvic restraint and adjustable strap around the ankles (Figure 6B).

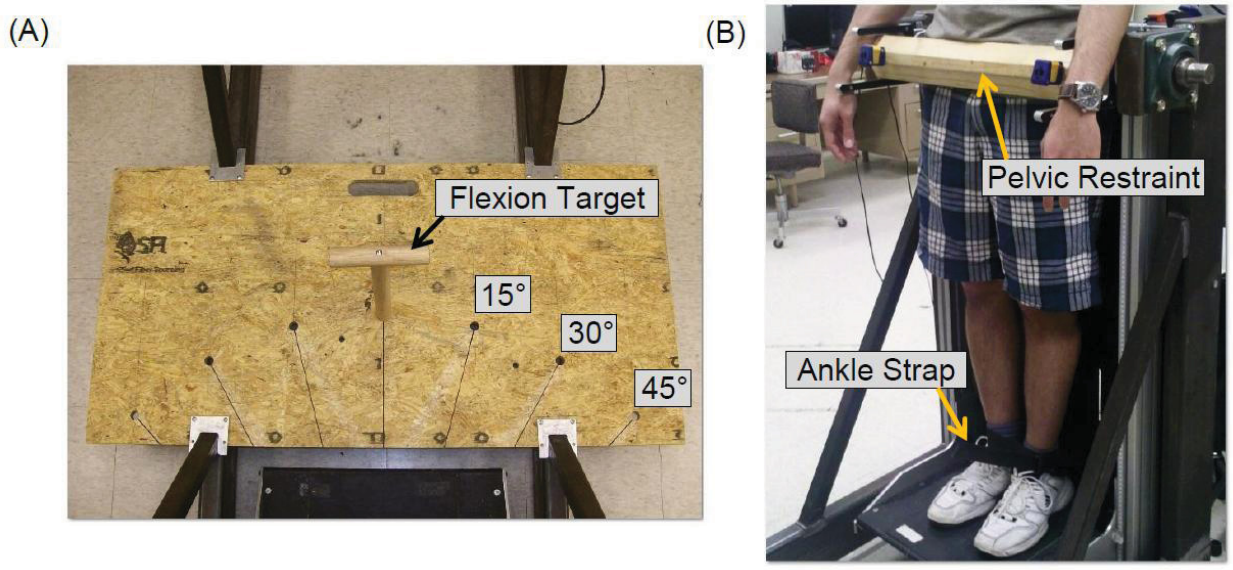

Figure 6. (A) Top view of the seven distinct conditions of rotation in the transverse plane. The moveable flexion target is depicted in the 0° (sagittally-symmetric) position. (B) Experimental set-up demonstrating the rigid pelvic restraint and ankle strap used to constrain the pelvis and lower limbs during flexion movements.

Initially, participants performed standing maximum voluntary contractions (MVCs) in trunk flexion, extension, and left/right lateral bending with the pelvis restrained (Figure 6B). During these, electromyographic (EMG) activities of the bilateral lumbar (L3) erector spinae (ES), rectus abdominis, and external obliques were recorded using bipolar Ag/AgCl surface electrodes, and following existing electrode placement protocols (Callaghan and Dunk, 2002; Mirka, 1991). Prior to applying electrodes, the skin was prepared using abrasion and cleaned with alcohol, and inter-electrode impedance was maintained below 10 KΩ. Raw EMG signals were preamplified (x100) near the collection site, bandpass filtered (10-500Hz), amplified and converted to RMS in hardware (Measurement Systems Inc., Ann Arbor, MI, USA), and then sampled at 1000 Hz. Peak EMG-RMS values were identified, and used subsequent to qualitatively monitor the magnitude of abdominal activity (see below).

Twenty-one movements, including 3 replications of each of the seven conditions, were then performed, with the presentation order of transverse rotation randomized. During each

movement, participants were instructed to slowly (~5 s) flex forward toward targets (Figure 6A) until reaching a relaxed, passive hanging position (~3 s) with minimal muscle activity and arms hanging relaxed, and then slowly (~5 s) return to upright standing posture. In the initial flexion phase, participants were instructed/required to approach but not actually touch the targets. Computer-generated auditory signals ("beeps") were used to assist participants in maintaining pacing during movements. Participants remained in a relaxed, upright posture for 30 seconds after each movement to minimize the effects of accumulated creep and fatigue (Shin and Mirka, 2007; Solomonow et al., 2003). During these movements, EMG was collected as during MVCs. In addition, lumbar kinematics were sampled at 100 Hz from two triaxial 6DOF inertial measurement units (IMUs: Xsens Technologies, XM-B-XB3, Enschede, The Netherlands) placed over the T12 spinous process and the superior aspect of the sacrum (S1) using double-sided tape.

4.3.3 Data Analysis

Peak lumbar flexion angle was defined as the relative difference between the T12 and S1 sensors in the standing (initial) vs. flexed hanging (final) postures (Newcomer et al., 2000; Ning et al., 2011; Van Herp et al., 2000). As such, a zero value corresponded to lumbar flexion in the relaxed, upright standing (initial) posture. All EMG data were rectified and then smoothed using a fourth order, zero-lag Butterworth low pass filter with a 5 Hz cutoff (Jin et al., 2012). Processed EMG data from the ES were then temporally matched to lumbar flexion angles to determine "EMG-off" angles (Ning et al., 2011; Solomonow et al., 2003), using within-trial references to determine thresholds for EMG timing (Jin et al., 2012). Within each movement, the difference ("DIFF") between peak lumbar flexion angle and EMG-off angle was also calculated. Abdominal EMG activities were analyzed qualitatively in real-time to ensure that

flexor contributions were < 5% of maximal values during both trunk movements and the passive hanging portion of each movement.

4.3.4 Statistical Analyses

To address the study hypotheses, mixed-factor analyses of variance (ANOVA) were used to assess peak lumbar flexion and EMG-off angles between and within groups. One set of ANOVAs was used to compare these responses between groups during sagittally-symmetric (i.e., 0°) movements. A second set of ANOVAs was used to assess the results of sagittally-asymmetric movements (i.e., 15°, 30°, and 45° to the right and left). These asymmetric movements were categorized as being directed to the LEFT and RIGHT for participants in the control group, and ipsilateral (IPSI) and contralateral (CONTRA) within the LLA group. For the latter, movements were defined as being performed away from (CONTRA) or towards (IPSI) the side of amputation. Initial analyses revealed no significant or substantial differences across the three movement replications in each rotation condition. A set of orthogonal *post hoc* contrasts was used to determine: 1) overall differences between groups; 2) bilateral differences among controls; and 3) bilateral differences among participants with LLA. All statistical analyses were performed using JMP (Version 9, SAS Institute, Cary, NC, USA), with statistical significance concluded when $p < 0.05$. Summary statistics are presented as means (SDs). As observed by Ning et al. (2011), and given the low occurrence of muscle relaxation, muscles on the same side as the direction of an asymmetric movement (e.g., left ES during a movement directed to the left) were excluded from these analyses. Because of the low occurrence of muscle relaxation during the 45° transverse rotation condition specifically, EMG-off and DIFF angles during these movements were also excluded from analyses.

4.4 Results

During sagittally-symmetric movements, peak lumbar flexion ($p = 0.26$) and EMG-off ($p = 0.27$) angles were similar between groups, with respective values of 44.1 (4.9)° and 36.3 (4.3)° among participants with LLA and 47.2 (5.3)° and 33.9 (4.0)° among controls. The DIFF angle was significantly ($p = 0.02$) larger among controls [13.3 (5.1)°] than participants with LLA [7.9 (3.2)°]. During sagittally-asymmetric movements, peak lumbar flexion angle significantly ($p < 0.001$) decreased with increasing transverse rotation. Specifically, peak lumbar flexion angles were 42.0 (5.3)°, 37.4 (4.3)°, and 33.2 (5.2)° for the 15°, 30°, and 45° transverse rotations, respectively. Peak lumbar flexion angles were also significantly ($p < 0.001$) smaller across all transverse rotations when flexing CONTRA (35.4 (6.8)°) vs. IPSI (38.4 (4.2)°) among participants with LLA, but bilaterally similar ($p = 0.52$) among controls [LEFT = 38.0 (6.4)° vs. RIGHT = 38.3 (6.4)°]. There was a significant ($p = 0.0005$) transverse rotation x movement direction interaction effect. Peak lumbar flexion angles were significantly smaller among participants with LLA during CONTRA movements for the 30° ($p < 0.0001$) and 45° ($p < 0.0001$) transverse rotations, but were bilaterally similar (all $p > 0.40$) among controls for all three transverse rotations (Figure 7).

EMG-off angles also significantly ($p < 0.0001$) decreased with increasing transverse rotation, with respective values of 35.2 (4.6)° and 31.3 (3.9)° for the 15° and 30° transverse rotations, but were similar between directions ($p = 0.36$) and groups ($p = 0.93$). DIFF angles were bilaterally similar at both transverse rotation angles among controls ($p = 0.13$), bilaterally similar at the 15° transverse rotation among participants with LLA ($p = 0.07$), but significantly ($p < 0.001$) smaller during CONTRA movements at the 30° transverse rotation among participants with LLA (Figure 8).

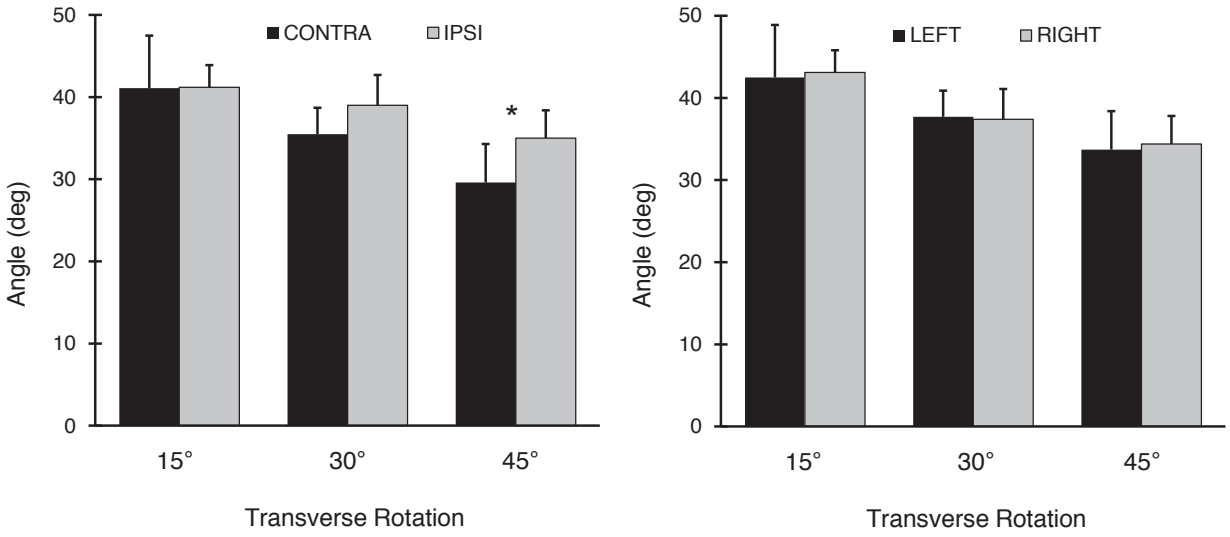

Figure 7. Peak lumbar flexion angles among participants with LLA (left) and controls (right). Error bars indicate standard deviations, and asterisks represent significant bilateral differences within a rotation condition.

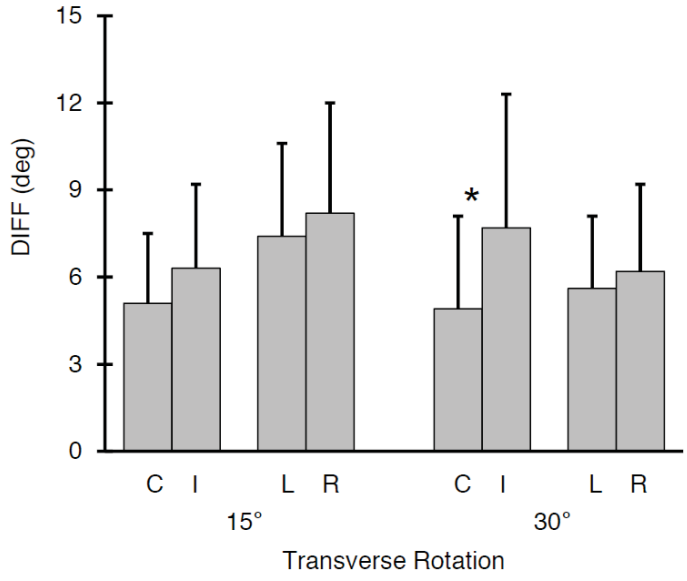

Figure 8. Differences between peak lumbar flexion and EMG-off angles (DIFF). Results are given for CONTRA (C) and IPSI (I) movements among participants with LLA, and LEFT (L) and RIGHT (R) movements among controls. Error bars indicate standard deviations, and the asterisk indicates a significant bilateral difference.

4.5 Discussion

While there were no significant overall group differences, peak lumbar flexion and EMG-off angles tended to be respectively smaller and larger among participants with LLA during sagittally-symmetric movements. There were, though, significantly smaller DIFF angles among participants with LLA. As suggested by Hashemirad et al., (2009), the central nervous system likely monitors the tension developed in posterior spinal tissues and "determines" when the passive tissues can adequately control the remaining portion of the forward flexion movement without need for active muscle responses. Solomonow et al. (2003) reported a decrease in the DIFF angle (due to increases in the EMG-off angle) following 10 minutes of static trunk flexion. Smaller DIFF angles among participants with LLA during sagittally-symmetric movements in the present study could therefore indicate similar reductions in passive contributions to forward flexion movements. If so, additional contributions from active muscles would be required throughout a longer portion of the movement. Though not formally analyzed here, larger peak magnitude of ES EMG activity during the flexion and extension movements were observed among participants with LLA, supporting the idea that increased muscle activity was required to overcome reductions in passive contributions to joint stiffness throughout the movements.

Overall, peak lumbar flexion and EMG-off angles decreased with increasing transverse rotation, and this effect likely resulted from additional frontal plane motion and a relatively earlier increase in passive tension developed in contralateral tissues (Ning et al., 2011). Movements here were performed bilaterally, allowing for separate (left and right) evaluations of muscle and tissue contributions to forward flexion movements. Asymmetric trends in the DIFF angles were observed among both groups, though these asymmetries were more substantial among participants with LLA with increased transverse rotation (Figure 8). While the EMG-off angles were bilaterally similar, peak lumbar flexion angles were significantly smaller during CONTRA

movements. Therefore, the ES muscles were activated throughout a longer portion of the range of motion during CONTRA movements. This was reflected by a significantly smaller DIFF angle during CONTRA movements at the 30° transverse rotation, and supports prior evidence of smaller relative passive trunk stiffness in relaxed upright standing ipsilateral vs. contralateral to the side of amputation (Chapter 3).

Static end range-of-motion (ROM) can be used as an indicator of joint flexibility/stiffness (McHugh et al., 1998). Based on previous work indicating lower trunk stiffness ipsilateral vs contralateral to the side of amputation (Chapter 3), it was expected that peak lumbar flexion angles would be larger overall among persons with LLA and larger during CONTRA movements. However, the observed peak lumbar flexion angles were contradictory to these expectations, and may have resulted from contributions of different tissues near the end of trunk ROM. Further, static trunk flexion ROM and the onset of trunk muscle relaxation can be influenced by the strength and relative length of muscles of the trunk and hip, as well as the coordination of trunk and hip movements (Gupta, 2001; Pal et al., 2007). Alterations in muscle properties and recruitment strategies within the lower-limb could therefore influence peak lumbar flexion angles and muscle coordination. Therefore, inherent bilateral differences in lower-limb musculature (e.g., tightness due to immobilization) among participants with LLA could have limited movement near the end of trunk range of motion. Such differences in hip muscles between groups may explain the smaller peak lumbar flexion angles observed during CONTRA movements among participants with LLA. Performing the flexion-extension movements freely (i.e., without a pelvic restraint) may provide more insight into the coordination of the lumbar spine and pelvis (“lumbopelvic rhythm”) among persons with LLA. Future work should also investigate dynamic measures of trunk flexibility and range of motion.

All participants with LLA in the current study exhibited bilateral relaxation of the ES during sagittally-symmetric movements, and contralateral relaxation during asymmetric movements. In contrast, patients with LBP typically do not exhibit a relaxation of the superficial back muscles during flexion movements (Shirado et al., 1995; Watson et al., 1997). It has been suggested that the absence of muscle relaxation during forward flexion among persons with current LBP may act to limit motion near the end of the range of motion, or may be volitional (guarding) and related to fear of further pain (Geisser et al., 2004; Triano and Schultz, 1987; Trost et al., 2012). However, none of the current participants (in either group) were experiencing LBP at the time of testing. It is also presently unclear how other trunk muscles contributed to the measured responses during these trunk movements. Deep trunk (stabilizing) muscles are more active towards the latter part of forward trunk flexion despite the relaxation of the superficial posterior muscles (Andersson et al., 1996), and can influence joint range of motion and stability (Quint et al., 1998). Future work should investigate such muscular contributions to flexion movements among persons with LLA.

4.6 Conclusions

Movements involving/requiring trunk flexion are frequent among activities of daily living, and alterations in the synergy between active and passive responses during such movements may lead to an increased risk for spinal instability and LBP. The current findings begin to identify how alterations in gait and movement subsequent to LLA can lead to decreases in the passive contributions to joint stability during trunk movements, and thus requiring increases in active muscle responses. Overall increases in trunk muscle activities can increase the magnitude of spinal load (Callaghan et al., 1998; Granata and Marras, 1995), and increases in spinal load have been related to LBP (Adams et al., 2006; McGill 2007; White and Panjabi, 1990). Further, altering the coordination of muscle responses could impact the type of spine loading

(compression vs. shear) throughout trunk movement, particularly during sagittally-asymmetric trunk movements, which can result in higher lateral shear forces than sagittally-symmetric movements (Davis and Marras, 2000). Large muscle forces also increase the loads in passive structures (e.g., disc or ligament), and can lead to errors in proprioceptive feedback (Panjabi, 1992; Solomonow, 2009). Hence, increases in active trunk muscle forces among persons with LLA during trunk movements may increase the risk for low-back injury in this population.

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Chapter 5: Conclusion

5.1 Overall Group-Level Differences

The present studies suggest that trunk passive and active neuromuscular responses are altered among persons with lower-limb amputation (LLA). Trunk stiffness around the neutral posture was relatively lower in all three perturbation directions among participants with LLA (Chapter 3). Relative decreases in the DIFF angle during controlled trunk flexion movements (Chapter 4), and increases in C_P amplitudes during seated balance trials (Chapter 2), also suggest reductions in passive contributions to spinal stability among persons with LLA. The magnitude and timing of trunk reflex responses were respectively decreased and delayed among persons with LLA (Chapter 3). Erector spinae muscles were active throughout a longer portion of controlled trunk movements (Chapter 4), and voluntary trunk muscle responses were significantly larger during seated balance trials among participants with LLA (Chapter 2).

Vertical and lateral displacements of the body's center of mass are typically limited to reduce energy expenditure during gait (Kuo et al., 2007). However, persons with LLA may adopt a neuromuscular/movement strategy that employs trunk weight/inertia to aid in stabilizing the entire body during gait and movement (Hof et al., 2007; Tura et al., 2010), and increases in trunk kinematics have been identified in such individuals (Goujon-Pillet et al., 2008; Jaegers et al., 1995; Morgenroth et al., 2010). Therefore, increased (and repeated) deformation of soft tissues in/surrounding the spine could cause reductions in the viscoelastic contributions to spinal stability and subsequent trunk neuromuscular dysfunction (Avela et al., 2004; Magnusson et al., 1995; Sbriccoli et al., 2005). While active trunk muscle responses and involuntary muscle reflexes complement passive contributions in the maintenance of spinal stability, repeated exposure to passive stretch of tissues and muscles reduce the force-generating capacity of

skeletal muscles and the magnitude of reflex responses. Further, a delayed onset of reflex response timing has been associated with decreases in passive stiffness, due to reduced sensitivity of mechanoreceptors (Rozzi et al., 1999).

5.2 Bilateral Differences

Several bilateral asymmetries in trunk behaviors were identified among persons with LLA. Trunk stiffness was significantly lower during ipsilateral vs. contralateral perturbations and, though not significant, maximum reflex force was 27% larger on the sound side (Chapter 3). The maximum reflex force occurred later ipsilateral vs. contralateral to the side of amputation (Chapter 3), and peak lumbar flexion angles were smaller when flexing contralateral to the side of the amputation (Chapter 4). Similarly, smaller DIFF angles during contralateral movements, particularly at larger transverse rotations, indicate decreased passive contributions during such movements. While there were no measured bilateral differences during seated balance trials (Chapter 2), the task itself does not easily allow for such analyses versus the other two studies.

Asymmetric kinematics and kinetics of gait and other movements among persons with LLA result from favoring the sound limb (Sagawa Jr et al., 2011). These movement patterns can lead to asymmetric distributions of loads in the musculoskeletal system, and may contribute to bilateral differences in trunk passive properties and neuromuscular responses. For example, osteopenia and osteoporosis in the residual limb are major concerns that arise from reduced loading on the amputated side (Burke et al., 1978). Disuse atrophy also decreases muscle size, efficiency of muscle recruitment, and the active and passive tension-generating capacity of muscle (Nigam et al., 2009). Atrophy of the psoas muscle on the amputated side, and a corresponding hypertrophy of the psoas muscle on the non-amputated side, have been identified among persons with LLA (Kulkarni et al., 2005). The bilateral differences observed in

the present studies suggest that asymmetric movement and favoring the sound limb affect trunk behaviors among persons with LLA.

5.3 Research Limitations and Future Directions

Several limitations were present in the current research, and should be addressed in future work. First, the relatively small sample size may limit generalizability. Second, challenges in recruiting persons with LLA for the present studies made it difficult to obtain a homogeneous group with respect to age, reason for amputation, amputation level, time since amputation, and duration of prosthetic use. However, the mean (SD) duration of prosthetic use among participants in the LLA group was 12.3 (10.1) years, and non-amputation controls were carefully matched, which should have controlled for important sources of confounding. Future work could explore potential dose-response relationships through more systematic recruitment criteria and with better access to such a population. For example, selective recruitment (and a larger sample size) could provide insight into when (relative to the initial use of a prosthetic device), and to what degree these changes in trunk behaviors start to occur. Important conclusions could also be drawn by comparing these trunk behaviors among persons with congenital or early-life amputations, to those of more recent traumatic LLAs (e.g., injured service members or victims of motor vehicle accidents). As such, understanding the temporal relationship between altered trunk behaviors and repeated exposure to abnormal gait and movement subsequent to LLA may help identify critical years for rehabilitation and preventative care.

5.4 Summary

The focus of this research was to investigate the link between repeated exposures to abnormal gait/movement and altered trunk passive properties and neuromuscular control among persons with LLA. The present results suggest that repetitive exposures to abnormal gait and movement, subsequent to LLA, alter trunk passive properties and neuromuscular control. Preferential use of the sound limb during gait and movement may also lead to asymmetries in these behaviors. Considering the range in the duration of prosthetic use among the LLA group, it is not presently clear exactly when these changes in trunk behaviors occur, or whether the extent of the changes is dependent on the level or reason for amputation. However, the current results begin to identify an association, and form the foundation for future work regarding chronic alterations in trunk behaviors resulting from repeated exposure to abnormal gait and movement.

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Appendix A: Informed Consent Form

VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Informed Consent for Participation in Research Projects Involving Human Subjects

Title of the Research Study:

Trunk Mechanics and Neuromuscular Control in Low Back Pain: Comparisons between Lower-Limb Amputees and Healthy Controls

Investigators:

Dr. Maury A. Nussbaum (nussbaum@vt.edu): Department of Industrial and Systems Engineering
Bradford Hendershot (hendershot@vt.edu): School of Biomedical Engineering and Sciences

I. Purpose of this Research

To better understand potential causes of musculoskeletal injury of the lower back. Of particular interest, is exploring how lower-limb amputation affects trunk mechanics and surrounding muscle responses. A total of up to 24 participants will be involved with this study, ranging in ages from 18-70 years, and including up to 12 lower-limb amputees and 12 matched, healthy controls.

II. Procedures

An experimental session is expected to last about two hours and you may be requested to return for repeated testing. All experiments will be performed in the Industrial Ergonomics and Biomechanics Laboratory located on the fifth floor of Whittemore Hall. We will tape adhesive markers and sensors on your skin around your abdomen and lower back. These sensors are electrodes that measure the activity of your muscles and position sensors to measure how you move. After some preliminary warm up stretches, we may ask you to push and/or pull as hard as you can against a resistance. We will then ask you to perform several slow flexion-extension movements of your upper body throughout your range of motion. We may apply quick but small perturbations to your trunk to record muscle reflexes. You will also maintain seated stability on an unstable chair.

III. Risks

The risks of this study are minor. However, they include a potential skin irritation to the adhesives used in the tape and electrode markers. You may also feel some temporary muscle soreness such as might occur after exercising. To minimize these risks you will be asked to warm-up before the tasks and tell us if you are aware of any history of skin-reaction to tape, history of musculoskeletal injury, cardiovascular limitations. During prolonged testing, you may feel dizzy or light-headed, and there is a small risk that you could faint. To minimize these risks, you will be asked several times if you are experiencing such symptoms; if so, you will be asked to walk around or sit down as appropriate. In addition, hunger may exacerbate such risks, so you will be asked to not come to experimental sessions hungry, and small snacks will be made available should you become hungry. If you seek medical/counseling services as a result of participation in this study, any expenses accrued are your responsibility and not that of the investigators, research team, or Virginia Tech.

IV. Benefits

You will receive no direct benefits from participating in this study. By participating, however, you will help to increase our understanding of potential mechanisms of LBP among lower-limb amputees. Understanding the causes of LBP in this population will aid in controlling the prevalence of the disorder. Further, results may have long-term implications for improvements in prosthetic design, post-amputation rehabilitation methods, as well as facilitate communication between patients and prosthetists/caregivers. We do not guarantee or promise that you will receive any of these benefits, and no promise of benefits has been made to encourage your participation.

V. Extent of Anonymity and Confidentiality

Experimental data collected from your participation will be coded and matched to this consent form so only members of the research team can determine your identity. Your identity will not be divulged to unauthorized people or agencies. Sometimes it is necessary for an investigator to break confidentiality if a significant health or safety concern is perceived or the participant is believed to be a threat to himself/herself or others.

VI. Compensation

You will be paid $10/hour for your participation in this study (Note: amputees will be paid $50/hour).

VII. Freedom to Withdraw

You are free to withdraw from this study at any time without penalty. If you choose to withdraw, you will be partially compensated for the portion of the project completed. You are free not to answer any questions or respond to experimental situations that you choose without penalty. There may be circumstances under which the investigator may determine that you should not continue as a subject, and if so you will be compensated for the portion of the project completed.

VIII. Approval of Research

This research project has been approved, as required, by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute and State University.

IX. Subject's Responsibilities

I voluntarily agree to participate in this study. I have the following responsibilities:

- Inform the investigators of all medical conditions that may influence performance or risk
- Comply to the best of my ability with the experimental and safety instructions
- Inform the investigators of any physical/mental discomfort resulting from the experimental protocol
- Inform the investigators of any feelings of dizziness, light-headedness, or fainting

X. Subject's Permission

I have read and understand the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent:

Subject Name (Print): _____

Subject Signature: _____ Date: _____

Should I have any pertinent questions about this research or its conduct, research subjects' rights, and whom to contact in the event of a research-related injury or other adverse outcome, I may contact:

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Subjects must be given a complete copy (or duplicate original) of the signed Informed Consent