Exercise-Induced Low Back Pain and Neuromuscular Control of the Spine - Experimentation and Simulation

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Abstract

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Low back pain (LBP) is associated with altered neuromuscular control of the trunk, as well as impaired performance during functional tasks highly dependent upon trunk neuromuscular control. Comparing measurements between individuals with and without LBP does not distinguish whether the LBP individual exhibits altered neuromuscular control only while experiencing LBP versus at all times. Additional insight was gained on the relationship between trunk neuromuscular control and LBP by investigating individuals who experience recurrent exercise-induced LBP (eiLBP). To differentiate the effects of LBP from individual differences, comparisons were made between episodes of pain and no pain within eiLBP individuals, and between eiLBP individuals while pain free and a group of healthy controls. Three studies were completed based on repeated measurements from both eiLBP and healthy individuals. Study 1 investigated effects of eiLBP on fundamental measures of neuromuscular control, including intrinsic trunk stiffness and the paraspinal reflex delay using a series of pseudo-random position perturbations. eiLBP individuals exhibited increased stiffness compared to healthy controls unaffected by the presence of pain, and increased reflex delays concurrent only with pain. Study 2 investigated effects of eiLBP on seated sway during a functional task involving maintaining balance. Seat and trunk kinematics were obtained while participants balanced on a wobble chair at two difficulty levels. eiLBP individuals exhibited impaired seat measures at all times, with altered trunk measures only while in pain and when the task was not challenging. Study 3 investigated effects of eiLBP on the underlying control of seated sway using a model of wobble chair balance. Quantified neuromuscular control indicated increases in proportional and noise gains for a challenging level compared to an easy level, more so for eiLBP individuals compared to controls and while experiencing pain compared to pain free. Overall, fundamental measures, seated sway measures, and identified control parameters using a model of wobble chair balance were all affected by the presence of pain within the eiLBP individuals and/or the eiLBP individuals compared to healthy controls. Therefore, this study shows that some characteristics appear to be inherent to the LBP individual, while others are only concurrent with pain.

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1 Overview

1.1 Motivation

Low back pain (LBP) is a significant health and economic problem in the United States and around the world,[1] accounting for 20% of occupational injuries and 40% of worker compensation claims.[2] Outside the workplace, 60-80% of all adults experience LBP at some point in their lifetime,[3] with more than $90 billion estimated in the United States in 1998 for associated health care expenditures.[4] One of the reasons for an increased risk of LBP could be related to the neuromuscular control of the trunk. An inappropriate response to trunk loading due to altered neuromuscular control could cause abnormal stress and strain distributions within the trunk, which are suggested as the proximate cause of LBP or injury.[5,6] Additionally, when performing a functional task, such as maintaining balance, the neuromuscular control system must meet the requirements of stability to avoid injury.[7-10]

LBP is associated with altered fundamental measures of neuromuscular control of the trunk,[10-13] as well as impaired performance during functional tasks that are highly dependent upon the neuromuscular control of the trunk.[10] However, studies that have found an association between LBP and neuromuscular control of the trunk compared measurements between individuals with and without LBP. This experimental design is limited because it does not distinguish whether the LBP individual exhibits altered neuromuscular control only while experiencing LBP, or at all times. Additional insight can be gained on the relationship between trunk neuromuscular control and LBP by investigating individuals who experience recurrent exercise-induced LBP (eiLB). For example, comparing measurements between episodes of pain (post-exercise) and no pain (post-recovery) within eiLBP individuals, as well as between eiLBP individuals while pain free and a group of healthy controls, could help differentiate the effects of LBP from individual differences.

1.2 Dissertation Organization

Three studies were conducted to increase understanding of the association between neuromuscular control of the trunk and LBP, each utilizing this advantage of repeated measurements on both eiLBP and healthy individuals. The remaining portion of this document is organized into six chapters, including literature reviews, three studies, and a summary.

Chapter 2 is a literature review on low back pain and neuromuscular control, for the purposes of explaining fundamental measures and functional performance of the trunk. Chapter 3 covers the process of and literature findings for modeling human movement, for both standing and seated postural control.

Chapter 4 is Study 1, "Effects of Exercise-Induced Low Back Pain on Intrinsic Trunk Stiffness and the Paraspinal Reflex Response." Intrinsic trunk stiffness and paraspinal reflex delay were determined using a series of pseudo-random position perturbations. Results increase understanding of the association between LBP and fundamental measures neuromuscular control of the trunk.

Chapter 5 is Study 2, "Effects of Exercise-Induced Low Back Pain on Seated Sway." Performance during wobble chair balance was investigated with measures of seated sway (seat and trunk angle variability and seat stability) for two difficulty levels. Results increase understanding of the association between LBP and performance of a functional task highly dependent upon neuromuscular control of the trunk.

Chapter 6 is Study 3, "Effects of Exercise-Induced Low Back Pain on the Neuromuscular Control of Seated Sway, a Model of Wobble Chair Balance." The model provides a new tool for quantifying control of the trunk during wobble chair balance, therefore distinguishing effects of pain within the eiLBP individual and between eiLBP individuals and healthy controls.

Chapter 7 is a summary chapter. It covers the three main studies in this document, their implications, and future work.

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2 Low Back Pain and Neuromuscular Control

This chapter covers literature findings regarding low back pain (LBP). It also covers multiple associations between LBP and fundamental and functional measures of neuromuscular control. Experimental considerations are also described for determining fundamental measures of neuromuscular control and quantifying performance of a functional task such as standing or seated postural control.

2.1 Low Back Pain

LBP is a significant health and economic problem in the United States and around the world,[1] accounting for 20% of occupational injuries and 40% of worker compensation claims.[2] Outside the workplace, 60-80% of all adults experience LBP at some point in their lifetime,[3] with more than $90 billion estimated in the United States in 1998 for associated health care expenditures.[4] LBP is associated with altered neuromuscular control of the trunk.[5-8] An inappropriate or insufficient response to trunk loading due to altered neuromuscular control could cause abnormal stress and strain distributions within the trunk, which are suggested as the proximate cause of LBP or injury.[9,10]

2.2 Low Back Pain and Fundamental Characteristics of Neuromuscular Control

Appropriate neuromuscular control is necessary for stability during multiple tasks, ranging from maintaining a neutral posture to correcting for postural or trunk disturbances. Neuromuscular control consists of fundamental characteristics such as intrinsic passive tissue properties, active muscle contribution, and reflex behavior.[11-14] LBP is associated with altered measures of these fundamental characteristics of trunk neuromuscular control. For example, individuals with chronic LBP are reported to have delayed reflex latencies following the sudden release of a trunk extension load.[5-7] For the same type of trunk perturbation, individuals who experience recurrent episodes of acute LBP exhibit increased effective trunk stiffness and damping,[8] yet this was determined while participants were not in pain. Effective is a term referring to a combination of reflexive and intrinsic contributions, with intrinsic describing the current state of active and

passive properties. When quantifying and interpreting neuromuscular control during or in response to a trunk perturbation, understanding each of these fundamental characteristics is essential.

2.2.1 Passive Tissue Properties

Passive tissue properties contribute to trunk stability,[11] and inadequate support may contribute to LBP. Passive tissues in the low back consist of spinal ligaments, intervertebral discs, vertebrae, and the passive components of muscle-tendon units.[15] Each of these provides resistance to movement, with a reduction in passive stiffness being related to trunk instability[16-18] Differences in passive trunk stiffness likely involve the "neutral zone" of the spine, i.e. the region of inter-segmental motion with minimal passive resistance.[19] A larger neutral zone, or decreased passive resistance, would cause greater unsupported trunk movement in response to a trunk disturbance, and could lead to LBP or injury. Alternatively, the individual with decreased passive stiffness could be exerting increased active effort to remain upright even in just a neutral posture. This was shown in an exaggerated sense where prolonged lumbar flexion resulted in the systematic transfer of an extension moment from passive tissues to active muscles.[20]

2.2.2 Active Muscle Contribution

Active muscle contribution aids in the control of trunk stability,[11,21] with increased trunk stiffness resulting from greater trunk exertion effort[12,22] and/or muscle co-contraction.[23] Decreased strength or an inability to actively stiffen the spine could restrict the stability of the spine and lead to LBP or injury. Fatigue studies show a reduction in force generating capacity, and thereby a reduction in active muscle stiffness, resulting in increased co-contraction.[24,25] Co-activation is believed to be compensatory for stability maintenance, but it also increases spinal compression.[26] Individuals with chronic LBP exhibit higher baseline EMG levels than healthy individuals in a normal upright stance,[27] and increased trunk muscle activity (i.e. ratios of antagonist to agonist and lumbar to thoracic erector spinae) during slow trunk motions about a neutral posture and isometric voluntary contractions while sitting.[28] It has been hypothesized that individuals with LBP compensate by increasing muscle stiffness to actively stabilize the spine, but with the

undesirable effect of increasing neuromuscular fatigue in the paraspinal musculature. Therefore, characteristics seen in LBP patients' such as decreased reflex magnitudes[27] and poorer performance in trunk balancing tasks,[6] as well as delayed or longer reflex latencies,[5,6,27] could be influenced by fatigue from constant active muscle activation for stabilization.

2.2.3 Paraspinal Reflex Response

Paraspinal reflexes are one of many mechanisms that contribute to the control of trunk stability,[11] and altered reflexes may contribute to LBP. The paraspinal muscle reflex acts as feedback response to kinetic and kinematic disturbances of the trunk.[29] Longer paraspinal reflex latencies have been reported in LBP patients, compared to healthy controls, after sudden loading[27] and unloading[5-7] of the trunk that resulted in trunk flexion. Similarly, patients with unilateral LBP exhibit longer erector spinae reflex latencies on the painful side than on the non-painful side.[27] Longer paraspinal muscle reflex latencies in LBP patients have also been correlated with impaired postural control of the trunk during unstable sitting.[6]

2.3 Low Back Pain Risk and Fundamental Characteristics of Neuromuscular Control

To increase understanding of LBP and neuromuscular control of the trunk, several studies have investigated fundamental characteristics of neuromuscular control for populations and conditions which are known to increase the risk of LBP. Several risk factors are associated with LBP. For example, gender has been identified as a risk factor associate with LBP. Females experience LBP or injury more than twice as frequently as males in occupational settings that involve lifting.[30-32] Females are also more likely to be limited in their activities due to LBP one year following an initial incident.[33] Two additional risk factors for work-related LBP include lifting loads and flexed trunk postures.[34-36] In fact, 66% of LBP injury from overexertion is due to lifting (i.e., spinal loads while in flexed trunk postures).[35] It is hypothesized that there are particular thresholds of lifting repetition and exertion, beyond which the risk of LBP or injury increases.[34] Asymmetries during lifting, such as lateral bending, uneven loads, and axial twisting, introduce even greater load to the spine, [37] also increasing the risk of LBP.

Active trunk extension exertions have been shown to increase effective trunk stiffness and reflex gain in response to force-input perturbations, while trunk angle increased effective stiffness and decreased reflex gain.[38] Extension exertions actively stiffened the spine, and authors state that increases in reflexes could be due to increased baseline electromyography (EMG) during exertion.[38] Effects of trunk angle were attributed to increased passive tissue contributions to stiffness, lessening the reflex response. On the other hand, following an extended period of flexion-relaxation, reflexes show a trend toward increased gain.[39] This is likely due to muscle and tissue creep, which temporarily decreases passive stiffness and is counteracted by an increase in active stiffness.[20]

Gender has also been explored for changes in neuromuscular control of the trunk. There were no differences between males and females in reflex latency[39] or effective trunk stiffness,[12,38] following force perturbations. However, the absolute force inputs caused higher trunk flexion velocities and a larger reflex response in the smaller massed females. When accounting for individual kinematics post-experiment (i.e. normalizing by trunk flexion velocity), it has been shown that females had quicker reflex latencies than males.[40] Furthermore, a subsequent trunk study applying position perturbations resulted in decreased intrinsic trunk stiffness in females, with lesser abilities to increase stiffness during active exertions than males.[41] Females also exhibit decreased passive stiffness in the cervical spine,[42] suggesting that shorter reflexes in the posterior neck[43] may be due to active tissues stretching sooner when perturbed. Gender risk for LBP may relate to neuromuscular control of the spine, but there are also structural differences between genders. For example, average lumbar and trunk angles were significantly more flexed for males than for females,[44] which could explain the decreased passive stiffness and quicker/larger reflexes seen in females. This could extend to prolonged sitting tasks, such that males and females may be exposed to different loading patterns and thus experience different pain generating pathways.[44]

2.4 Experimental Considerations for determining Fundamental Characteristics of Neuromuscular Control

2.4.1 Advantages of Position-Controlled Perturbations and Percent-Effort Preloads

While relatively easy to implement, the use of force perturbations to characterize neuromuscular control of the trunk can potentially introduce confounding effects on reflexes and stiffness measurements. For example, as is seen in gender studies,[12,38] applying an absolute force input results in larger kinematic responses females, who generally have smaller mass than males. This is problematic because reflexes are dependent upon muscle lengthening velocity,[13,45,46] and the input could have potentially influenced their larger reflex response. To address this problem, some investigators adjusted the applied load to be a fixed percentage of participants' trunk and head masses[47] while others normalized the applied impulse force by trunk flexion velocity[40] prior to characterization of trunk neuromuscular control. However, these strategies do not ensure identical trunk kinematics due to other characteristics of the spine involved in the response. For example, the former neglected differences due to damping resistance of viscoelastic tissues, and the latter did not account for inertial differences in impulse response. Therefore, the remaining gender difference following normalization could still be confounded.[40] Another limitation of using force perturbations is the inability to separate intrinsic and reflex responses, since force perturbations and the resulting trunk response typically occur over longer durations than paraspinal reflex latencies.[29,48] This is a clear limitation if the intrinsic and reflex responses differ between various risk factors. In addition to suspect inputs, the condition of an absolute preload (i.e., the magnitude of trunk extension exertion quantified by an absolute force value) potentially puts individuals at different effort levels due to strength differences in males and females. In fact, preloads themselves could mask gender differences in latency by stiffening the spine,[22] resulting in a reduced demand for quicker reflexes.

These limitations lead to the practical application of position-controlled perturbations[49-52] and percent-effort preloads for trunk response analyses. With position perturbations, a motor is controlled to move the trunk a prescribed distance over a prescribed time. This method has two

immediate benefits. First, it has the potential to eliminate variations in trunk kinematics across research participants that can confound both stiffness and reflex measurements. Second, trunk movement can be completed prior to typical paraspinal reflex latencies, allowing the intrinsic response to be separated from the reflex response. With percent effort preloads, participants do not recruit such large differences in muscle activation for each trunk extension exertion.

2.4.2 2-DOF Dynamic Linear Modeling

For all perturbation types, the dynamical system between the individual and the machine needs to be understood and accounted for. Some system identification methods predict dynamic properties of the trunk in response to force input perturbations.[12,46,53] This process fits a predicted force curve to the recorded force input during the perturbation, as well as measured displacements of the trunk. Similar methods for fitting to a predicted force curve can be utilized when the pre-determined (and recorded) displacement is the input and the measured force response transmits between the person and a motor through a rigid attachment. With position-control, the input displacement could theoretically be measured at the trunk or with a conversion of the motor's rotational displacement. However, the trunk properties resulting from curve-fitting are highly dependent upon the rigidity of the system. Since soft tissue exists between the contact surface of the posterior harness and the spine, the trunk movement lags that of the motor input.[51] Therefore, a 2-DOF representation of the trunk and the harness/rod connection could aid in a more accurate differentiation of the trunk's force response properties.[41,49-52]

2.5 Performance of a Functional Task is dependent on Neuromuscular Control

When performing a functional task, the neuromuscular control system must meet the requirements of stability to perform well, and in some cases avoid injury.[6,11,54,55] Fundamental characteristics of neuromuscular control are involved and directly relate (not necessarily equally) to multiple components including (but not limited to) proprioceptive input, central nervous system processing, and coordinated muscle activation. This section describes commonly implemented sway measures for quantifying sway performance, presents findings on standing

and seated postural control (in general and associated with LBP), and presents LBP associations for other measures and functional tasks. Postural control referred to here is the neuromuscular control during a specified task.

2.5.1 Sway Measures

Common measures conducted on spontaneous sway for analyzing balance performance involve utilizing the center of pressure (COP) underneath the feet. COP measures include both time and frequency domain variability. Prieto et al (1996) and Maurer and Peterka (2005) describe multiple measures. Time domain measures of the COP include mean distance (MeanD), root mean squared distance (RmsD), maximum distance (MaxD), and mean velocity (MeanV). Frequency domain measures of the COP include power and a mean (MeanF), 95% power frequency (95%PF), and centroidal frequency (CentrF) of the power spectrum. Increased sway measures related to displacements and velocities suggest impaired postural control.

In addition to time and frequency domain measures, balance performance can also be analyzed with non-linear stability measures. For example, the stabilogram diffusion function (SDF) can be used to examine the stability diffusion exponent, which demonstrates bi-linear behavior with regions representing open-loop and closed-loop systems.[56,57] For this analysis, the variance is computed from the mean squared distances traveled by the signal within each time interval of 1 sample to 10 seconds. Therefore, measures of short-term (D_S) and long-term (D_L) coefficients, as well as short-term (H_S) and long-term (H_L) exponents, describe these regions, separated by a critical point in time (CT_t) at which variance has a particular amplitude (CT_a).[56] In each region, coefficients are calculated as the slope of the best fit line of the linear-linear plot of variance versus time interval, while exponents were the slopes of the best fit line of the log-log plot of variance versus time interval. Short-term measures depict stability in terms of open-loop feedback and sway that is more prone to persist in the direction is it currently headed, while long-term measures depict stability in terms of close-loop feedback and sway that is more prone to anti-persist from the direction is it currently headed.[56,57] Larger SDF measures imply decreased stability.

2.5.2 Postural Control (Standing and Seated)

Numerous studies have investigated balance performance by assessing standing sway. Reduced performance has been observed in the elderly, characterized by increased COP velocity[58-66] and increased short-term SDF analyses.[62] Individuals with known postural deficits such as Parkinson's disease[67-69] and peripheral neuropathy[70-72] also exhibit increased sway. In terms of LBP, individuals with chronic LBP exhibited increased COP measures of RMS displacement, velocity, and frequency compared to healthy controls during quiet standing after a prolonged standing task, suggesting an inability to generate healthy responses to challenging tasks.[73]

In an effort to evaluate how the neuromuscular control of the lumbar trunk performs during a functional task, numerous studies have examined balance performance in seated sway while subjects balance on an unstable seat. Maintaining seated balance is highly dependent upon control of the trunk/spine. As with standing sway, increased measures of seated sway imply impaired neuromuscular control. This is further supported by seated sway measures (MaxD, RmsD, path length, and SDF coefficients and critical points) positively correlating with age and body weight.[74] Increased seated sway measures are also associated with LBP. For example, Radebold et al (2001) reported increased MeanV, increased SDF coefficients, and longer SDF critical points in individuals with chronic LBP compared to a healthy control group. [6] These findings could be due to increased trunk stiffness in LBP individuals since MeanV also increases with increased active trunk stiffness,[75] while a lumbosacral orthosis (i.e. increased passive trunk stiffness) presents no difference.[75,76] It is important to note that the sway measures also highly correlate with anthropometric measurements and reduced stance boundaries.[62] Therefore, careful planning must be taken when performing and interpreting such postural analyses.

A custom-made wobble chair that accounts for differences in subject anthropometry has been developed more recently to quantify postural control of the trunk during seated balance performance tasks.[77,78] Similar to the process for determining kinetic standing and seated sway measures, the wobble chair apparatus has been implemented to quantify kinematic seated sway measures, using variability (RMS and 95% ellipse area) and non-linear stability (SDF and Lyapunov) analyses on the seat angle of the wobble chair.[77] The wobble chair has previously

been used to examine postural control of the trunk during flexion and extension exertions,[77] where it was found that postural control of the trunk was more poor during flexion versus extension exertions. Seated postural control was also examined with the wobble chair to determine effects of whole body vibration (WBV), another implied risk factor for LBP, on seated postural control .[78] It was found that all sway measures increased with WBV, suggesting WBV impairs postural control during unstable sitting.

2.5.3 LBP associations with Alternative Measures of Postural Control

Several other measures have identified differences in neuromuscular control between LBP and healthy individuals. For example, for three tasks including sitting, stable support standing, and unstable support standing, the ratio of ankle muscle proprioceptive inputs versus back muscles proprioceptive inputs were higher in a non-specific LBP group, suggesting less ability to rely on back muscle proprioceptive inputs for postural control.[79] For seven postural tasks involving manipulation of visual, vestibular, and proprioceptive input, as well as body orientation, the RmsD increased in the medial-lateral direction for chronic LBP individuals with removal of vision, and especially when combined with increased task complexity.[80] Individuals with recurrent LBP recruited predominantly ankle control versus the low back during postural sway, and lacked the adaptation to increase back musculature with acute back muscle fatigue, compared to controls.[81] This suggests an altered neuromuscular control strategy compensating for decreased function while in pain.

In an effort to determine the source of LBP differences in neuromuscular control, studies have investigated changes in neuromuscular control after experimentally-inducing LBP. For example, decreased trunk motion was observed while walking[82] and during quiet standing[83] as a result of injections of a saline solution into the lumbar longissimus muscle to induce pain. Noxious heat applied on the skin can also induce LBP, and decreased lumbar spine movement was observed with reference to the hip while performing trunk flexion.[84]

2.6 Conclusions

LBP is associated with multiple alterations of both fundamental measures of trunk neuromuscular control, as well as functional measures during a task dependent upon trunk neuromuscular control.[6] However, studies that recognize an association between LBP and neuromuscular control of the trunk compared measurements between individuals with and without LBP. Additional insight on whether LBP individuals exhibit altered neuromuscular control at all times, or only while experiencing LBP, is needed. This could be accomplished by investigating individuals who experience recurrent exercise-induced LBP (eiLB), comparing their measurements between episodes of pain and no pain, as well as while pain free with a group of healthy controls.

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3 Dynamics and Control of Human Movement

This chapter covers the process and relevant literature findings for modeling human movement. Starting with an overview of forward dynamics, sections cover inverted pendulum modeling, feedback control, simulated annealing optimization, and multiple existing models of both standing and seated postural control.

3.1 Dynamic Modeling

There are two common biomechanics processes for analyzing the human body. The process of inverse dynamics determines internal forces and moments (kinetics) from measured motion (kinematics) and possibly external kinetics. In this case, human movement and ground reaction forces can be measured directly to determine and compare, for example, the knee joint power while walking. It is common, however for a research question to involve determining how a change in knee joint power would affect the performance of a particular task. The process of forward dynamics determines kinematics using known internal and external kinetics as seen in Figure 3-1. This technique is advantageous because it allows for kinetics to be altered within model simulations in order to obtain desired kinematics. This section walks through how the kinetic inputs of forward dynamic modeling are initially defined and integrated with equations of motion, as well as how various control designs influence the change in kinematics.

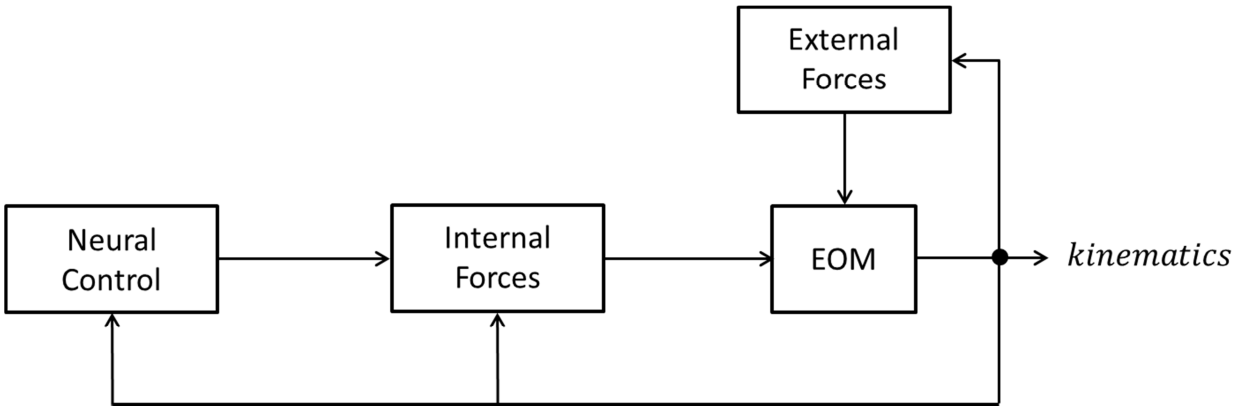

Figure 3-1 Schematic of Forward Dynamics (kinematics from kinetics).
Inverse dynamics would be the opposing process (kinetics from kinematics).

Inverted pendulum models are commonly used to represent human balance.[1-4] With free body diagrams, internal and external forces acting on a body can be clearly defined, as shown in the 1-segment example below (Figure 3-2) representing sagittal upright stance controlled by a torque (T) at the ankle (A). The equation of motion for the segment is shown to the left, where the center of mass (COM) of the segment has a moment of inertia (I), a deviation from vertical (θ), a radial distance (r) to the ankle (A), a mass (m), and a gravitational acceleration (g).

$$\ddot{\theta}_{COM}(t) = \frac{T_A(t) - m_{COM}g * r_{COM}\, sin(\theta(t))}{I}$$

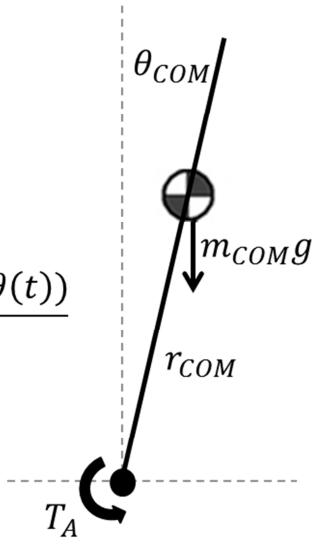

Figure 3-2 Schematic of a 1-segment inverted pendulum representing sagittal upright stance controlled at the ankle. Internal and external forces and moments acting on the system are shown.

Defining internal and external forces and moments can be extended to describe dynamics of multiple (body) segments with various inputs. Lagrange dynamics are typically used to derive the equations of motion for these more complex systems.[5,6] The Lagrangian of a mechanism is the difference between the kinetic energy of a system and its potential energy. Equation 3.1 can be used to acquire the Lagrangian equations of motion.

$$\frac{d}{dt}\left(\frac{\partial L}{\partial \dot{q}_i}\right) - \frac{\partial L}{\partial q_i} = Q_i \quad i = 1, \ldots n \tag{3.1}$$

Here, L is the Lagrangian, q_i is the *ith* generalized coordinate or degree of freedom of the system, and Q_i represents the *ith* generalized force and moment acting on, or in, the system. For a system of n degrees of freedom (or generalized coordinates), n equations describe the coupled motion.

Once the equations of motion are fully defined, the current coordinates, velocities, and accelerations are used to determine the subsequent motion for each time step. Integration can be solved numerically using an iterative Runge-Kutta 4th order procedure.[7] This can be accomplished via custom code or built-in MATLAB (MathWorks, Natick, MA, USA) solvers such as the Dormand-Prince algorithm (ode5).

3.2 Feedback Control

One type of control used in forward dynamic simulations, and the primary interest of this dissertation, is feedback control. A closed-loop feedback system (Figure 3-3) discretely signals for a desired output based on the error of a previous output, instantaneously and/or following a specified delay.

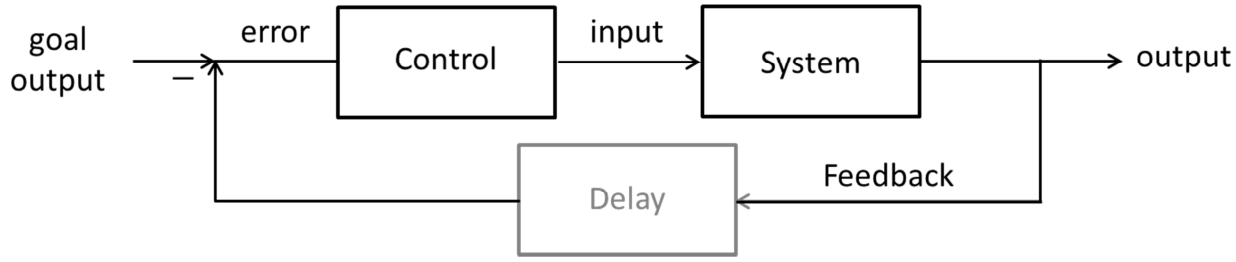

Figure 3-3 Schematic of a closed loop feedback system with an optional delay in grey.

In control theory, a transfer function directly relates the Laplace transform of an output to the Laplace transform of an input. For a dynamic system with feedback, the "control" transfer function precedes (i.e. drives) a dynamic transfer function on the system. Equation 3.2 describes this feedback system.

$$Y(s) = \frac{C(s)G(s)}{1+C(s)G(s)}X(s) \tag{3.2}$$

Recalling the inverted pendulum model, G(s) would produce the motion of the dynamics, with C(s) as the added controller. PID refers to a method of feedback control that uses time-invariant proportional, integral, and derivative gains to correct for the deviations from a desired output, or error. For a PID feedback controller, C(s) would follow Equation 3.3. Therefore, a closed-loop PID feedback system would fit into Figure 3-3 as shown in Figure 3-4.

$$C(s) = P + \frac{I}{s} + Ds \tag{3.3}$$

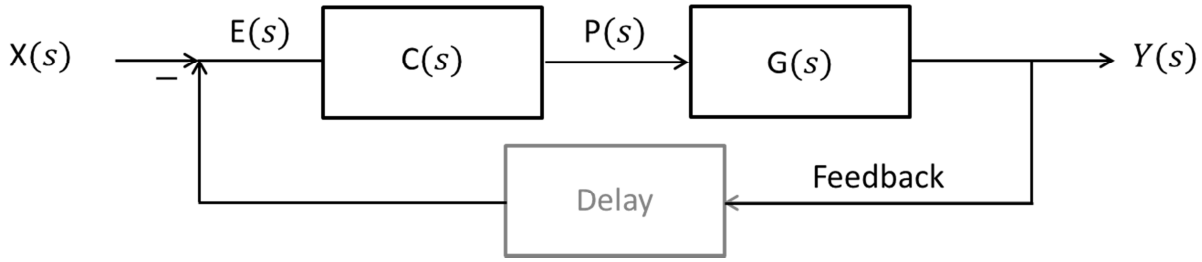

Figure 3-4 Schematic of a delayed PID feedback controller. For the purposes of forward dynamics, the dynamic EOM transfer function G(s) relates kinematic output Y(s) to kinetic input P(s). The PID control transfer function C(s) relates kinetic input P(s) to the current model error E(s), determined as desired kinematic output X(s) minus actual kinematic output Y(s).

3.3 Optimization

Appropriate PID gains are necessary for functional feedback control. When these are unknown, optimization techniques can be implemented to determine ideal control. An objective function is created to describe the error of a system, based on the desired kinematics in forward dynamic modeling. Error is minimized to the specifications of the programmer. An objective function can take the form of a full time series of performance, summary measures of a time series, or an overall performance goal event to be achieved. The latter does not require the existence of actual experimental data, but great understanding of the task is necessary to define the cost function.

There are multiple local optimization techniques built into software programs, e.g. fminsearch in MATLAB, that minimize an objective function well with a "good" initial guesses. Simulated annealing, however, is a technique capable of randomly searching outside local optimums with the aim of finding a global optimum.[8-10]

Simulated annealing is a computational method that iteratively optimizes a problem to minimize a solution with regard to a given measure. The goal is global optimization within a large search space to find an acceptable solution in a fixed amount of time rather than conducting an exhaustive search of the entire space. This does not necessarily ensure accurate global optimization, but its probabilistic nature aims to avoid getting stuck in a local optimum by allowing increased solutions to be accepted or rejected based on the difference from a previous function and a decreasing global value, termed temperature. When the temperature is larger, this process is fairly random, but decreasing solutions are more frequently accepted as temperature decreases.

3.4 Models of Postural Control

Multiple feedback models have aimed to derive the postural control necessary during performance of a functional task, utilizing active and/or passive control, with proportional, integrative, and/or derivative gains, within optimizations for local or ideally global minima. A few of these are presented here.

3.4.1 Standing Postural Control

Qu and Nussbaum (2011) created a 3-dimensional, 2-segment inverted pendulum model of quiet standing.[11] Specifically, COP based measures of quiet standing were simulated in both the anterior-posterior and medial-lateral directions using torque actuators at the ankle and hip. Model parameters were optimized using measures of standing sway from human subjects. An "optimal control" procedure included a weighted cost function where certain measures were more influential on the error, random disturbance gains, and sensory delays. A genetic algorithm and simulated annealing were used to identify model parameters for the human subjects. This

model demonstrated the ability of forward dynamics simulations to accurately simulate 3-dimensional standing sway behaviors.

Maurer and Peterka (2005) created a 2-dimentional, 1-segment inverted pendulum model of standing postural control.[4] Specifically, COP based measures of quiet standing were simulated in the anterior-posterior direction using a torque actuator at the ankle. Model parameters included a PID feedback controller based on the whole system COM and noise gain. The cost function included common standing sway measures from elderly and young human subjects. The MATLAB optimization procedure *fminsearch* was implemented to identify the appropriate model parameters for simulating increased sway in the elderly versus the young. Using the model of standing balance, modest increases in stiffness and damping and a fairly large increase in noise level were found with aging. Active mechanisms were also found to be the main contributors to upright stance, similar to other models. [12-16]

Maurer and Peterka (2006) also created a 2-dimentional, 1-segment inverted pendulum model of postural responses following a perturbation.[3] COM and COP excursions were simulated from ankle torque control. Model parameters incorporated ankle proprioceptors, vestibular sensors and plantar pressure sensors. The cost function included gain and phase of the postural responses from normal subjects and those with vestibular dysfunction. The MATLAB optimization procedure *fminsearch* was implemented to identify the appropriate model parameters for simulating both normal subject and vestibular loss patient postural responses. Normal subjects altered their postural strategy by strongly weighting feedback from plantar somatosensory force sensors.

3.4.2 Seated Postural Control

Tanaka et al. (2010) created a 2-dimensional, 2-segment inverted pendulum model of seated postural control.[17] Specifically, trajectories based on an initial state were simulated from low back (L_4L_5) torque control. Model parameters included a PD feedback controller based on the whole-system COM. Optimizations utilized finite time Lyapunov exponent analyses determined from seated sway measures in human subjects. A boundary between regions of stability and

failure (i.e., falling) was determined, as well as an equilibrium manifold, for the task of wobble chair balance.

Kamper et al. (2000) created a 10-segment chain of active trunk feedback using a Working Model software package to simulate the postural control of seated wheelchair individuals.[18] Full state feedback was optimized using measured attempts to remain stable during the application of significant disturbance moments, similar to those experienced during braking in a vehicle, for both able-bodied and spinal cord-injured subjects. While subjects exhibited more complex control schemes, the model was able to simulate overall stability. Therefore, it is believed that the model could prove beneficial to future research examining the effects of various restraints on stability.

3.5 Chapter 3 References

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4 Effects of Exercise-Induced Low Back Pain on Intrinsic Trunk Stiffness and the Paraspinal Reflex Response

4.1 Introduction

Low back pain (LBP) is a significant health and economic problem in the United States and around the world.[1] An estimated 60-80% of all adults experience LBP at some point in their lifetime,[2] and a 2004 study reported that annual health care costs in the US associated with LBP exceeded $90 billion.[3] Abnormal stress and strain distributions within the trunk have been suggested as the proximate cause of LBP or injury.[4,5] These distributions could result from altered neuromuscular control or an inappropriate or insufficient response to trunk loading.

Numerous descriptive studies have reported differences in neuromuscular control of the trunk between individuals with and without LBP. Individuals with chronic LBP exhibit an increase in paraspinal reflex delay,[6-8] increased sway measures while sitting on an unstable seat,[7] and increased trunk muscle activity (i.e. ratios of antagonist to agonist and lumbar to thoracic erector spinae) during slow trunk motions about a neutral posture and isometric voluntary contractions while sitting.[9] Individuals who experience recurrent episodes of acute LBP exhibit increased effective trunk stiffness, yet while not in pain, as measured from trunk motion following the sudden release of a trunk extension load.[10] These studies provide a rich source of information on the biomechanical factors associated with LBP. However, they are unable to determine whether the measured differences in neuromuscular control were due to LBP individuals exhibiting altered control, or from the effects of pain itself on neuromuscular control.

In an effort to determine the source of these differences in neuromuscular control, other studies have investigated changes in neuromuscular control after experimentally-inducing LBP. Experimentally-induced LBP, caused by injecting a saline solution into the lumbar longissimus muscle, decreased trunk motion while walking[11] and during quiet standing[12] and altered trunk muscle activation (increased onsets and decreased amplitudes) during a postural task involving rapid arm movements.[13] In another study, noxious heat applied on the skin to induce LBP decreased lumbar spine movement with reference to the hip while performing trunk flexion.[14]

Increased muscle activation and decreased motion, which would be consistent with increased trunk stiffness, is thought to be a strategic response of the central nervous system to prevent pain and further injury.[9,12,14,15] Studies that recognize an association between LBP and neuromuscular control of the trunk compared measurements between individuals with and without LBP. This experimental design is limited because it does not distinguish whether the LBP individual exhibits altered neuromuscular control only while experiencing LBP, or also when pain free. Additional insight can be gained on the relationship between trunk neuromuscular control and LBP by investigating individuals who experience recurrent exercise-induced LBP (eiLB). Comparing fundamental measures of neuromuscular control between episodes of pain (post-exercise) and no pain (post-recovery) within eiLBP individuals, as well as between eiLBP individuals while pain free and a group of healthy controls, could help differentiate the effects of LBP from individual differences.

Therefore, the purpose of this study was to compare trunk neuromuscular control between episodes of pain and no pain among individuals who experience recurrent acute eiLBP. In addition, trunk neuromuscular control was compared with individuals without a history of LBP. Our general expectations were that neuromuscular control would differ between groups when the eiLBP group was not experiencing pain, and that this difference would be exaggerated when the eiLBP group was experiencing pain. The following three hypotheses were tested: 1) neuromuscular control would differ between a healthy control group and an eiLBP group when not experiencing pain, 2) exercise would induce LBP and change neuromuscular control in the eiLBP group, and 3) exercise would neither induce LBP nor change neuromuscular control in the control group. Trunk neuromuscular control was quantified using measures of intrinsic trunk stiffness and paraspinal reflex delay, and differences/changes in either measure constituted a difference/change in neuromuscular control.

4.2 Methods

Participants included 17 male members of the Virginia Tech Triathlon Club. Females were not included due to previous associations of gender with intrinsic stiffness[16] and reflex delay.[17] Eight of these participants experience acute eiLBP (mean height ± SD: 1.83 ± 4.7 m; mass: 72.9

± 2.7 kg; age: 20.7 ± 1.0 yr), and nine controls report no history of LBP (height: 1.79 ± 3.2 m; mass: 70.8 ± 7.3 kg; age: 20.4 ± 1.6 yr). To minimize effects of age and weight, participants were to have an age of 18-49 and a body mass index less than 30 kg/m^2. eiLBP participants were required to pass a screening by a chiropractic physician that evaluated their ability to complete the experiment without further injury, and ensured agreement with specific inclusion and exclusion criteria. Inclusion criteria included acute eiLBP for at least than six months, and exclusion criteria included any neurological deficits, vestibular or visual disorders, major structural deformities of the spine, genetic spinal disorders, spinal surgery within the past five years, or spinal mechanical implants. This study was approved by the Virginia Tech Institutional Review Board, and written consent was obtained from all participants prior to participation.

Neuromuscular control was characterized for all participants during two experimental sessions. Two sessions were determined based on the fact that specific days of exercise, acute eiLBP, and subsided LBP were known to occur in particular triathlon individuals. The first session was 1-2 days following a weekend race (post-exercise), when the eiLBP group was experiencing an episode of LBP. The second session was 4-5 days following the weekend race (post-recovery) when the LBP had subsided (see below). The control group participated in experimental sessions on the same days following a race and did not experience eiLBP. A visual analog scale[18] (VAS) was used for rating LBP during each session. The VAS quantified LBP on a numerical scale with text descriptors from 0 (none) to 10 (agonizing pain). During the first session, the eiLBP group reported a VAS of 2.54 ± 0.91, which was between a rating of 2 (annoying) and 4 (uncomfortable) and is similar to that reported for chronic LBP individuals who exhibited increased sway measures while sitting compared to controls.[7] During the second session, each eiLBP participant reported a decreased VAS, with an average of 1.16 ± 0.74 (paired *t*-tests, $p<0.001$). All members of the control group reported a VAS of 0 during both sessions.

Intrinsic trunk stiffness and paraspinal reflex delay were determined using sudden trunk flexion position perturbations.[16,19-21] The experimental setup and data analysis has been described in detail elsewhere[20] and will only be summarized here. Participants sat in an upright posture in a rigid metal frame (Figure 4-1) with their pelvis strapped to the frame, and attached to a

servomotor (Kollmorgen AKM53K, Radford, VA, USA) at the T_8 level of the spine via a rigid harness/rod connection. A seated position was implanted for consistency with concurrent data collection of seated balance included as Study 2 within this document. The motor height, frame, and rod length were adjustable so that the rod connection was horizontal and participants could sit upright. Foot height was controlled to avoid hip angles deviating from 90 degrees, which could affect pelvic tilt, and a harness height was recorded to allow for reproducing this configuration within the second session. During each 40-second trial, the motor applied a series of 12 anterior and 12 posterior position perturbations, each moving the rod connection 10 mm with a peak velocity of 233.1 mm/s. Each perturbation was completed within ~40 ms, which is quicker than typical erector spinae delays.[22,23] Pseudorandom delays between each perturbation minimized participant anticipation, and participants were instructed to maintain an upright trunk and otherwise not attempt to resist or intervene with the perturbations. Visual inspection of electromyography (EMG) activity as described below ensured no co-contraction during the perturbations. A practice trial was completed prior to one trial for data analysis.

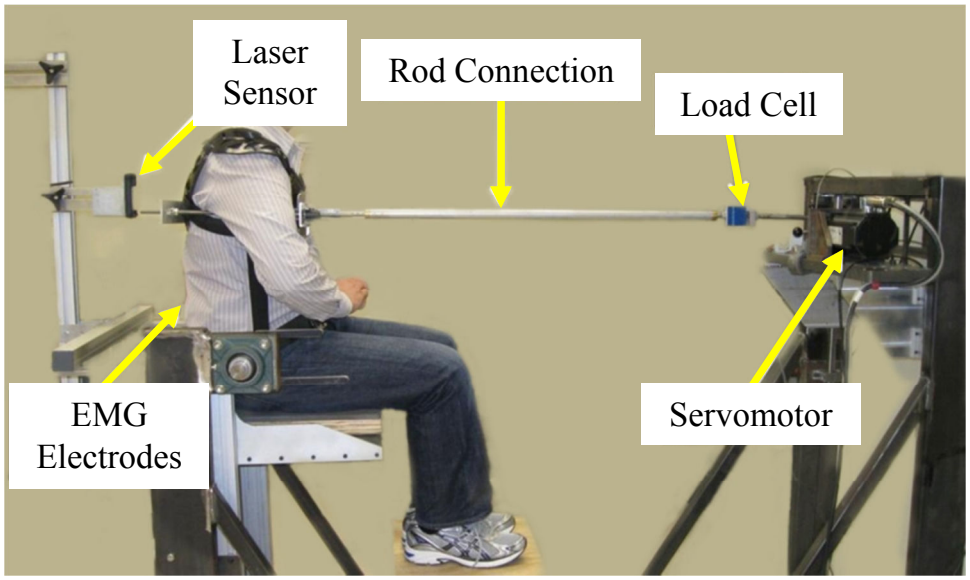

Figure 4-1. Experimental set up for seated trunk response measurements.

During each trial, motor displacement was sampled at 1000 Hz using a high accuracy encoder on the shaft of the motor. Trunk position was also sampled at 1000 Hz with a high accuracy CCD laser displacement sensor (Keyence LK-G 150, Osaka, Japan) focused on the midline of the

dorsal aspect of the harness just above the rod height. To account for the vertical offset between the laser sensor and the connecting rod, laser measurements were multiplied by the ratio of these heights from L_5S_1. Forces in the rod connecting the shaft of the motor to the harness were sampled at 1000 Hz using an in-line load cell (Interface SM2000, Scottsdale, AZ, USA). EMG electrodes (bipolar Ag/AgCl) were placed bilaterally over the erector spinae (ES) (~3 cm from the midline at L_3 and L_1 levels)[24] and rectus abdominus (RA) muscles, ES for determining paraspinal reflex delay and both ES and RA to ensure no co-contraction during perturbations. Raw EMG signals were amplified (Measurement Systems Inc., Ann Arbor, MI, USA), sampled at 1000 Hz, bandpass filtered (20 - 500 Hz), full-wave rectified, and smoothed using a 25 Hz zero-phase-lag low-pass Butterworth filter. Displacement and force data were similarly low-pass filtered at 10 Hz (7^{th}-order, zero-phase-lag Butterworth filter).

Intrinsic trunk stiffness was estimated with a two degree-of-freedom model of the trunk and harness/rod connection described in detail elsewhere.[19-21] Briefly, inputs to the model were the displacements collected from the motor encoder and laser sensor along with their numerically calculated 1^{st} and 2^{nd} derivatives. The output was an estimated force response. Model parameters of stiffness, damping, and effective mass (i.e. the driving point mass) were determined for each degree of freedom using a curve fit routine in MATLAB (MathWorks, Natick, MA, USA) that minimized the sum of the squared difference between estimated and measured force time series for each anterior perturbation. Initial efforts revealed an inability to consistently differentiate stiffness and damping, likely due to the short time interval over which our analysis was conducted. As such, trunk damping was set to zero, similar to previous approaches.[19,21,25] Therefore, changes in trunk mechanical behavior in this study were represented by changes in stiffness and effective mass. The model was fit to each of the 12 anterior trunk position perturbations within each trial using only data when the load cell measured a tensile force in the load cell, and only the value of the trunk stiffness derived from the best model fit within each trial was used. The reflex delay was determined for the same perturbation, and was defined as the time between motor position onset and the onset of L_1 level EMG muscle activity (averaged across left and right). Onset was defined as the instant that motor position or muscle activity exceeded its pre-perturbation (~40 ms) average plus two standard deviations.

Separate two-way mixed-factor analyses of variance were used to investigate the effects of group, session, and their interaction on intrinsic stiffness, reflex delay, effective mass, and peak trunk velocity. Simple effects were investigated following a significant group × session interaction. All statistical analyses were conducted using JMP 8 (SAS Software, Cary, NC, USA) with a significance level of $p \leq 0.05$.

4.3 Results

Intrinsic trunk stiffness exhibited no effect of group (p=0.222), a decrease with session (p=0.017), and a significant group × session interaction (p=0.032; Figure 4-2a). Simple effects revealed that trunk stiffness did not change within the eiLBP group from post-exercise to post-recovery (p=0.826), yet it decreased 22% within the control group (p=0.002). Trunk stiffness was not different between eiLBP (9.02 ± 2.35 N/mm) and control (9.05 ± 1.70 N/mm) groups post-exercise (p=0.978), but it was 26% higher in the eiLBP group (8.90 ± 0.80 N/mm) compared to the control group (7.05 ± 1.51 N/mm) post-recovery (p=0.033).

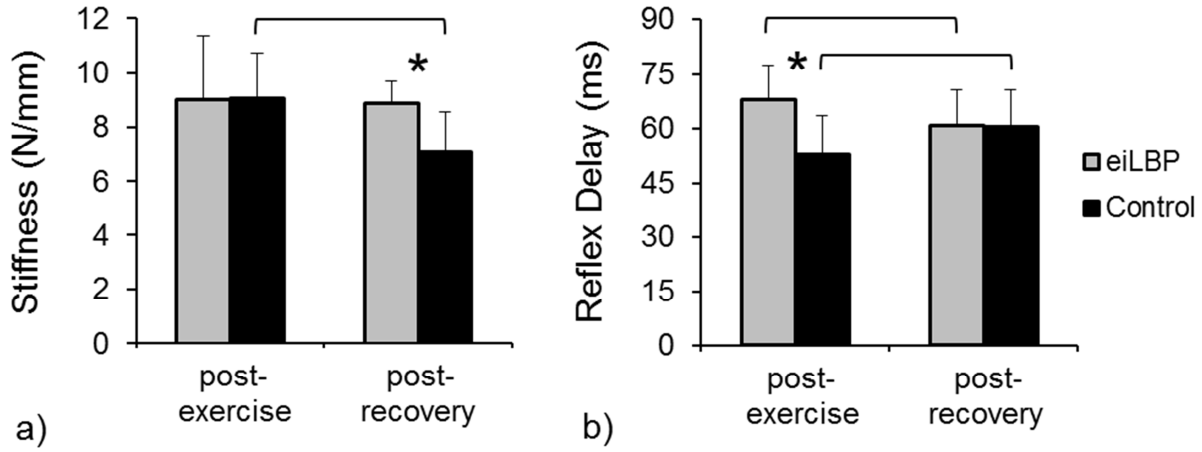

Figure 4-2. Intrinsic trunk stiffness (a) and paraspinal reflex delay (b), separated by group and session. An asterisk indicates a group difference within session, and a line indicates a session difference within group. Error bars indicate standard deviation.

Paraspinal reflex delay exhibited no effect of group (p=0.110) or session (p=0.888), and a significant group × session interaction (p=0.001; Figure 4-2b). Simple effects revealed that reflex delay decreased 11% within the eiLBP group from post-exercise to post-recovery (p=0.013), yet it increased 15% within the control group (p=0.006). Reflex delay was 29% longer in the eiLBP group (68.1 ± 9.4 ms) compared to the control group (52.7 ± 11.1 ms) post-exercise (p=0.006), but it was not different between eiLBP (60.8 ± 10.0 ms) and control (60.6 ± 10.0 ms) groups post-recovery (p=0.969).

Effective mass exhibited a decrease with session (one: 15.9 ± 3.25 kg; two: 13.6 ± 3.98 kg; p=0.010), but no effect of group (eiLBP: 15.7 ± 3.70 kg; control: 13.9 ± 3.74 kg; p=0.268) or a group × session interaction (p=0.415). Peak trunk velocity exhibited no effect of group (eiLBP: 221 ± 22 mm/s; control: 214 ± 20 mm/s; p=0.508), session (one: 220 ± 20 mm/s; two: 214 ± 22 mm/s; p=0.230), or a group × session interaction (p=0.920).

4.4 Discussion

The purpose of this study was to investigate the effects of acute eiLBP on intrinsic trunk stiffness and paraspinal reflex delay. Measurements were collected while eiLBP participants were experiencing LBP and again after LBP had subsided, as well as in a group of controls who had no history of LBP. Our first hypothesis was that neuromuscular control would differ between a healthy control group and a eiLBP group when not experiencing pain. This hypothesis was supported because the eiLBP group exhibited higher trunk stiffness (yet not reflex delay) post-recovery. Our second hypothesis was that exercise would induce LBP and change neuromuscular control in the eiLBP group. This hypothesis was supported because exercise did induce LBP within the eiLBP group and an increased reflex delay (but not stiffness). Our third hypothesis was that exercise would neither induce LBP nor change neuromuscular control in the control group. This hypothesis was rejected because, although exercise did not induce LBP in the control group, it did induce an increase in stiffness and a decrease in reflex delay. Our results indicate differences in neuromuscular control between groups both when the eiLBP group was experiencing pain and when they were not experiencing pain. However, the differences when the eiLBP group was experiencing pain involved a longer reflex delay (yet no difference in

stiffness), and the difference when the eiLBP group was not experiencing pain involved higher trunk stiffness (yet no difference in reflex delay).

As with previous models,[10,16,19-22,26-28] the model used here to determine trunk intrinsic stiffness assumed stiffness and effective mass were constant over the trunk's response to the perturbation (i.e. a linear time-invariant system). Although the behavior of the trunk is non-linear,[27,28] the trunk displacements (~10mm) and perturbation durations (~40ms) used here were significantly shorter than those used elsewhere[10,26] and would seem to make this assumption more tenable. The forces predicted by the model were highly correlated with experimentally-measured forces (mean $r = 0.976$), suggesting the model provided a reasonable representation of the system. As in other trunk perturbation studies,[10,25] effective trunk mass (i.e. the driving point mass) was underestimated (14.9 kg) compared with anthropometric predictions[29] of trunk mass (26.2 kg), and can be explained (at least partly) by a non-rigid trunk. Effective trunk mass also decreased an average of 14% from post-exercise to post-recovery. It should be noted that the predicted effective mass and intrinsic stiffness from the model are coupled due to the fact that the trunk is not rigid.[10] For example, a decrease (or increase) in trunk stiffness would lead to more (or less) inter-segmental motion, less (or more) trunk mass moved by the perturbation, and therefore a lower (or higher) predicted effective mass by the model. Similarly, a decrease (or increase) in trunk mass would lead to a lower (or higher) predicted trunk stiffness. As such, the decrease in effective trunk mass seen here from post-exercise to session was similar to the corresponding 12% decrease in intrinsic stiffness.

Differences in stiffness and reflex delay between groups reported here are consistent with those reported elsewhere. Individuals with a history of recurrent acute LBP had 22% higher effective trunk stiffness (2.00 N/mm) when they were not experiencing LBP compared to individuals without LBP (1.64 N/mm).[10] This difference is similar to the 26% higher intrinsic stiffness in the eiLBP group (8.90 N/mm) compared to the control group (7.05 N/mm) reported here post-recovery when the eiLBP group was not experiencing LBP. Regarding reflex delay, individuals with chronic LBP had ~26% longer paraspinal reflex delay (80-85ms) compared to individuals without LBP (62-69ms).[6-8] This difference is similar to the 29% longer reflex delay in the eiLBP group (68ms) compared to the control group (53ms) reported here post-exercise when the eiLBP

group was experiencing LBP. Intrinsic stiffness found here in the control group post-recovery (7.05 N/mm) is comparable if not slightly larger than values reported in studies using similar trunk perturbation methods on standing males (6.29 ± 2.84 N/mm[16] and 4.91 N/mm[21]), but they are ~3-4 times larger than effective stiffness values quantified from force perturbations over longer periods of time (~370ms[10]). Additionally, reflex delay values reported here were slightly lower in magnitude than those detected with force perturbations.[6-8] Reasons for higher stiffness and lower reflex delay seen here are likely due to methodological differences between studies. The present study employed faster and shorter trunk perturbations that were completed before any observable reflex. Also, participants in the present study were in a seated position that would stretch the posterior tissues of the trunk and likely increase stiffness values[16,26,30] and decrease reflex delays.

Based on the effects of eiLBP and exercise that does not induce LBP, it is apparent that both groups do not change the same with exercise as measured by intrinsic stiffness and paraspinal reflex delay. eiLBP did not affect intrinsic stiffness, but it did increase reflex delay. The magnitude of intrinsic stiffness in the eiLBP group, which remained the same throughout both periods of pain and no pain, did not differ from the higher intrinsic stiffness in the control group due to exercise. Thus, this consistently increased stiffness may represent a protective mechanism by the body to limit local motion and increase stability. Other studies have hypothesized greater stiffness in those with LBP to be a compensatory stabilization technique based upon higher baseline EMG levels in a normal upright stance. [17,31] Changes in stiffness through increased co-contraction[32] could also have an effect on paraspinal reflex delay such that an increase in stiffness can cause a decrease in delay. Reasons for this could be that increased stiffness introduces tension in the paraspinal muscles, removing any "slack" which will allow them to respond more quickly to a change in length. Consistent with this, the control group in the present study exhibited an increase in stiffness and a decrease in reflex delay with exercise (from post-recovery to post-exercise). Increased stiffness is similar to previous findings in exercise studies that induced what is commonly termed muscle soreness, yet this was alongside an increased reflex delay.[33-35] The eiLBP group did not exhibit an increase in stiffness with exercise, yet they did increase reflex delay. Perhaps the higher intrinsic stiffness in the eiLBP

group when not experiencing pain, compared to controls, limits how exercise/pain further increases stiffness for offsetting an increase in reflex delay.

In conclusion, the present study was the first to our knowledge to compare trunk neuromuscular control between episodes of pain (post-exercise) and no pain (post-recovery) within individuals who experience eiLBP, as well as compare neuromuscular control to a healthy control group. This posed an opportunity for differentiating effects of current LBP from individual characteristics within those who experience eiLBP. Higher intrinsic stiffness within the eiLBP group while not in pain, and no difference while in pain, resulted from an increase in stiffness within the control group due to exercise (that did not induce LBP). No difference in reflex delay while not in pain, and longer reflex delays within the eiLBP group while in pain, resulted from an increase within the eiLBP group and a decrease within the control group. These results suggested that individuals who experience eiLBP exhibited increased stiffness compared to healthy controls that was unaffected by the presence of pain, and increased reflex delays concurrent only with pain.

4.5 Acknowledgements

This work is dedicated to the late Dr. Kevin P. Granata. This work was supported by awards R01AR046111 from the National Institute of Arthritis and Musculoskeletal and Skin Diseases of the National Institutes of Health (NIH), and R01OH008504 from the National Institute for Occupational Safety and Health of the Centers for Disease Control and Prevention (CDC). Its contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH and CDC.

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5 Effects of Exercise-Induced Low Back Pain on Seated Sway

5.1 Introduction

Low back pain (LBP) is a significant health and economic problem in the United States and around the world.[1] An estimated 60-80% of all adults experience LBP at some point in their lifetime,[2] and a 2004 study reported that annual health care costs in the US associated with LBP exceeded $90 billion.[3] In effort to better understand factors contributing to LBP, fundamental measures of trunk neuromuscular control have been quantified by characterizing the trunk response to sudden force[4-7] and position[8-10] perturbations. LBP has been associated with altered trunk neuromuscular control compared to individuals without LBP including delayed paraspinal reflexes[4-6] and increased trunk stiffness.[7]

In addition to these fundamental measures of trunk neuromuscular control, measures of seated sway while sitting on an unstable seat have been used to quantify trunk neuromuscular control. Attempting to sit as still as possible on an unstable seat is a task with a relatively unambiguous goal that is dependent upon multiple components of trunk neuromuscular control including (but not limited to) proprioceptive input from trunk, central nervous system processing, and well-coordinated trunk muscle activation. Thus, it may provide a more global and functional measure of trunk neuromuscular control compared to fundamental measures in response to sudden perturbations. Several studies have shown seated sway to be sensitive to various extrinsic and intrinsic factors. For example, seated sway as quantified using the center of pressure (COP) under an unstable seat is positively correlated with age and body weight.[11] COP velocity also increases with increased active trunk stiffness,[12] but exhibits no change when wearing a lumbosacral orthosis to artificially increase trunk stiffness.[12,13] Seated sway as quantified using kinematic measures of seat angle is also sensitive to influencing factors including whole body vibration, which is thought to be a risk factor for LBP.[14]

Alterations in seated sway are also associated with LBP. Radebold et al. (2001) reported increased COP-based measures of sway in individuals with chronic LBP compared to a healthy control group.[5] Unfortunately, this descriptive experimental design, while informative, is limited

in that it is unable to distinguish whether the LBP individual exhibits altered neuromuscular control only while experiencing LBP, or also when pain free. Additional insight can be gained on the relationship between trunk neuromuscular control and LBP by investigating individuals who experience recurrent exercise-induced LBP (eiLB). Comparing measurements of seated sway between episodes of pain (post-exercise) and no pain (post-recovery) within eiLBP individuals, as well as between eiLBP individuals while pain free and a group of healthy controls, could help differentiate the effects of LBP from individual differences. Therefore, the purpose of this study was to investigate effects of eiLBP on seated sway. The following three hypotheses were tested: 1) seated sway would differ in the eiLBP group when not experiencing pain compared to a healthy control group, 2) exercise would induce LBP and change seated sway in the eiLBP group, and 3) exercise would neither induce LBP nor change seated sway in the control group. If these hypotheses are supported, results would suggest that altered performance of seated sway is a characteristic of the eiLBP individual even when not experiencing pain, and alterations are exaggerated with the presence of pain.

5.2 Methods

Participants included 17 male members of the Virginia Tech Triathlon Club. Eight of these participants experience acute eiLBP (mean height ± SD: 1.83 ± 4.7 m; mass: 72.9 ± 2.7 kg; age: 20.7 ± 1.0 yr) and nine controls report no history of LBP (height: 1.79 ± 3.2 m; mass: 70.8 ± 7.3 kg; age: 20.4 ± 1.6 yr). To minimize effects of age and weight, participants were to have an age of 18-49 and a body mass index less than 30 kg/m^2. eiLBP participants were required to pass a screening by a chiropractic physician that evaluated their ability to complete the experiment without further injury, and ensured agreement with specific inclusion and exclusion criteria. Inclusion criteria included acute eiLBP for at least than six months, and exclusion criteria included any neurological deficits, vestibular or visual disorders, major structural deformities of the spine, genetic spinal disorders, spinal surgery within the past five years, or spinal mechanical implants. This study was approved by the Virginia Tech Institutional Review Board, and written consent was obtained from all participants prior to participation.

Seated sway was quantified for all participants during two experimental sessions. Two sessions were determined based on the fact that specific days of exercise, acute eiLBP, and subsided LBP were known to occur in particular triathlon individuals. The first session was 1-2 days following a weekend race (post-exercise), when the eiLBP group was experiencing an episode of LBP. The second session was 4-5 days following the weekend race (post-recovery) when the LBP had subsided (see below). The control group participated in experimental sessions on the same days following a race and did not experience eiLBP. A visual analog scale[15] (VAS) was used for rating LBP during each session. The VAS quantified LBP on a numerical scale with text descriptors from 0 (none) to 10 (agonizing pain). During the first session, the eiLBP group reported a VAS of 2.54 ± 0.91, which was between a rating of 2 (annoying) and 4 (uncomfortable) and is similar to that reported for chronic LBP individuals who exhibited increased sway measures while sitting compared to controls.[5] During the second session, each eiLBP participant reported a decreased VAS, with an average of 1.16 ± 0.74 (paired *t*-tests, $p<0.001$). All members of the control group reported a VAS of 0 during both sessions.

Seated sway was measured while participants sat in an upright posture on a custom-made wobble chair (Figure 5-1).[14,16] The wobble chair consisted of a rigid seat surface that pivoted about a transverse axis such that seat movement only occurred in the sagittal plane. Participants wore a belt to secure their pelvis to the seat. An adjustable foot support attached to the seat surface supported the lower limbs while the knees and ankles were positioned at approximately 90 degrees. An adjustable seat translation also allowed the participant to be placed in the correct anterior-posterior position above the pivot point, such that the seat was balanced when the participant was sitting upright. Participants were instructed to try to sit as still as possible while balancing the seat above the pivot point. Movement during the task occurred predominantly at the lumbar spine, and the pelvis and lower extremities moved as one segment. The maximum angular excursion of the seat that could occur from neutral before impacting the base of the seat surface was ±10 degrees.

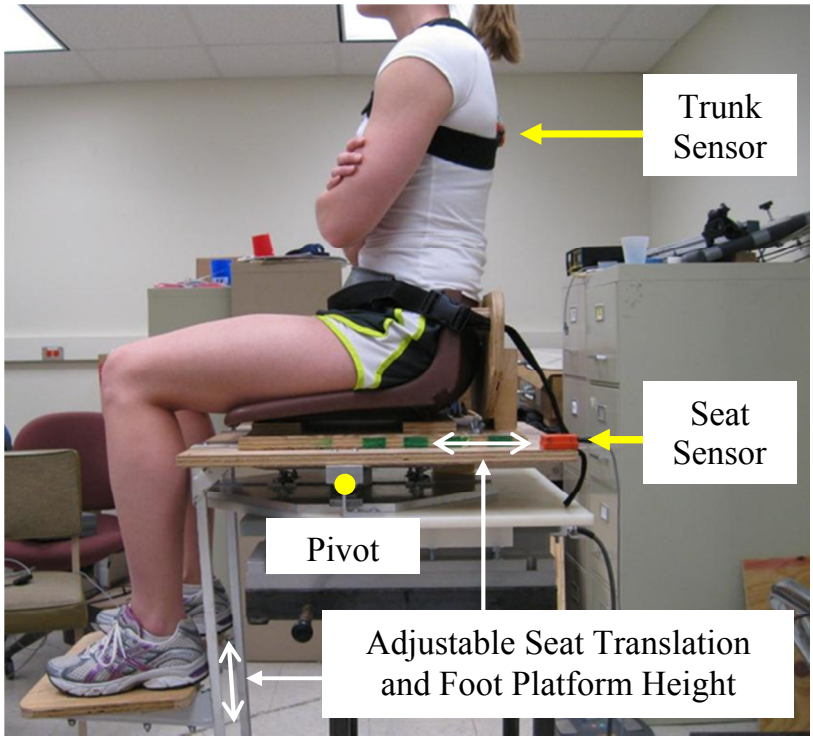

Figure 5-1 Wobble chair experimental set-up. Instrumentation included a forceplete for calibration purposes, as well as two inertial sensors for sagittal seat and trunk angle data collections. Springs are located underneath the seat, depicted more clearly in the next figure.

When seated on the wobble chair, destabilizing moments arise from gravitational effects associated with small angle deviations from the neutral position (i.e. where the chair-subject system center of mass (COM) was positioned directly above the pivot). Springs under the seat (Figure 5-2) were used to offset the gravitational moment, $mgh\sin(\theta)$, to account for varying participant mass and to determine the difficulty level of balancing on the wobble chair. The stabilizing restorative moment provided by the springs toward the neutral position of the seat, $F_sL=kL^2\sin(\theta)$, was manipulated by translating the position of the springs a distance (L) closer to or further away from the center pivot point. The spring distance was proportional to the linearized gradient of gravitational moment, $M_{Gravity}$, determined in static calibration measurements for two known tilt angles. A spring position corresponding to 100% of $M_{Gravity}$ would mean that the gravitational moment due to deviations from neutral are completely offset by the springs. If the stabilizing restorative moment from the springs is less than the destabilizing gravitational moment, the system is inherently unstable. When the system is

unstable, movements at the lumbar spine are necessary to correct for disturbances in seat angle to maintain an upright seated posture.

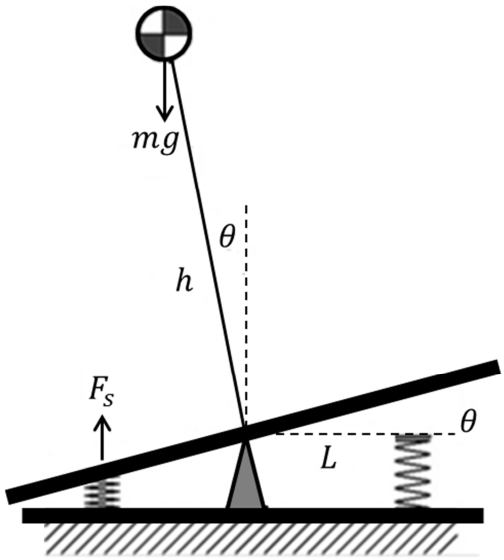

Figure 5-2 Restorative moments from the springs offset a percentage of $M_{Gravity}$. F_S is the restorative force from one spring, counteracting the center of mass (m) located at the radial height (h) of the chair-subject system above the seat surface.

Within each session, two difficulty levels (i.e. spring positions corresponding to an easy 70% and a challenging 45% $M_{Gravity}$) were presented to all participants with increasing difficulty. Measurements at each difficulty level consisted of five repetitions, for a total of ten trials per session. Each trial was 30 seconds long and began after the seat was unclamped and once the participant had maintained balance for about five seconds. Participants were allowed to stand and provided adequate rest between all trials to avoid any effects due to prolonged sitting.[17,18] During each trial, seat and trunk angles were sampled at 100 Hz using tri-axial inertial position sensors (Xsens Technology), one placed on the seat surface and another on the T8 level of the midline dorsal spine. Sensors were oriented similarly to record pitch angles in the sagittal plane. Angles were processed with a 4th-order, zero-phase-lag low-pass Butterworth filter at 20 Hz. All values were demeaned so that the mean value of each trial was zero. Representative sway paths of sagittal seat and trunk angles are shown in Figure 5-3.

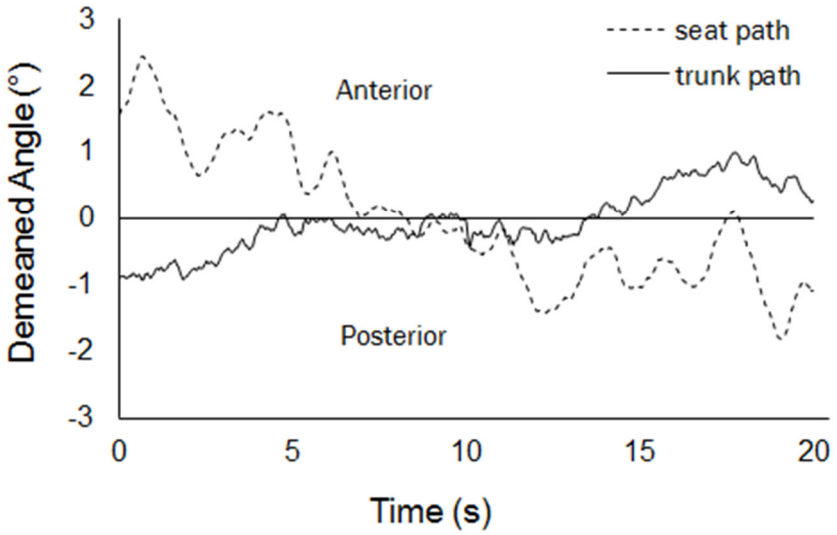

Figure 5-3 Representative sway of anterior-posterior seat and trunk angles.

Seated sway was quantified in three ways: 1) the number of trials where the seat impacted the base, 2) measures of seat and trunk angle variability, and 3) measures of non-linear stability using seat angle. Of all 30-second trials, the third trial in each level consistently had 20-second segments available that lacked a seat impact. Therefore trial three was used for subsequent sway analyses. Measures of seat and trunk angle variability were adapted from Prieto et al. (1996).[19] Displacement measurements included a mean distance (MeanD), square root of the average squared distance (RmsD) and maximum distance (MaxD). Mean velocity (MeanV) was the average of the absolute value of the time series velocity. Power was the integrated area of the FFT of the time series, and frequencies determined from the power spectrum array included a mean (MeanF), 95% power frequency (95%PF), and centroidal frequency (CentrF). Measures of non-linear stability on seat angle sway using the stabilogram diffusion function (SDF) were determined as seen in Collins and Deluca (1993).[20] To determine SDF measures, first, variance was computed from the mean squared distances traveled by the signal within each time interval of 1 sample to 10 seconds.[20] The stability diffusion exponent demonstrates bi-linear behavior, with regions representing open-loop and closed-loop systems.[11,20] Therefore, measures of short-term (D_S) and long-term (D_L) coefficients, as well as short-term (H_S) and long-term (H_L) exponents, were chosen to describe these regions, separated by a critical point in time (CT_t) at which variance has a particular amplitude (CT_a).[20] In each region, coefficients were calculated as

the slope of the best fit line of the linear-linear plot of variance versus time interval, while exponents were the slopes of the best fit line of the log-log plot of variance versus time interval.

Chi-squared tests were used to investigate differences in the number of trials where the seat impacted the based between selected combinations of session, difficulty level, and group. Our three hypotheses were tested using planned contrasts following three-way mixed-factor analyses of variance (ANOVAs) performed on the ranks (due to some non-normal distributions) of seat and trunk measures. Contrasts were performed at both difficulty levels between groups within each session, and between sessions within each group. Additional contrasts were preformed between difficulty levels within each group and session. All statistical analyses were conducted using JMP 8 (SAS Software, Cary, NC, USA) with a significance level of as $p \leq 0.05$.

5.3 Results

The seat did not impact the base during any trials at the easy level, but did during 26 of 170 trials at the challenging level (Table 5-1), dispersed throughout all five trials in each level. Post-exercise, the number of trials where the seat impacted the base was not different between groups (p=0.518). Within the eiLBP group, the number of trials where the seat impacted the base was not different post-exercise (when they were experiencing LBP) compared to post-recovery (p=0.610). Within the control group, the number of trials where the seat impacted the base was higher post-exercise compared to post-recovery (p=0.032). Post-recovery (when LBP had subsided in the eiLBP group), the number of trials where the seat impacted the base was higher in the eiLBP group compared to the control group (p=0.022).

Table 5-1 Trails during which the Seat Impacted the Base

Group	Post-Exercise		Post-Recovery	
	Easy	Challenging	Easy	Challenging
Control	0/45	7/45	0/45	1/45
eiLBP	0/40	8/40	0/40	10/40

Seated sway exhibited differences between groups and sessions (Table 5-2). Post-recovery when the LBP had subsided in the eiLBP group and the control group was not experiencing pain, five of 22 sway measures differed between groups at the challenging difficulty level. These included the eiLBP group exhibiting 11% lower seat 95%PF, 46% higher seat MeanD, 35% higher seat RmsD, 32% higher seat MaxD, and 74% higher seat Power. Also post-recovery, two of 22 sway measures differed between groups at the easy difficulty level. These included 15% lower seat 95%PF and 11% higher seat H_S in the eiLBP group. The eiLBP group did not exhibit any changes in seated sway between sessions at either difficulty level. The control group exhibited a change in only one of 22 sway measures at the easy difficulty level. This was a 33% lower seat D_S post-recovery compared to post-exercise. Post-exercise when the eiLBP group was experiencing pain, one of 22 sway measures differed between groups at the challenging difficulty level. This was a 15% lower seat 95%PF in the eiLBP group. Also post-exercise, four of 22 sway measures differed between groups at the easy difficulty level. These included 14% higher seat H_S and 47% lower trunk Mean, 54% lower trunk 95%PF, and 29% lower trunk CentrF in the eiLBP group.

Seated sway also exhibited differences between difficulty levels, yet seven instances existed where differences between levels occurred for one group only. For example, post-recovery when LBP had subsided in the eiLBP group and the control group was not experiencing pain, only the eiLBP group increased trunk 95%PF (63%) with difficulty level. Also post-recovery, only the control group increased H_S (10%) with difficulty level. Post-exercise when the eiLBP group was experiencing pain, only the eiLBP decreased leat 95%PF (12%), increased trunk MeanF (93%), 95%PF (145%), and CentrF (46%) with difficulty level. Also post-exercise, only the control group increased H_S (10%) with difficulty level.

Table 5-2 Medians (25th Quartile) by Level, Session, and Group

Measure	Easy Level, 70% $M_{Gravity}$				Challenging Level, 45% $M_{Gravity}$			
	Post-Exercise		Post-Recovery		Post-Exercise		Post-Recovery	
	Control	eiLBP	Control	eiLBP	Control	eiLBP	Control	eiLBP
Seat Angle								
MeanD (deg)	0.417 (0.334)	0.569 (0.439)	0.421 (0.296)	0.500 (0.342)	1.139 (0.743)	1.307 (1.037)	1.099 (0.655)	**1.602 (1.108)**
RmsD (deg)	0.506 (0.415)	0.698 (0.525)	0.546 (0.398)	0.616 (0.429)	1.431 (0.956)	1.589 (1.272)	1.432 (0.819)	**1.928 (1.447)**
MaxD (deg)	2.201 (2.031)	3.003 (2.387)	2.703 (1.961)	3.065 (1.908)	6.081 (4.970)	6.751 (4.980)	5.881 (3.752)	**7.758 (6.793)**
MeanV (deg/s)	0.991 (0.749)	0.742 (0.667)	0.819 (0.717)	0.811 (0.712)	1.662 (1.371)	1.604 (1.533)	1.362 (1.199)	1.564 (1.496)
MeanF (Hz)	0.356 (0.249)	0.258 (0.231)	0.298 (0.258)	0.302 (0.261)	0.278 (0.236)	0.194 (0.179)	0.270 (0.220)	0.193 (0.154)
Power (kdeg^2)	0.511 (0.346)	0.974 (0.554)	0.596 (0.318)	0.759 (0.369)	4.093 (1.831)	5.093 (2.899)	4.099 (1.342)	**7.120 (4.191)**
95%PF (Hz)	0.750 (0.650)	0.625 (0.600)	0.850 (0.725)	**0.725 (0.625)**	0.650 (0.600)	**0.550 (0.511)**	0.600 (0.575)	**0.536 (0.413)**
CentrF (Hz)	0.770 (0.446)	0.917 (0.481)	0.812 (0.630)	1.027 (0.604)	0.989 (0.544)	0.491 (0.287)	0.927 (0.474)	0.381 (0.308)
CT_t (s)	1.300 (1.150)	1.485 (1.418)	1.110 (0.930)	1.290 (1.088)	1.330 (1.125)	1.415 (1.198)	1.280 (1.140)	1.730 (1.208)
CT_a (deg^2)	0.422 (0.234)	0.518 (0.327)	0.260 (0.230)	0.394 (0.257)	1.340 (1.106)	2.736 (2.133)	2.064 (0.909)	3.623 (2.349)
D_S (deg^2/s)	0.189 (0.120)	0.178 (0.133)	*0.127 (0.069)*	0.162 (0.119)	0.832 (0.530)	1.096 (0.681)	0.686 (0.427)	1.350 (0.922)
D_L (deg^2/s)	0.015 (0.003)	0.052 (0.030)	0.019 (0.011)	0.028 (0.003)	0.159 0.004	0.299 (0.021)	0.079 (0.016)	0.260 (0.087)
H_S	0.688 (0.669)	**0.785 (0.744)**	0.682 (0.617)	**0.758 (0.728)**	0.755 (0.722)	0.791 (0.753)	0.752 (0.690)	0.766 (0.749)
H_L	0.189 (0.069)	0.327 (0.215)	0.235 (0.166)	0.202 (0.051)	0.265 (0.021)	0.308 (0.089)	0.235 (0.058)	0.299 (0.161)
Trunk Angle								
MeanD (deg)	0.411 (0.274)	0.584 (0.310)	0.605 (0.293)	0.426 (0.273)	0.476 (0.319)	0.424 (0.334)	0.486 (0.395)	0.460 (0.405)
RmsD (deg)	0.463 (0.349)	0.697 (0.369)	0.722 (0.356)	0.527 (0.347)	0.575 (0.382)	0.526 (0.414)	0.592 (0.476)	0.571 (0.498)
MaxD (deg)	2.044 (1.693)	2.703 (1.596)	2.508 (1.647)	2.224 (1.662)	3.293 (1.708)	2.479 (1.977)	2.576 (2.189)	2.956 (2.460)
MeanV (deg/s)	1.021 (0.813)	0.744 (0.707)	0.912 (0.887)	0.792 (0.715)	1.311 (1.045)	1.336 (1.152)	1.274 (1.036)	1.106 (1.056)
MeanF (Hz)	0.427 (0.273)	**0.227 (0.200)**	0.338 (0.282)	0.248 (0.221)	0.419 (0.312)	0.438 (0.355)	0.354 (0.274)	0.359 (0.282)
Power (kdeg^2)	0.428 (0.250)	0.971 (0.274)	1.040 (0.254)	0.564 (0.240)	0.488 (0.295)	0.533 (0.248)	0.701 (0.434)	0.622 (0.496)
95%PF (Hz)	1.200 (0.825)	**0.550 (0.400)**	1.100 (0.650)	0.750 (0.538)	1.300 (1.225)	1.350 (1.113)	1.150 (0.875)	1.225 (1.038)
CentrF (Hz)	1.262 (1.015)	**0.897 (0.770)**	1.149 (0.867)	0.901 (0.571)	1.144 (0.743)	1.306 (1.015)	1.145 (0.708)	0.969 (0.687)

Bold indicates a statistically significant difference ($p \leq 0.05$) between groups within session and difficulty level.

Italicized indicates a statistically significant difference ($p \leq 0.05$) between sessions within group and difficulty level.

Underlined indicates a statistically significant difference ($p \leq 0.05$) between difficulty levels within group and session.

5.4 Discussion

The purpose of this study was to investigate the effects of acute eiLBP on seated sway. Measurements were collected while eiLBP participants were experiencing LBP and again after LBP had subsided, as well as in a group of controls who had no history of LBP. Our first hypothesis was that seated sway would differ between the control group and eiLBP group when not experiencing pain. This hypothesis was supported because, post-recovery, five of 22 seated sway measures differed between groups at the challenging level and two of 22 differed between groups at the easy level. Our second hypothesis was that exercise would induce LBP and change seated sway in the eiLBP group. This hypothesis was rejected because, although exercise did induce LBP within the eiLBP group, no changes between sessions were found for the eiLBP group. Our third hypothesis was that exercise would neither induce LBP nor change seated sway in the control group. This hypothesis was rejected because, although exercise did not induce LBP in the control group, it induced a change in one of 22 seated sway measures at the easy level. While seated sway within the eiLBP group did not change significantly between sessions (second hypothesis), some group differences post-exercise are not consistent with those post-recovery (first hypothesis). For example, post-exercise, one of 22 seated sway measures differed between groups at the challenging level and four of 22 differed between groups at the easy level, versus five and two post-recovery, respectively. Our results indicate differences in seated sway between groups both when the eiLBP group was experiencing pain and when they were not experiencing pain, with slight variations due to the presence of pain in the eiLBP group.

It is interesting to compare previously reported effects of LBP on seat sway to our results. Radebold et al. (2001) reported larger anterior-posterior displacements and path lengths in individuals with chronic LBP compared to individuals without LBP, with larger differences at an increased difficulty level.[5] This group comparison is most similar to comparing groups post-exercise in the current study when the eiLBP group was experiencing LBP. At this time, the eiLBP group had lower seat frequencies in the challenging level, as well as larger SDF short-term exponents and lower trunk frequencies in the easy level, yet no displacements were different between groups. Larger SDF short-term exponents in the eiLBP group imply decreased stability in terms of open-loop feedback, such that sway is more prone to persist in the direction

is it currently headed.[20,21] Additionally, while larger SDF short-term exponents were only observed in the eiLBP group for the easy level, the control group increased with difficulty level towards the magnitude exhibited by the eiLBP group during both sessions. Several methodological differences between Radebold et al. (2001) and the current study could account for differences in results. First, Radebold et al. tested individuals with chronic LBP while the current study tested individuals with recurrent acute eiLBP. It is possible that trunk neuromuscular control differs between these two types of LBP. Second, Radebold et al. (2001) employed COP-based measures of seated sway while the current study employed kinematic measures of seated sway, which could result in differences in various components of and sensitivity to trunk neuromuscular control. Third, the unstable seat design differed between studies and could also result in differences in sensitivity to trunk neuromuscular control.

Specific group differences within both sessions of the current study suggest that seated sway is impaired in eiLBP individuals. Post-exercise, the eiLBP group had lower seat frequencies in the challenging level, as well as larger SDF short-term exponents and lower trunk frequencies in the easy level, compared to controls. Post-recovery, the eiLBP group had larger displacements and larger SDF short-term exponents in the easy level, compared to controls. Increased displacements and SDF measures are thought to be undesirable in terms of seated sway in that the former increases with age and body weight,[11] and both increase after exposure to whole body vibration (a risk factor for LBP).[14] Recall, larger SDF short-term exponents imply decreased stability in terms of open-loop feedback.[20,21] Larger displacements, decreased open-loop stability, and decreased seat frequencies could be thought to associate with decreased trunk stiffness, yet LBP individuals are reported to have increased trunk stiffness.[7] Thus, LBP individuals may be compensating for larger sway disturbances with increased stiffness. However, during this functional task of seated sway, they still do not correct quite to the effect of the control group. Reasons for this inability could be decreased sensory function. These known impairments of increased displacements and SDF short-term exponents in the eiLBP group, in addition to observed decreased seat frequencies, could be due to larger reflex delays in LBP individuals.[4-6] Larger sensory delays could result in larger gravitational deviations from neutral (larger displacements), less frequent balance corrections (lower seat frequencies), and larger sway variances per time period (larger SDF short-term exponents).

This study allowed for differentiating effects of current LBP from individual characteristics within those who experience eiLBP. It is apparent that groups were not affected in the same way as a result of exercise or eiLBP. From post-exercise to post-recovery, only the control group decreased their number of trials with a seat impact, as well as their SDF short-term coefficients in the easy level suggesting an improvement in open-loop feedback. This is not thought to be a result of practice since each group had the same number of trials for balancing. Additionally, participants verbally confirmed they became accustomed to the task during the first few seconds of the first trial in each level, which was not included in the sway measure analyses. While no session differences were observed within the eiLBP group, they exhibited more group differences in seat measures post-recovery than post-exercise. Post-recovery (while not in pain), the eiLBP group had seat measures of lower frequencies and larger displacements, power, and SDF short-term exponents compared to controls. Post-exercise (while experiencing pain), the eiLBP group only had lower seat frequencies and larger SDF short-term exponenets compared to controls. Also while in pain, the eiLBP group exibited lower trunk frequencies in the easy level. It is possible that these lower trunk frequencies in the eiLBP group while in pain were a compensatory mechanism that could only be maintained when the difficulty level was not so challenging as to require the same trunk frequencies as the control group.

In conclusion, the present study compared seated sway between episodes of pain (post-exercise) and no pain (post-recovery) within individuals who experience eiLBP, as well as with a healthy control group. Individuals who experience eiLBP generally exhibited decreased frequencies of the seat and decreased open-loop stability. Additionally, trunk frequencies were decreased while individuals who experience eiLBP were currently in pain for the easy level. Results suggest that individuals who experience eiLBP exhibit impaired seat measures at all times, with altered trunk measures only while in pain and when the task was not challenging.

5.5 Acknowledgements

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6 Effects of Exercise-Induced Low Back Pain on the Neuromuscular Control of Seated Sway, a Model of Wobble Chair Balance

6.1 Introduction

Low back pain (LBP) is associated with altered neuromuscular control of the trunk including delayed paraspinal reflexes[1-3] and increased effective trunk stiffness[4] following the sudden release of a trunk extensor load that results in abrupt trunk flexion. Additionally, LBP is associated with altered performance of a functional task such as balancing while sitting on an unstable seat,[2] which is highly dependent upon neuromuscular control of the trunk. Multiple feedback models have been created to parameterize the postural control system during quiet standing, finding that active control over passive control is the main contributor to upright stance.[5-9] For example, Maurer and Peterka (2005) employed a proportional-integral-differential (PID) controller to approximate the underlying neuromuscular control of the ankle during quiet standing.[10] This model was able to reproduce realistic standing sway obtained from human subjects, and these investigators were able to identify differences in control parameters between young and older adults[10] who exhibited differences in standing sway.[11,12]

Attempting to sit as still as possible on an unstable seat (e.g. wobble chair) is similar to attempting to stand as still as possible during quiet standing in that it is a task with a relatively unambiguous goal highly dependent upon neuromuscular control. As such, a similar modeling approach as used by Maurer and Peterka (2005) may help to identify differences in underlying neuromuscular control of the trunk associated with LBP. Tanaka et al, (2010) modeled wobble chair balance to identify the boundary between regions of stability and loss of balance, as well as to determine an equilibrium manifold.[13] Identifying control parameters from measures of seated sway during wobble chair balance (collected in Study 2) could provide information on the neuromuscular control of the trunk during a functional task. Additional insight could be provided on the relationship between neuromuscular control of the trunk and LBP by identifying control parameters for individuals who experience acute exercise-induced LBP (eiLBP). Comparing measurements between episodes of pain (post-exercise) and no pain (post-recovery)

within eiLBP individuals, as well as between eiLBP individuals while pain free and a group of healthy controls, could help differentiate the effects of LBP from individual differences.

In an effort to better understand neuromuscular control of the trunk and determine if the control strategy within the central nervous system is altered in the presence of LBP, the purpose of this study was to create a model of wobble chair balance and investigate the effects of eiLBP on the control of seated sway. The control strategy was parameterized using a proportional-differential (PD) controller. PD control parameters were determined using a systems identification approach and experimental measures of seated sway during wobble chair balance, similar to methods used in previous inverted pendulum models simulating standing sway.[10,14] Control parameters were identified for and compared between all human subject conditions of Study 2. It was hypothesized that the model could reproduce realistic seated sway measures, and that control parameters within the model would differ with LBP. A working model would create a tool for quantifying neuromuscular control of the trunk during wobble chair balance, and therefore distinguishing effects of pain within the eiLBP individual, as well as between the eiLBP individual and healthy controls.

6.2 Methods

The modeling and systems identification approach employed here was similar to that originally performed by Maurer and Peterka for standing balance.[10] This approach involved using seated sway measures obtained from human subjects testing to obtain estimates of control parameters for wobble chair balance, and then comparing these control parameters between episodes of pain and no pain among individuals who experience recurrent acute eiLBP, and between individuals with eiLBP when pain free to healthy controls.

Participants included 17 male members of the Virginia Tech Triathlon Club. Eight of these participants experience acute eiLBP (mean height ± SD: 1.83 ± 4.7 m; mass: 72.9 ± 2.7 kg; age: 20.7 ± 1.0 yr) and nine controls report no history of LBP (height: 1.79 ± 3.2 m; mass: 70.8 ± 7.3 kg; age: 20.4 ± 1.6 yr). Inclusion and exclusion criteria for the eiLBP group can be found in Study 2. Performance of wobble chair balance was quantified for all participants during two

experimental sessions. Two sessions were determined based on the fact that specific days of exercise, acute eiLBP, and subsided LBP were known to occur in particular triathlon individuals. The first session was 1-2 days following a weekend race (post-exercise), when the eiLBP group was experiencing an episode of LBP. The second session was 4-5 days following the weekend race (post-recovery) when the LBP had subsided (see below). The control group participated in experimental sessions on the same days following a race and did not experience eiLBP. A visual analog scale[15] (VAS) was used for rating LBP during each session. The VAS quantified LBP on a numerical scale with text descriptors from 0 (none) to 10 (agonizing pain). During the first session, the eiLBP group reported a VAS of 2.54 ± 0.91, which was between a rating of 2 (annoying) and 4 (uncomfortable) and is similar to that reported for chronic LBP individuals who exhibited increased sway measures while sitting compared to controls.[2] During the second session, the eiLBP group reported a VAS of 1.16 ± 0.74, which was significantly lower than in post-exercise (paired *t*-test, $p<0.001$). All members of the control group reported a VAS of 0 during both sessions. Measurements were collected at two difficulty levels of balance as described below for each session. To quantify performance of wobble chair balance, the ability to keep the seat upright was determined, and summary sway measures of seat and trunk angle variability were calculated. See Study 2 (Chapter 3) for details on human subject data collection. These data served as input to the computer model.

The custom MATLAB (MathWorks, Natick, MA, USA) computer model consisted of a double inverted pendulum representing the chair-subject system during seated sway, and a simplified model of the seated postural control system. The model of seated postural control included an input noise gain and linear time-invariant feedback: active control with proportional and differential gains (each for two aspects of neural consideration). These control parameters were thought to represent aspects of the actual postural control system in humans (low back driven here). A custom simulated annealing optimization algorithm was used for system identification to determine the values of these control parameters by solving for parameters that best reproduce experimental summary measures of seated sway. Simulated sway was compared with experimental sway to determine how well the model reproduced experimental sway, and various optimizations were conducted to test the model for consistency and an ability to converge to different control parameters for different human subject conditions. Control parameters were

also compared between experimental sessions, groups, and difficulty levels, as well as with the fundamental measures of trunk neuromuscular control from Study 1.

Musculoskeletal model of wobble chair balance

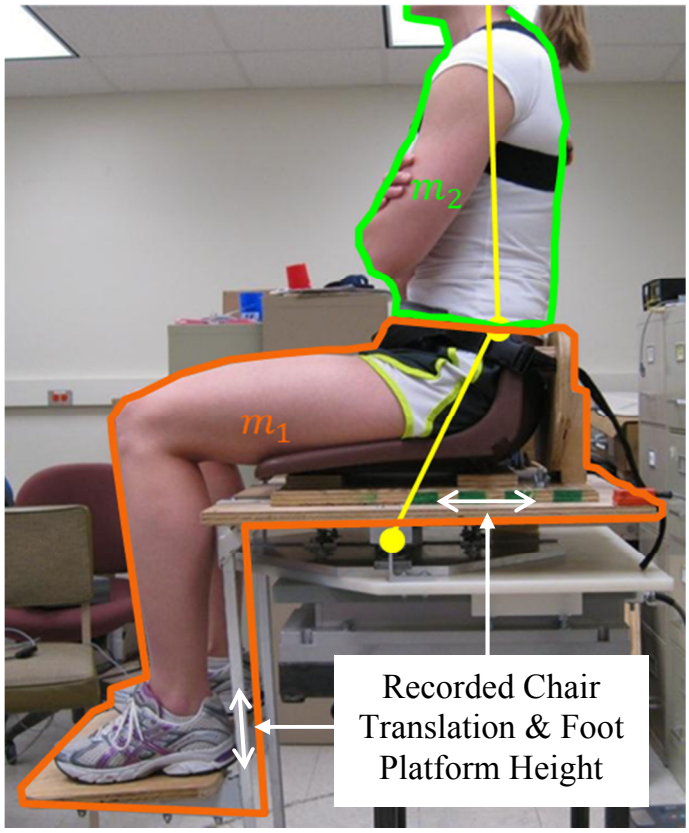
The sagittal plane model (Figure 6-1) consisted of two rigid segments oriented upright similar to a double inverted pendulum. The bottom of the double inverted pendulum was inertially-fixed and represented the pivot point under the wobble chair. The lower segment (chair segment) represented the lower body and chair moving together about the pivot axis. The upper segment (trunk segment) represented the head, arms, and trunk above the L_5S_1 joint. A frictionless pin joint between the two segments represented the L_5S_1 joint, where one net muscle moment was applied to control the motion of the model. Upper and lower segment endpoints, COM locations, and inertial characteristics were defined using known chair properties and established anthropometric relationships[16] of body segments in the experimental seated position. The neutral position of the model consisted of a horizontal seat surface and an upright trunk such that the entire chair-subject system COM was positioned directly above the pivot axis (established during initial testing by translating the chair anteriorly or posteriorly). Known reference points on the chair and recorded adjustments of the foot support and chair translation allowed for determining these specific placements of the segments above the pivot axis. Destabilizing moments arose from gravitational effects associated with small angle deviations from the neutral position. As in the wobble chair itself, elastic restorative moments were supplied by springs which were translated closer to or further away from the center pivot axis, effectively applying a rotational stiffness toward the neutral positioning of the seat surface. Equations of motion for the 2 segments were derived using Lagrangian dynamics, and the motion of the two segments were determined using a 4^{th} order Runge-Kutta numerical integration procedure.[17]

Anthropometric specifications included a weight of 71.9 kg and a height of 1.81 m for both groups, the average for all subjects. Additionally, anterior-posterior spring distances from the pivot for the model simulations were 13.6 cm for the easy level of 70% $M_{Gravity}$ and 11.0 cm for the challenging level of 45% $M_{Gravity}$, the average per level. Actual anthropometric measurements and spring distances were similar across levels and groups, yet these were controlled to allow for any differences in model control parameters for each condition to be due

only to differences in human subject sway measures within the cost function. While the custom model can determine inertial characteristics for the seated participant and chair (and thus each model segment) for any height and weight, and can simulate kinematics for any spring setting, utilizing group means also significantly decreased computational time.

Model of the seated postural control system

A feedback model of the postural control system during unstable sitting (Figure 6-2) was used to determine the net muscle moment to apply at the L_5S_1 joint of the musculoskeletal model. The L_5S_1 net muscle moment of the current wobble chair model was determined with PD control of both the chair segment and the entire chair-subject system COM kinematics, with a neutral position as the goal. Both of these were thought to be important in how the body controlled and maintained balance on the unstable seat. For example, if only the system COM was considered, the model could theoretically balance above the pivot with minimal COM deviation from vertical, but this would not restrict the seat surface from rocking back and forth outside its boundaries (at least not with a physiological net muscle moment at L_5S_1). Second, if only the seat surface position was considered for active control, it could theoretically balance about a horizontal plane, but this would not restrict the 2-segments above the pivot from collapsing to an unnatural trunk flexion. Noise was also introduced to the system, similar to Maurer and Peterka,[10] as a random disturbance torque with a normal distribution, mean of zero, and variance of one. Passive gains, as well as an active integrative gain, were not included in the model since previous models show minimal passive influence on sway measures.[10] An active sensory delay was also not employed due to an inability to find an appropriate working set of control parameters under the physiological constraints implemented here. While feedback for active control inherently has a delay, this limitation was not compounded by trying to distinguish active from more instantaneous passive feedback since passive properties were not included in the model.



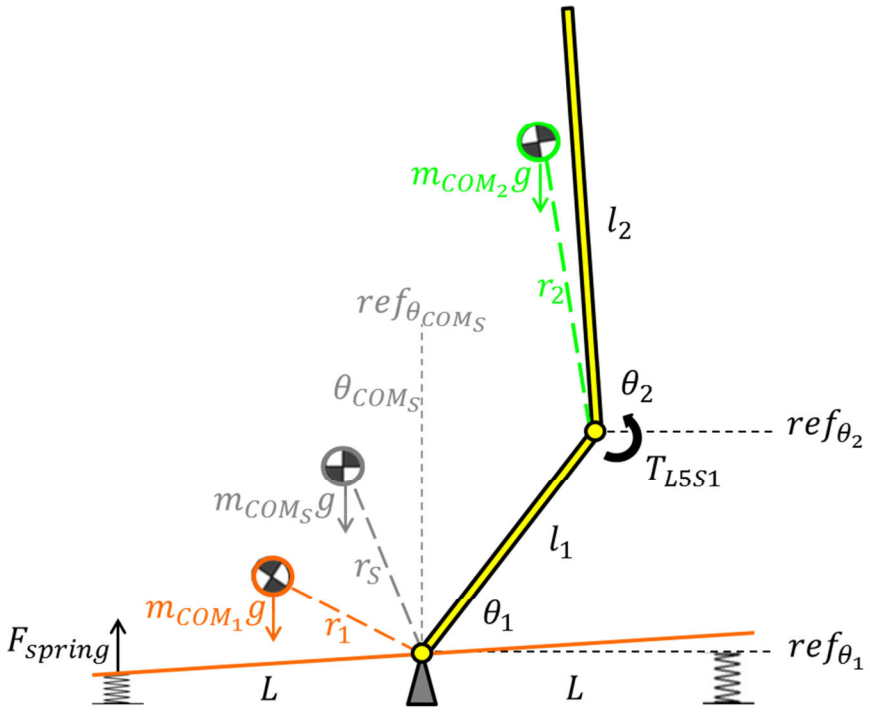


Figure 6-1 Musculoskeletal model. S = system, 1 = chair/lower body segment, and 2 = head/arms/trunk segment. Segment 1, COM_1, and the seat surface move together about the pivot axis. Segment 2 and COM_2 move together about the L5S1 joint, where a net torque (T) will be applied to control motion. COM_S has a vertical reference, and the seat surface has a horizontal reference.

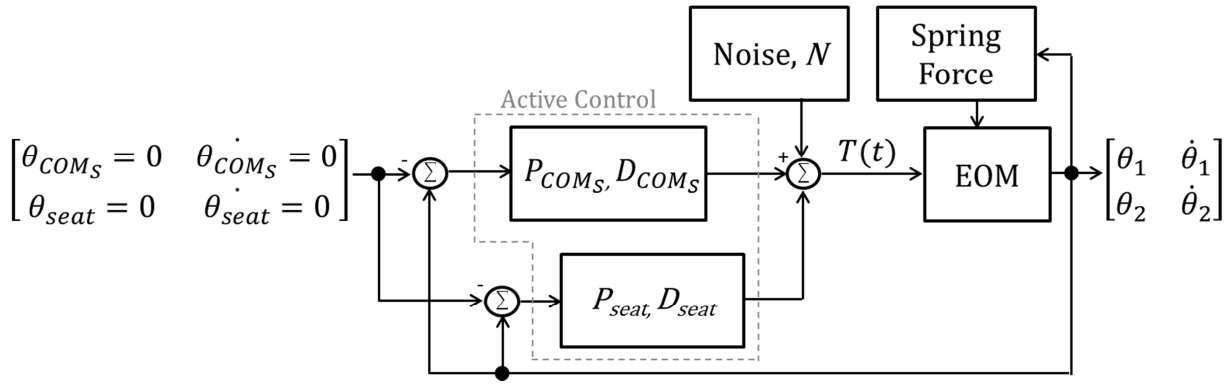


Figure 6-2 Model of the seated postural control system. Forward dynamic simulations utilizing equations of motion (EOM) influenced by an external restorative moment, due to one of the springs, and an internally applied torque (*T*) at L5S1. *T* is due to a random disturbance (noise gain *N*), as well as proportional and differential active control (gains *P* and *D*) based on deviations of COM_S from vertical and the seat surface from horizontal. S = system, 1 = chair/lower body seg, and 2 = head/arms/trunk seg.

Optimization of Control Parameters

Similar to the optimization technique seen in Qu and Nussbaum (2011),[18] a simulated annealing algorithm[15, 16] was customized to identify control parameters of feedback gain (P_{COM}, D_{COM}, P_{seat}, D_{seat}) and a noise gain (*N*) for each combination of human subject experimental conditions from Study 2: session (one was pain in the eiLBP group, two was no pain in the eiLBP group), difficulty level (easy was 70% $M_{Gravity}$, challenging was 45% $M_{Gravity}$), and group (control, eiLBP). Parameters were optimized by determining the control parameters that minimized an objective function summing the error between summary measures of seated sway collected from human subjects and summary measures of seated sway simulated with the model. Mean sway measures of seat and trunk angle variability were placed in the objective function for each condition.

Each simulation was run with set of random control parameters for 300 seconds, which is equivalent to about fifteen 20-second trials. Simulations were aborted and discarded if the net muscle moment at L_5S_1 exceeded a physiological strength constraint of 250 Nm representing a 50% male[19] or the seat angle exceeded the experimental sagittal boundaries of ±10 degrees. Simulated sway was filtered similarly as the human subject data (refer to Study 2) and analyzed over the full 300 seconds in the time domain and with 20-second blocks in the frequency domain (for consistency between human subject experimental sway measures and those simulated with

greater than 20 seconds of data). The error function included differences between measured and simulated sway for each summary measure in the objective function. To achieve a symmetrical distribution around the experimental value, the normalized error function in Equation 6.1 was utilized.

$$E = \sum_{i=1}^{n} \left| \frac{M_i - \widehat{M}_i}{M_i + \widehat{M}_i} \right| \qquad (6.1)$$

Here, n=16 was the number of seated sway measures, M_i was the reference (human subject testing) seated sway measure, and $\widehat{M}_i$ was the simulated sway measure obtained using a set of model parameters within each iteration of the optimization procedure. Specific sway measures (reference and simulated) can be found in Table 6-1. Simulated annealing customizations were: an initial temperature of 4 (where initial cost function error was ~10-12), a 0.85 temperature rate, 20 iterations between step sizes, and 100 step size adjustments between temperature changes.

Model Validation

The ability of the model to simulate realistic sway was determined by comparing simulated sway measures with the actual human subject sway measures that were placed into the objective function for identifying control parameters. Having simulated sway measures be within one standard deviation (SD) of the human subjects' sway measures provides evidence the model accurately reproduced realistic seated sway.

The ability of the model to identify control parameters from summary measures of seated sway in human subjects was determined for repeatability and an ability to identify different control parameters for different human subject conditions. To evaluate the repeatability of the model to converge to the same solution, three optimizations were conducted with different initial parameter values and the same sway measures in the cost function.

6.3 Results

A comparison between simulated sway measures from the model of wobble chair balance and the human subject sway measures (placed into the objective function) is shown in Table 6-1 for one basic condition (control group, post-recovery, easy level). Simulated sway deviation is presented as a fraction of one human subject SD from the human subject mean. Note that only simulated MaxD was different from human subject MaxD by more than one SD.

Table 6-1 Realistic Simulated Sway Measures

Measure	Human Subject Sway		Simulated Sway	
	Mean	SD	Value	Deviation
Seat Angle				
MeanD (deg)	0.484	(0.199)	0.574	0.451
RmsD (deg)	0.594	(0.217)	0.731	0.632
MaxD (deg)	2.584	(0.690)	3.829	1.805
MeanV (deg/s)	0.896	(0.302)	0.969	0.241
MeanF (Hz)	0.317	(0.083)	0.310	0.091
Power ($kdeg^2$)	0.789	(0.520)	0.789	0.000
95%PF (Hz)	0.861	(0.215)	0.985	0.576
CentrF (Hz)	0.965	(0.413)	0.779	0.450
Trunk Angle				
MeanD (deg)	0.519	(0.188)	0.411	0.575
RmsD (deg)	0.611	(0.214)	0.525	0.400
MaxD (deg)	2.454	(0.783)	2.766	0.398
MeanV (deg/s)	1.022	(0.381)	0.931	0.238
MeanF (Hz)	0.329	(0.063)	0.380	0.810
Power ($kdeg^2$)	0.827	(0.498)	0.397	0.863
95%PF (Hz)	1.067	(0.376)	1.249	0.484
CentrF (Hz)	1.167	(0.321)	0.892	0.856

Underlined indicates simulated sway deviated more than one human subject SD

Three identical optimizations (Table 6-2) were conducted for one basic condition (control group, post-recovery, easy level) to ensure consistent error minimization and therefore consistent control parameter identification. Each optimization resulted in its own random path of optimal control parameters before reaching the practically identical final values of minimized error.

Table 6-2 Repeated Optimizations

Control Parameter	Optimization Conducted		
	1	2	3
P_{COMs} (Nm/rad)	297	299	299
D_{COMs} (Nm·s/rad)	938	948	952
N (Nm)	4668	4715	4733
P_{seat} (Nm/rad)	579	584	585
D_{seat} (Nm·s/rad)	123	125	125
Minimized Error	1.6769	1.6765	1.6772

The model to identified different control parameters for different human subject conditions as seen in the eight optimizations in Table 6-3. These represent each combination of human subject experimental conditions from Study 2: session (pain or no pain in the eiLBP group), difficulty level (easy or challenging), and group (control or eiLBP). Minimized error for the normalized error function is listed for each optimization. Initial error was 10-12.

Table 6-3 Optimized Control Parameters

Control Parameter	Easy Level				Challenging Level			
	Post-Exercise		Post-Recovery		Post-Exercise		Post-Recovery	
	Controls	eiLBP	Controls	eiLBP	Controls	eiLBP	Controls	eiLBP
P_{COMs} (Nm/rad)	249	278	297	219	3791	12045	11702	704
D_{COMs} (Nm·s/rad)	681	460	938	557	635	1552	422	131
N (Nm)	3305	3210	4668	3102	12010	36070	15848	2228
P_{seat} (Nm/rad)	430	629	579	353	1600	5281	3907	320
D_{seat} (Nm·s/rad)	80	64	123	104	132	271	60	35
Minimized Error	1.315	1.781	1.677	1.762	1.824	1.666	2.191	1.902

Resulting percent differences between control parameters are shown in Table 6-4. A few trends can be observed. First, considering percent differences between groups for each level/session combination (Table 6-4A), the largest differences were due to increased eiLBP gains observed post-exercise of the challenging level, more so with proportional and noise gains than differential gains. Second, considering the percent differences from post-exercise to post-recovery for each group/level condition (Table 6-4B), the larger differences were within the challenging level for both groups. Specifically, the eiLBP group decreased gains between sessions, while the control group increased proportional and noise gains, and decreased differential gains, between sessions. Furthermore, proportional gains for the whole system COM deviation increased much more than proportional gains for the seat surface deviation across groups and sessions. Third, considering percent differences from the easy level to the challenging level for each group/session condition (Table 6-4C), the largest differences were within the eiLBP group post-exercise, again more so with proportional and noise gains than differential gains.

Table 6-4 Percent Differences between Control Parameters

Control Parameter		A. % Difference in eiLBP group from Controls			
		Post-Exercise		Post-Recovery	
		Easy	Challenging	Easy	Challenging
P_{COMs}	(Nm/rad)	12	218	-26	-94
D_{COMs}	(Nm·s/rad)	-32	145	-41	-69
N	(Nm)	-3	200	-34	-86
P_{seat}	(Nm/rad)	46	230	-39	-92
D_{seat}	(Nm·s/rad)	-21	106	-15	-42
Control Parameter		B. % Difference in Post-Recovery from Post-Exercise			
		Easy Level		Challenging Level	
		Controls	eiLBP	Controls	eiLBP
P_{COMs}	(Nm/rad)	19	-22	209	-94
D_{COMs}	(Nm·s/rad)	38	21	-33	-92
N	(Nm)	41	-3	32	-94
P_{seat}	(Nm/rad)	35	-44	144	-94
D_{seat}	(Nm·s/rad)	53	64	-55	-87
Control Parameter		C. % Difference in Challenging Level from Easy Level			
		Post-Exercise		Post-Recovery	
		Controls	eiLBP	Controls	eiLBP
P_{COMs}	(Nm/rad)	1424	4225	3841	222
D_{COMs}	(Nm·s/rad)	-7	237	-55	-76
N	(Nm)	263	1024	240	-28
P_{seat}	(Nm/rad)	272	740	575	-9
D_{seat}	(Nm·s/rad)	64	325	-52	-67

6.4 Discussion

The purpose of this study was to determine the effects of eiLBP on control parameters that characterize the neuromuscular control of the trunk while sitting on an unstable seat. Our results showed that a two-segment torque driven feedback model of wobble chair balance was able to realistically reproduce seated sway measures of both seat and trunk kinematics obtained in Study 2. The simulated annealing optimization procedure was also able to identify control parameters that commonly associate with physical aspects of neuromuscular control. The model of wobble chair balance directly relates identified control parameters and seated sway measures (Study 2). Control parameters were found to vary between human subject groups, as well as for

experimental conditions of difficulty level and session. This suggests that the neuromuscular control for a task of wobble chair balance varies within individuals and task condition, allowing for the purpose of this study to distinguish effects of pain within an eiLBP individual, as well as between the eiLBP individual and healthy controls. Additional relationships were sought between the control identified here and the fundamental measures obtained in response to sudden trunk position perturbations (Study 1), yet no observable relations were discerned. Therefore, the wobble chair model does not provide similar information as with fundamental measures of neuromuscular control of the trunk.

Active control parameters of proportional and differential gains (for both the chair segment and the entire chair-subject system COM kinematics), as well as noise gain, were sufficient for recreating realistic seated sway. Sway measures obtained from the model simulations were shown to be within 1 SD of human subject sway (except MaxD, which was within 2 SD and could result from the fact that multiple balance points exist for a two-segment system and the model goal was the neutral position). Conversely, the information contained in these seated sway measures was also sufficient for identifying different control parameters for the eight simulations representing each human subject testing experimental condition. Repeat optimizations with practically identical optimal control further supported model ability.

Utilizing human subject seated sway, the optimization procedure converged to control parameters that found general increases in proportional and noise gains in eiLBP individuals compared to controls, while the eiLBP group was experiencing pain, compared to when they were pain free, and for the challenging level compared to the easy level. Increased noise with increased difficulty level suggests insufficient sensory information (or more information than can be processed in a particular time interval) while performing a challenging balance task. Therefore, increased noise in the eiLBP group while experiencing pain could be due to sensory degradation, similar to the increased noise in modeled elderly standing sway.[10] As such, sway detection in eiLBP individuals may not be sufficient for correcting sway disturbances as compared with controls. Increases in modeled proportional gains generally coincided with increases in noise. Here the active proportional gains can be thought of as a stiffness contribution to the control of seated balance. Inherent trunk measurements and seated sway

performance consistent with this in an actual individual may imply that stiffness was compensating for increased sway (noise). While it may appear that stiffness and noise are influencing larger simulated seated sway, stiffness could be compensating for a portion of the increases in sway due to noise.

A few limitations warrant discussion. First, to minimize computational time, control parameters were obtained for each group and human subject experimental condition rather than for each individual participant at each condition. As such, group means of control parameters could not be compared using inferential statistics. Future work could include model simulations for individual seated sway measures, thus providing multiple sets of control parameters per condition for statistical comparisons. Also, these results do necessarily represent the actual neuromuscular control strategy adopted during wobble chair balance. Previous standing models have incorporated additional sensory, passive, and feed-forward mechanisms.[10,14] Thus, additional strategies likely account for some of the differences observed here in human subject seated sway during wobble chair balance. Therefore, current model results should only be considered with respect to the active feedback strategy implemented here for simulating seated postural control. Lastly, an active sensory delay was also not employed due to an inability to find an appropriate working set of control parameters under the physiological constraints implemented here. While feedback for active control is known to have a delay, it is typically delayed from passive feedback which was not included in this model.

This study showed that realistic simulations of seated sway can be produced by optimizing control parameters (those representing physiological aspects of neuromuscular control) with a simple feedback model of wobble chair balance. Control parameters were identified that reproduced human subject sway measures obtained from eiLBP and control groups, at two different difficulty levels, and for two different experimental sessions. This allowed for distinguishing effects of pain within the eiLBP individual, as well as between the eiLBP individual and healthy controls. Results include increases in proportional and noise gains for the challenging level compared to the easy level, with larger increases observed in eiLBP individuals compared to controls and while the eiLBP group was experiencing pain compared to when they were pain free. A new tool has been created for quantifying neuromuscular control of the trunk

during wobble chair balance. This tool shows that control of a functional task dependent upon trunk neuromuscular control is affected by the presence of pain within eiLBP individuals and/or eiLBP individuals while pain free compared to controls.

6.5 Acknowledgements

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7 Summary of Main Studies

Three studies were conducted on individuals who experience exercise-induced LBP (eiLBP) and healthy controls, both while the LBP group was experiencing pain and while pain free, to investigate the effects of eiLBP on neuromuscular control of the trunk. First, fundamental measures were determined following sudden flexion position perturbations. The eiLBP group exhibited higher intrinsic stiffness while not in pain (post-recovery) compared to controls, due to a decrease in stiffness within the control group from post-exercise (not associated with LBP) to post-recovery. Additionally, the eiLBP group had longer reflex delays while in pain (post-exercise) compared to controls, due to both a decrease in reflex delay from post exercise to post-recovery within the eiLBP group and an increase within the control group. Results suggest that eiLBP individuals exhibit increased stiffness compared to healthy controls, unaffected by the presence of pain, and increased reflex delays concurrent only with pain. Second, performance of seated sway was determined during a functional task of wobble chair balance. The eiLBP group exhibited impaired seat angle measures at all times, with altered trunk angle measures only while in pain and when the task was not challenging, suggesting differences compared to controls both while experiencing eiLBP, as well as while not in pain. Third, a model of wobble chair balance was created to identify control parameters of seated sway from human subjects. Results indicated increases in proportional and noise gains for the challenging level compared to the easy level, with larger increases observed in eiLBP individuals compared to controls as well as while the eiLBP group was experiencing pain compared to when they were pain free. The wobble chair model showed that control of a functional task is likely affected by the presence of pain within eiLBP individuals and/or eiLBP individuals while pain free compared to controls.

Fundamental measures, measures of seated sway during wobble chair balance, and identified control parameters using a model of wobble chair balance were all affected by eiLBP. Therefore, this study shows that some characteristics appear to be inherent to the LBP individual, while others are only concurrent with pain. Future work could extend the current model to control of subject specific seated sway, enabling statistical comparisons between conditions. Additional future work should focus on determining whether certain characteristics predispose individuals to LBP or are a result from LBP or injury.

Expected Publications

Chapter 4:	Effects of Exercise-Induced Low Back Pain on Intrinsic Trunk Stiffness and the Paraspinal Reflex Response	Journal of Biomechanics
Chapter 5:	Effects of Exercise-Induced Low Back Pain on Seated Sway	Spine
Chapter 6:	Effects of Exercise-Induced Low Back Pain on the Neuromuscular Control of Seated Sway, a Model of Wobble Chair Balance	Experimental Brain Research
previous:	Females Exhibit Shorter Paraspinal Reflex Latencies than Males in Response to Sudden Trunk Flexion Perturbations	Clinical Biomechanics 2010
	Effects of Gender, Preload, and Trunk Angle on Intrinsic Trunk Stiffness following Sudden Flexion Position Perturbations	Journal of Musculoskeletal Research 2012