Kinematic and Muscular Assessment of Spinal Stability and the Connection to Lower Back Pain

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Declaration of Originality

I declare that the contents of this thesis are my own work, conducted at Imperial College London within the Department of Civil Engineering and the Department of Surgery, except where otherwise acknowledged.
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Abstract

Previous researchers have utilized computational models and human movement analysis to improve our understanding of spinal function. However, the causes and optimal treatments for nonspecific lower back pain (LBP) remain unclear. Previous motion analysis studies into spinal disease have focused primarily on tracking spinal movements, neglecting the connection between the lower limbs and spinal function. However, nonspecific LBP is not isolated to the spinal column. The human postural response requires a complex milieu of skeletal and muscle function and therefore pain often stems from deficiencies in other areas, such as the knees and pelvis. The current lack of knowledge surrounding the functional implications of LBP may explain the diversity in success from general treatments currently offered to LBP patients.

The purpose of this study was to evaluate both the kinematic differences and variation in muscle activation patterns between subjects with a history of LBP (hLBP) (n=10) and healthy individuals (n=16) in response to a balance perturbation. Further analysis aimed to integrate these datasets to provide a clinical picture of body position in both the trunk and lower limbs. The findings support the hypothesis that there are inherent differences in movement patterns between healthy and hLBP subjects. The results revealed the use of an upper body-focused strategy in the healthy cohort for balance control. Specifically, this involved activation of the erector spinae and external oblique muscles, thus resulting in a significantly smaller range of motion at the lumbar spine. The hLBP cohort implemented a lower limb-focused strategy, relying on activation of the semitendinosus and soleus muscles. Based on the findings in this study, further work is recommended to 1) better refine the spinal model, 2) compare hLBP subjects to patients acutely suffering from LBP, and 3) investigate a wider range of muscles to more robustly investigate muscle synergies.
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<td>ASIS</td>
<td>Anterior superior iliac spine</td>
</tr>
<tr>
<td>CoP</td>
<td>Center of Pressure</td>
</tr>
<tr>
<td>DDD</td>
<td>Degenerative disc disease</td>
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<tr>
<td>EMG</td>
<td>Electromyography</td>
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<td>EO</td>
<td>External oblique</td>
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<td>ES</td>
<td>Erector spinae</td>
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<td>FP</td>
<td>Force plate</td>
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<td>GM</td>
<td>Gluteus medius</td>
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<tr>
<td>GRF</td>
<td>Ground reaction force</td>
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<tr>
<td>HJC</td>
<td>Hip joint center</td>
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<tr>
<td>hLBP</td>
<td>History of lower back pain</td>
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<tr>
<td>ICC</td>
<td>Intraclass correlation coefficient</td>
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<tr>
<td>IK</td>
<td>Inverse kinematics</td>
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<tr>
<td>IVD</td>
<td>Intervertebral disc</td>
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<tr>
<td>LBP</td>
<td>Lower back pain</td>
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<tr>
<td>LES</td>
<td>Lumbar erector spinae</td>
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<td>MRI</td>
<td>Magnetic Resonance Imaging</td>
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<tr>
<td>NICE</td>
<td>National Institute for Healthcare and Excellence</td>
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<tr>
<td>NSLBP</td>
<td>Nonspecific lower back pain</td>
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<tr>
<td>PSIS</td>
<td>Posterior superior iliac spine</td>
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<td>RA</td>
<td>Rectus abdominus</td>
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<tr>
<td>RoM</td>
<td>Range of motion</td>
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<td>SBST</td>
<td>Start Back Screening Tool</td>
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<td>TMS</td>
<td>Transcranial Magnetic Stimulation</td>
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<td>Transversus abdominus</td>
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<td>VL</td>
<td>Vastus lateralis</td>
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<td>WBB</td>
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Chapter 1

Introduction

Previous researchers have utilized in vivo studies, computational models, and human movement analysis to better understand the function of the spine [11,38,50,64]. However, the causes and optimal treatments for nonspecific lower back pain remain unclear. Movement analysis, often performed using stereophotogrammetric systems, has allowed researchers and clinicians alike to quantify human movement during everyday activities [14,18,19]. Movement analysis systems are composed of markers that are visible to 8-14 cameras positioned around the subject. The output of the tracking technology is a 3-dimensional map of the subject, from which the kinematics and kinetics of the subject may be derived. Previous motion analysis studies, in which spinal problems were analyzed, have focused primarily on tracking spinal movements, neglecting the connection between the lower limbs and spinal function [37,38]. However, nonspecific back pain is not isolated merely to the spinal column. For many patients, the problem may stem from other areas, such as the knees, pelvis, or other portions of the lower limbs, as demonstrated by Giles et al in a study of leg length inequality [22]. As a result, patients are often given generalized regimens to treat back pain, with extreme variation in success between patients [4,62]. These treatments include a recommendation to remain active, personalized plans for strengthening of the abdomen and gluteal muscles, pain management, medication, and other alternative
It is has been shown that individuals with a history of lower back pain exhibit altered movement patterns that are caused by changes in both voluntary and reactive neuromuscular control [27, 30]. Therefore, an effective method for creating differentiable movement patterns is to subject a volunteer to a perturbation (postural disturbance). A perturbation causes the volunteer to react automatically and with less volitional control over their movement pattern. Previous studies have examined the response of the trunk to postural perturbations and shown that subjects with chronic non-specific lower back pain (NSLBP) exhibit several strategies distinct from the healthy cohort [25, 30]. These strategies include trunk stiffening and a delay in EMG activity by the trunk muscles.

Better understanding and quantification of these strategies will allow for clearer insight into the various sources of pain and how these areas may be targeted in rehabilitation therapies. Therefore, the major aim of this study was to investigate the kinematics (i.e. body movements) of the spine and lower limbs after volunteers have been subjected to a sudden perturbation. A secondary aim of the project is to create a computational model of the kinematics of the spine-pelvis-lower legs complex during a perturbation response. The overlying hypothesis is that distinct differences in movement strategy will be observed between healthy controls and subjects with a history of NSLBP in response to a balance disturbance, specifically pertaining to joint range of motion, timing of response to perturbation, and muscle activation. The null hypothesis is that no difference in movement strategies will be observed in response to a balance disturbance between the control subjects and subjects with NSLBP.
Chapter 2

Background

2.1 Introduction to Low Back Pain

2.1.1 Definition of Lower Back Pain

Non-specific lower back pain (NSLBP) has recently been defined as:

"not attributable to a recognisable, known specific pathology (eg, infection, tumour, osteoporosis, fracture, structural deformity, inflammatory disorder, radicular syndrome, or cauda equina syndrome).” [4]

Once a disorder of predominately westernized societies during the latter half of the 20th century, it is now a worldwide phenomenon [20] with a lifetime prevalence of 84%. However, it is estimated that less than one-third of people with NSLBP seek treatment [48]. Once thought to be associated with sciatica, NSLBP is now known to be a separate problem as sciatica is marked by radicular pain in the lower limbs and not in the back [8]. NSLBP is a symptom that cannot be associated with a single identifiable disease, thus making it difficult to treat and subsequently an important area for medical research.
2.1.2 Chronic Lower Back Pain

Chronic NSLBP is even more complex to treat than a single episode of NSLBP and can be a source of frustration for both the patient and the physician. It is estimated that 23% of the population will develop chronic back pain at some point [2]. Furthermore, 75% of patients presenting to primary care for NSLBP fail to recover completely in terms of pain and disability after one year [1], often developing into recurrent episodes of NSLBP over many years. In addition, chronic NSLBP may take several forms, with some patients reporting symptoms that fluctuate in severity with no pain-free periods and others with an episodic pattern of symptoms with intermittent periods of no pain [1].

2.1.3 Spinal & Associated Anatomy

In order to begin understanding the causes of NSLBP and the reason for the episodic patterns, it is important to appreciate the key features of spinal anatomy that make it unique from other regions of the human body. In addition, it is helpful to understand how actions or injuries of the lower limb may impact NSLBP. The vertebral column sits at the dorsal aspect of the torso and protects the spinal cord and canal. One important function of the vertebral column is to provide axial rigidity to the trunk and resistance to bending or collapse, thus affording upright walking [1]. However, rigidity is not its sole function, as the trunk must have flexibility to allow for mobility, allowing movement between the skull and pelvis. In between each vertebra lies an intervertebral disc (IVD), which permits articulation of the vertebrae and allows for bending, twisting, and sliding moments. The spine articulates with the pelvis through the sacrum and the sacroiliac joint and thus is able to transmit forces applied at the trunk into the pelvis and lower limbs.

In addition to the bones of the spine, it is essential to examine the musculature that exerts forces on the bones, thus permitting motion. The musculature of the trunk is composed of both superficial and deep muscles, each with a distinct role to play in
Figure 2.1: The major spinal muscles, including the erector spinae and multifidus. The external oblique is also visible in this image as it wraps around from the anterior trunk (adapted from the Atlas of Human Anatomy, 2010). [45]).

movement control. The superficial muscles, such as the erector spinae, control the gross movements of the trunk [1]. The erector spinae runs the length of the vertebral column and consists of a series of aggregated muscles arising from vertebrae of the thoracic and lumbar spine segments (Figure 2.1). When the trunk flexes, extends, or performs lateral bending, this muscle is predominantly used. Additionally, several abdominal muscles also contribute to the larger movements of the trunk, such as the rectus abdominus and internal/external obliques (Figure 2.2). These muscles are capable of flexing or rotating the thorax, and therefore move the lumbar spine. For smaller, postural adjustments, the trunk utilizes deeper sets of muscles. This includes the transversus and multifidus, which aid in postural control [1].
2.1.4 Possible Causes of Back Pain

Even with the current understanding of the bones and musculature of the spine, the causes of nonspecific chronic NSLBP are not well understood. The onset of NSLBP is not traumatic, unlike many other types of injuries, and therefore it is often difficult to predict the source of the pain or attribute it to a specific event or injury. In addition, NSLBP spans a wide range of occupations, including those requiring heavy manual labour and positions in which very light work is performed, making it difficult to link a certain activity to the NSLBP. The spine is a stable system, defined by its ability to return to its original position following a disturbance and thus possessing a restoring moment. The robustness of this system is influenced by the tuning of several physical parameters, including tissue degeneration, lifestyle factors, physical activity, and genetic factors. It is hypothesized that certain physical properties will increase the robustness of the spinal system and therefore maintain equilibrium and quickly respond to disturbance [51] (Figure 2.3).
One such property is the overall stiffness of the system. If a system, in this case the spine, is overly stiffened it may only respond by momentum-induced patterns. Momentum can be induced by a force acting upon the system, such as a perturbation. A less stiff system may be capable of making internal adjustments and is therefore not bound to the patterns created by externally applied forces. In the case of the spine, reduced muscle stiffness will likely allow the subject to make discrete adjustments to particular muscles in order to avoid structural damage. The topic of system stiffness in relation to NSLBP will be further addressed in the Discussion section where it will be examined in conjunction with the results of the study.

In addition to the factors of stiffness and robustness, there is little evidence of tissue damage in NSLBP, as seen by in vivo imaging or clinical examination. To further complicate issues, psychosocial issues are known to influence NSLBP reporting. Due to these factors, clinicians and researchers have found it difficult to pinpoint certain factors as causes of NSLBP. Several causes have been hypothesized and tested in numerous studies, summarized by Weber & Burton, 1986 [62] (Figure 2.4).

**Lumbar Disc Degeneration**

Several studies with large test populations have shown a strong link between intervertebral disc (IVD) health and NSLBP, with IVD degeneration correlating to increased risk of NSLBP. Degeneration of IVDs is classified as degenerative disc disease (DDD) and is associated with gross structural changes to the disc. Typically, DDD is marked by tears in the annulus fibrosis, buckling and bulging of the annulus, reduced IVD height, reduced hydration of the nucleus, and endplate defects, leading to complete structural failure of the disc [1]. In DDD, these failures are often permanent due to the low metabolic rate of adult discs and their associated low capacity for healing. The low metabolic rate results from the poor nutritional flow into the IVD environment, low blood allocation to the IVDs, and no neural supply to the region [1]. However, when examining individual patients, degeneration apparent in MRI scans cannot be linked to pain in all cases. Often, patients will have marked degenerate IVDs, yet
CHAPTER 2. BACKGROUND

Figure 2.3: Robustness of a system in response to changing system parameters (adapted from Reeves et al, 2007 [51]).

Figure 2.4: Demonstration of factors and pathways hypothesized to impact back pain (adapted from Weber & Burton, 1986 [62]).
no pain [60]. This mismatch of structure to function in the spine demonstrates that
tissue-level physiology may not provide a complete explanation for NSLBP symptoms.

Lifestyle & Physical Activity

It has been demonstrated that lifestyle factors such as obesity and smoking increase
the risk for the development of NSLBP [53, 54]. However, the mechanism behind the
increased risk has yet to be fully identified. Several studies have alluded to the poss-
sibility of spinal disuse and deconditioning as driving forces behind NSLBP in obese
cohorts, although this has not been proven. It has also been suggested that extra loads
carried by obese populations leads to postural compensation, further contributing to
NSLBP [24]. In smoking populations, it has been suggested that smoking leads to
reduced perfusion to and thus malnutrition of IVDs due to vasoconstriction. Addition-
ally, reduced blood flow to IVDs may lead to slower healing and thus more IVD
lesions. Systemically, smoking leads to up-regulation of pro-inflammatory cytokines,
which have been shown to amplify pain [54].

Genetic Factors

Various studies have shown that genetic factors play a role in the development of
both lower back pain and IVD degeneration [32]. Notably, heritability studies have
estimated genetic contribution to NSLBP to be approximately 30-46% when inves-
tigating twin pairs [5]. Therefore genetic composition may be a contributing factor
when examining low back pain as certain cohorts may be predisposed to the condition.

Psychosocial Elements

The psychosocial components of NSLBP have been studied in several cohorts, partic-
ularly the elements of psychological distress, depressive mood, and somatization [49].
Importantly, it has been found that these factors play a role in the progression to
chronicity in NSLBP. Distress is arguably the most robust predictor of an unfavorable
outcome, and was found to be a significant predictor of NSLBP, independent of pain
and function at baseline [10]. Studies of NSLBP in the workplace indicate several psychosocial risk factors, including a poor work social environment and a mismatch between job and education level [33]. The interplay of psychosocial and personality factors is an important element in the study of NSLBP and has been thought to play a strong role in the evolution from acute pain to chronic NSLBP.

**Damage Mechanisms**

It is well established that mechanical forces have a role in NSLBP through mechanisms of non-traumatic structural damage [1]. Mechanical damage implies a disruption to a particular structure, thus permanently weakening the ability of the tissue to withstand mechanical loading. In non-traumatic injury, or injury that occurs slowly over time, it is thought that most degeneration occurs through the mechanism of “micro-damage”. However, in biological systems, small amounts of micro-damage can initiate helpful adaptive remodeling, therefore forming stronger tissues. In order for fatigue failure to occur, damage must occur faster than remodeling is able to repair the tissue [1]. The most common mechanical failures of the spine are vertebral compression fractures, particularly at the endplate, osteoporotic compression fractures of the vertebral body, internal IVD disruptions, disc prolapse, and spondylolysis. Although mechanical damage is an important factor in the development of NSLBP for some patients structural damage does not always lead to the symptom of NSLBP. Likewise, symptoms of NSLBP are not always accompanied by structural failure [60]. It is possible that certain portions of the population possess greater robustness in particular components of the musculoskeletal system, thus offsetting IVD damage.

**2.2 Current Back Pain Treatment & Management**

Because NSLBP is a symptom with unknown cause, there is no consensus about the best treatment practices. In the United Kingdom, the National Institute for Healthcare and Excellence (NICE) issued a set of guidelines for the treatment and management
of recurrent lower back pain (http://www.nice.org.uk/CG88). The guidelines give several treatment options, including physical activity, manual therapy, acupuncture, or psychological treatment. It also specifies that patient preference is a major factor in the treatment decision. However, the most effective forms of physical therapy, exercise, and manipulation for NSLBP patients remain unclear [34].

These challenges exist because the mechanisms behind NSLBP and the physiological effects of current treatment practices are unknown. Evaluation of thirteen countries in 2010 determined that the primary clinical recommendations to patients are to remain as active as possible and reduce pain with paracetamol/acetaminophen [35]. The same review study found that consistent features of early treatment were gradual activation of patients, discouragement of bed rest, and recognition of psychological factors in chronic back pain patients. However, this review found very little consensus on a rehabilitation program that is appropriate for individuals suffering from back pain or any evidence that one form of exercise is superior to another.

In 2011, Hill et al. published the results of a stratified primary care strategy for chronic lower back pain, entitled STarT Back [26]. Start Back Screening Tool (SBST) stratifies patients according to prognosis (low, medium, or high risk). The method was shown to be effective, with lowered rates of prolonged disability for patients in the STarT Back program when compared to a control group in conventional primary care regimens. In addition, improvement was seen in secondary measures, including physical and emotional functioning, pain intensity, quality of life, days off from work, and treatment satisfaction. Although STarT Back demonstrates a strong leap forward for back pain management, the root cause of chronic lower back pain continues to elude researchers and clinicians. This gap in understanding demonstrates a need for additional research in the area of NSLBP, particularly into the movement patterns of patients and how they differ from individuals without NSLBP. Perhaps with this additional information, therapeutic interventions can be better targeted, leading to higher rates of recovery, and fewer patients who are permanently disabled by NSLBP.
2.2.1 Bergmark’s Theory

The concept of postural muscle activation, as explored by Anders Bergmark, focuses on posture as an active measure controlled by various deep muscles. The spine is a highly complex structure, containing both active and passive components. The passive components, such as the bony and cartilaginous elements, ligaments, tendons, and fascia, exhibit force-dependent deformation. However, the active components, such as muscles, do not follow this rule and thus introduce greater complexity to the system [7].

In order to mechanically model the spine, Bergmark suggests a separation between local (deep) and global (superficial) muscle systems. Muscles of the local system, including all muscles with an origin or insertion at the vertebrae, control the curvature and stiffness of the spine. This can be considered as postural control or mechanical stability. Muscles in the global system transfer load directly between the thorax and the pelvis and respond to external loading conditions.

2.2.2 Current Research in Spine Stability

Almost two decades later, the concept of spinal stability within the context of a dynamic system continues to be researched and debated. Reeves et al. (2007) explored the concept of spine stiffening strategies and questioned if a stiffer spine would reduce the risk of injury [51]. During some functional tasks, such as in contact sports, slightly increased trunk stiffness can be an asset because the spine is protected from injury. (Though even in sport a completely activated spine is likely undesirable.) However, in circumstances where fine motor control is required, such as during balance or gait, it is possible that less stiffness would be optimal. Reduced stiffness and increased flexibility allow for loads to be transferred in multiple ways, thus reducing repetitive motions and possibly reducing injurious movements.

By Reeves definition the spine is inherently unstable due to its inverse-pendulum shape and therefore in order to maintain stability the spine is highly dependent on feedback.
control. Mechanisms of spinal control are highly complex and require constant supervision from monitors such as muscle spindles and other mechanoreceptors embedded within tissues. The three central aspects of the spinal control system are the IVDs, trunk muscles, and central nervous system (reflex and voluntary) (Figure 2.5).

However, examining particular parts of the spinal control system has proven to be challenging due to the heterogeneity of the NSLBP population. In order to more accurately study the effects of pain on movement strategies, several laboratories have utilized experimentally induced pain using saline injections [27]. The results of Hodges et al. experiments suggest that experimentally induced pain is capable of affecting the feed-forward postural activity of the trunk muscles, particularly the transversus abdominus (TrA). Unfortunately, experimentally induced pain is fundamentally different from clinical pain and therefore NSLBP researchers cannot rely on this technique alone to investigate the spinal control system.
2.3 Previous Back Pain Biomechanics Research

Research into the causes of and treatments for NSLBP span many areas of medicine, including pharmaceuticals, physical therapy, biomechanics, pain management, psychology, chiropractic manipulation, and soft tissue properties. The focus of this review is to introduce previous biomechanics research of the spine and demonstrate areas where further work is necessary.

2.3.1 Inverse Pendulum Model

Previous work has modeled the spine as an inverted pendulum in which a slender column supports the load of the upper body [41, 51]. To maintain stability, an inverted pendulum requires a shifting base [51]. This condition for stability can be illustrated by balancing a pencil, point down, on a fingertip; when the pencil begins to fall; movement of the finger adjusts the center-of-gravity over the point, restoring stability [40]. In the spine this base is the pelvis and lower limbs; thus the spine can be stabilized, in the sagittal plane, by flexion of the hip, knee and ankle joints; for three-dimensional stability, lateral bending at some or all of these joints will be involved. However, it is unlikely that the spine consists of a single pendulum operating about a single point at the pelvis. A more robust model would require multiple pendulums balancing atop one another, each with a shifting base as well as rotational motion and friction at each level. These may be representative of each individual vertebrae or regions of the spine, such as lumbar and thoracic vertebral sections. Multiple pendulums must exist because the spine is not a rigid vertical column, but is curved and capable of changing its curvature as a result of muscle action [3]. This becomes an incredibly complex problem to solve and has been shown to be statically indeterminate [65] because there is more than one strategy that can be utilized for adopting a particular posture.

When considering the idea of either a single pendulum or compound pendulums as
a model of the spine, it is important to remember that the pendulum is a damped oscillator due to forces originating within tissues surrounding the spine. For example, the muscles and ligaments may be modeled as springs and the joints as dashpots. In prior experiments, the trunk has been compared to a mass-spring-damper system in response to a small perturbation [39]. This implies that when the pendulum is displaced, it will eventually return to its equilibrium position. The behavior of the system is thus determined by the damping ratio, rendering the system overdamped, critically damped, or underdamped. Therefore, in addition to the stiffness of the system playing a critical role in behavior, a second input of damping is hypothesized to be integral for spine stability [51]. These two inputs represent position-related feedback (stiffness) and velocity-related feedback (damping).

The human body, however, does not consist of a dashpot apparatus and thus there must be other mechanisms through which damping is achieved. The inverse pendulum spine is stabilized by the muscles surrounding it and their behavior is adjusted by the parameters of both stiffness and damping coefficients. Therefore, it can be hypothesized that muscles must exhibit adaptive control and agonist and antagonist muscles must be utilized to achieve the appropriate return path to equilibrium.

2.3.2 Existing Musculoskeletal Models of the Spine

In the last decade, the options for detailed musculoskeletal modeling have greatly expanded, leading to the development of several models of the spine [11, 17, 64]. These models vary in the details incorporated, ranging from skeletal bone to the incorporation of muscles and IVDs.

There exist several small-scale finite element models of the IVD [52, 64]. Model predictions in these studies allowed for an estimation of IVD stress distributions as well as investigation of pressure and swelling within the tissue. Although these studies demonstrated good agreement with in vivo and in vitro measurements, the results of these models are limited due to the exclusion of the surrounding tissue and verte-
brae normally present in the living human trunk. Research teams have also developed
detailed models of certain regions within the spine, such as the cervical [17] and lum-
bar [11] segments. These provide excellent predictive models for a particular region,
but often neglect the contributions from other regions of the body. The lumbar spine
model developed by Christophy et al. in OpenSim has the ability to connect with
other OpenSim models, thus giving it the potential to incorporate loading generated
elsewhere in the body.

2.3.3 Joint Kinematics, Forces, and Moments

In order to understand the mechanical influences behind NSLBP, many researchers
have turned to analysis of joint kinematics, forces, and moments to examine the inher-
etent differences in movement between healthy individuals and NSLBP patients. The
earliest studies utilizing motion analysis techniques focused on everyday movements,
including gait, sit-to-stand, and forward bending. During gait, NSLBP subjects ex-
hibit increased muscle stiffness and less variability at the pelvis and lumbar spine
in the transverse plane [37]. However, a larger range of motion and more variable
movement was observed in the frontal plane for NSLBP subjects, particularly at the
highest, most uncomfortable gait velocities. A second study by the same group further
demonstrated that NSLBP subjects showed a reduced ability to adapt to high gait ve-
locities, specifically in trunk-pelvis coordination [37]. In a study of forward bending
it was shown that NSLBP subjects demonstrated greater use of the lumbar spine in
the early stages of forward bending in comparison to hip motion [21]. In contrast, the
healthy subjects relied primarily on hip motion throughout the entire task.

More recent studies have come to focus on postural disturbances due to its effec-
tiveness in creating non-voluntary responses and thus differentiable movement pat-
terns [9, 25, 30, 31, 56]. A perturbation causes the volunteer to react automatically
and not consciously control their movement, unlike voluntary tasks. Various research
groups have introduced a postural disturbance in different ways, including seated per-
turbations [6, 50], load-driven disturbances [42], and standing perturbations using a moving platform [9, 31]. Several studies have shown compromised movement patterns in NSLBP patients, including stiffening of the L5-S1 joint and delayed response in spinal movement [42]. In addition, one study has shown that NSLBP subjects have shorter peak torque latencies, or earlier onset of corrective torques, in the flexion/extension plane of the trunk [31].

2.3.4 Muscle Activation

Multiple studies have demonstrated a marked change in muscle activation in populations suffering from NSLBP [9, 30]. One of the most studied spinal muscles is the erector spinae for its role in spine extension and ease of detection by surface electromyography (EMG). NSLBP sufferers have been shown to exhibit a higher resting activation at the erector spinae [30, 36] as well as heightened activation during dynamic movements [31]. It has been hypothesized that increased muscle activation allows for greater spinal stability for individuals with NSLBP [59]. This has been labeled as a trunk stiffening strategy [31] and extends beyond the erector spinae to include muscles such as the internal and external obliques, rectus abdominus, tibialis anterior, and gastrocnemius. This was demonstrated by comparing the muscle activation of healthy controls to that of NSLBP sufferers in response to a balance disturbance in several directions [31].

It is also important to understand the motor patterning of muscle firing as it relates to NSLBP. Recently, it was found that NSLBP sufferers develop a top-down approach to muscle activation, where the lumbar extensors are activated prior to the gluteus maximus [44]. The pain-free control cohort demonstrated a more typical “bottom-up” approach, where the gluteus maximus is recruited prior to lumbar extensor activation. This study demonstrates significant neuromuscular control differences in the NSLBP cohort as opposed to a healthy population during everyday activities, such as standing.
In addition to investigating differences in muscle activation and patterning, it is of particular interest to better understand any inherent changes to the corticomotor excitability of the trunk muscles in NSLBP patients. Hodges & Tsoa have shown this to be the case while studying transversus abdominus (TrA), external obliques (EO), and lumbar erector spinae (LES). While using transcranial magnetic stimulation (TMS) and measuring muscle response using EMG, it was shown that the deep trunk muscle TrA showed reduced excitability in the NSLBP group in comparison to control subjects [58]. Conversely, the more superficial muscles, EO and LES, demonstrated increased excitability during pain. These findings are particularly thought provoking because it means that not all muscles change in one way in patients with NSLBP. Instead, the response is quite variable between muscles.

Researchers have also begun to explore the connection between the lower limbs and the spine, as opposed to the vertebral column in isolation from the rest of the body. Jones et al found that in some directions of balance disturbance, muscles of the lower limbs also have a heightened activation level in NSLBP sufferers [31]. The Jones et al study also demonstrated that these neuromuscular alterations also persist between pain periods, indicating a marked difference in movement. However, it is currently unknown whether these differences represent movement patterns that have formed in response to NSLBP or if they demonstrate underlying deficiencies that lead to the development of NSLBP itself.

2.4 Project Outline

The overall aim of this Masters thesis is to examine the kinematic and muscular responses of healthy volunteers and NSLBP patients to an unanticipated perturbation. It is hypothesized that a better understanding of the differences between these two cohorts will provide insight into the mechanisms of low back pain and lead to more targeted therapies and thus more successful treatment outcomes.

The following thesis is presented in five sections: Instrumentation, Computational
CHAPTER 2. BACKGROUND

Models, Clinical Testing, Results, and Discussion. The Instrumentation Chapter will present the equipment set-up as well as the implementation of the instrumentation. Topics covered will include the use of and modifications to a perturbation platform, set-up of a Vicon system, use of a Wii Balance Board, and utilization of accelerometers to measure joint angles in non-laboratory environments.

The Computational Models Chapter will cover the modeling component of the project, with particular focus on BodyBuilder and OpenSim software. The models created for this project are outlined in detail and specific codes can be found in the Appendix. This chapter also examines the strong and weak points of both modeling softwares and compares outputs from both, which is rarely done in kinematics studies.

The Clinical Testing Chapter introduces the subject characteristics, the marker and cluster set-up used for tracking joint kinematics, the way in which subjects were prepared for testing, and the perturbation platform protocol. Notably, this chapter explains the rationale behind the marker and cluster set-up and introduces a unique marker set-up that has not been used previously.

The Results Chapter gives the results of the study and begins by introducing several case studies. The case studies include two healthy subjects and two history of LBP subjects, providing a framework for comparison on an individual subject level before moving into the full cohorts. Major results of the study are presented for joint kinematics and muscle activation as measured by electromyography. In addition, several newer areas of analysis and possible future directions of research are presented in the form of ternplots and muscle synergies.

And finally the Discussion Chapter summarizes the most important learning points from this study. This chapter also revisits limitations of the study and makes suggestions for future avenues of research.
Chapter 3

Equipment Methodology

3.1 Perturbation Platform

A moving perturbation platform, developed at the University of Nottingham, was the primary piece of equipment utilized in this study. The platform is 84.5 cm in length, 61.5 cm in width, and 19 cm in height (Figure 3.1). The outer frame (blue) is made of wood and the inner moving platform is made of steel. Although the platform is capable of rotational and translation movement, for the purposes of this project the platform was fixed to a single degree of freedom (translational or uniform movement without rotation). A single piston pneumatically drives the platform with pressurized air from a compressor (Clarke International, Epping, UK). The pressurized air is delivered to the piston via plastic tubing and runs through a control panel where the direction and throttle of the piston can be adjusted. Upon piston movement, the platform, equipped with ball bearings, runs along cylindrical rails.

Prior to laboratory use, the pre-existing platform was adjusted to make it fully functional and fit for the requirements of this study. Most importantly, the original cylindrical rod rails (of an unknown, albeit softer, material) were replaced with a stiffer steel rod (LJM Linear Bearing shaft, 16 mm diameter) (RS Components, Corby, UK). The original cylindrical rods were made of a softer metal and had failed with use and
were unable to sustain wear. The ball bearings were also replaced to match the new sizing of the metal rods (LBBR Linear Ball Bearing with cap, 16 mm diameter). Finally, the platform was fixed to the outer metal frame to prevent it tilting forwards and backwards; this was accomplished using four steel G-clamps.

During testing, the platform moved with an average peak velocity of $0.33 \pm 0.03 \text{m/s}$ and an average peak acceleration of $0.81 \pm 0.40 \text{m/s}^2$. The larger variation in acceleration is an artifact of subjects with different masses and the rudimentary throttle control. The throttle controls the volume of pressurized air that is allowed to move through the pneumatic system and drive the piston. The current design of the platform permits limited control over the velocity and acceleration of the platform and requires manual control of the platform via a lever on the control panel. Although the current platform is suitable for pilot testing, as completed in this study, future work will require a more robust system. A redesign of the platform has been undertaken to address this [15].

### 3.2 Vicon System

The Vicon motion capture system (Vicon, Oxford, UK) allows for 3-dimensional optical tracking of reflective markers in a capture volume. In this project, reflective markers were placed at anatomical landmarks on the trunk and lower legs for joint angle measurement. In addition, the Vicon system is capable of interpreting third-party
CHAPTER 3. EQUIPMENT METHODOLOGY

3.2.1 Cameras

The Vicon system consists of ten T160s, a 16-megapixel motion capture camera. Each camera captures a 2-dimensional image and transmits the data to the Vicon MX Giganet hardware. The marker data was captured at a sampling frequency \( f_s \) of 100 Hz. Therefore, 100 frames of data were captured every second from each Vicon camera. Through the Giganet hardware, motion capture data was transmitted to a PC running Vicon Nexus software. The software is capable of transforming the 2-dimensional images from each camera into a 3-dimensional motion capture output of reflective markers.

3.2.2 Electromyography

Electromyography (EMG) signals \( f_s = 1000 \) Hz were collected from seven muscles of the lower limb and trunk. These signals were captured by myon electromyography (Motion Lab Systems, Baar, Switzerland) using disposable bipolar surface Ag-AgCl gel electrodes and wireless data loggers. During testing, EMG data was fed through the data loggers and transmitted via radio to the myon receiver. This connects directly to the Vicon MX Giganet and thus is synchronized to the Vicon data. In post-processing, any DC offset observed was removed from the data. Subsequently, the data was band-pass filtered using a 4\(^{th}\) order filter between 10 and 400 Hz. Finally, the signal was rectified and a linear envelope was constructed using a 4\(^{th}\) order low-pass filter of 6 Hz.

Processed EMG signals were normalized to muscle activation during quiet standing as opposed to the more conventional method where EMG signals are normalized to a maximum muscle output. This technique was chosen in order to minimize error due to normalization. It has been shown that subjects with LBP and other injuries are often not able to reach full maximal voluntary contraction due to pain or poor
CHAPTER 3. EQUIPMENT METHODOLOGY

3.2 Muscle activation during quiet standing for the soleus (SL), semitendinosus (SM), vastus lateralis (VL), gluteus medius (GM), external oblique (EO), rectus abdominus (RA), and erector spinae (ES).

Conditioning [61]. Alternatively, normalization using the EMG signal during a quiet standing position is far more consistent between the different cohorts. In this study, it was found that the difference in muscle activation during quiet standing between healthy controls and subjects with a history of NSLBP was not significantly different (Figure 3.2).

3.3 Wii Balance Board

The Wii Balance Board (WBB) (Nintendo Co. Ltd., Kyoto, Japan) is a commercial balance board accessory intended for use with the Wii Fit game suite. Quickly following its release in 2007, the research community saw its potential to clinically assess standing balance [12,23]. Equipped with a pressure sensor at each of the four corners of the platform, the WBB is capable of measuring both the vertical component of force as well as the center of pressure (CoP) of the applied force. However, the WBB is not capable of measuring the shear components of the ground reaction force (GRF). Despite the inability to measure shear forces, the WBB offers several advantages over a standard laboratory force plate (FP). The WBB ranges in price from £67 to £80 and is thus much more cost-effective than standard FPs, which cost in excess of £10,000. Additionally, it is lightweight, with a mass of 4.7 kg, making it more easily transportable. Particularly important for moving platform applications, the lightweight
WBB is less prone to influences from inertial components than FP s. Finally, unlike standard FP s, the WBB is small enough to sit on top of the pre-existing perturbation platform.

### 3.3.1 Validity and Reliability

The validity and reliability of the WBB has been assessed for standing balance activities [12]. In this study, the CoP path length was compared to that measured by a laboratory-grade FP during balance on both one and two feet. The WBB was found to have excellent test-retest reliability and also showed no significant differences from a FP in its ability to accurately measure the CoP during various balance activities. The CoP path length test retest reliability within-device intraclass correlation coefficient (ICC) was 0.66 - 0.94 and between-device ICC was 0.77 - 0.89. The ICC is a descriptive statistic that can be used when quantitative measurements are made on units that are organized into groups, such as the FP measurements versus the WBB measurements.

While the results presented above provide confidence for the use of the WBB, the work presented here is the first to make use of the WBB in a more dynamic activity. Although the subject is primarily maintaining standing balance throughout the test protocol, there is an element of postural sway that may contribute shear forces that cannot be detected by the WBB. However, previous studies utilizing moving platforms have shown these forces to be relatively small (approximately 17 to 38 N in a cohort of normal adults) when compared to the vertical component, which is typically equal to or higher than body weight [28].

### 3.3.2 Configuration for Research Purposes

During normal use, force and CoP data is collected from the WBB, transmitted to a Wii Console, and interpreted by Wii Fit software. In the laboratory environment, this protocol was reconfigured for greater functionality and ease of use. Data collected by
the WBB was transmitted via Bluetooth to a laptop computer. It was subsequently read into LabVIEW software (National Instruments, Austin, TX) and interpreted by a preexisting NET managed library (WiimoteLib, developed by Brian Peek of ASPSOFT, Inc.). The open source LabVIEW interface, WiimoteManage (National Instruments), was augmented to output force readings from each of the four sensors in Newtons, a time stamp, and the elapsed data collection time.

### 3.3.3 Calibration

To ensure accuracy of the WBB in collecting GRFs and the CoP, the WBB was calibrated both in October of 2012 and again in May of 2013 to examine if any drift occurred. The WBB was calibrated over the range of 0 to 100 kg at 10 kg intervals and at 9 different points on the surface of the board (Figure 3.3). The calibration was performed using a vertical point loading apparatus with a stylus for directing the application of force and a loading shelf on which to add mass (Figure 3.4, developed by Murphy et al [43].

At each loading level and position, the mass was allowed to settle for 10 seconds,
thus ensuring a steady signal over which to average the readings from the WBB sensors. After calculating the mean signal at each sensor for each loading level/position combination, the vertical force component and CoP were calculated using the methods of Murphy et al. In Matlab (Mathworks, Natick, Massachusetts, USA), the left divide function was used in a minimization-of-errors process. For calculation of both the magnitude of force and the CoP, the measurements from each sensor were placed in an \( n_1 \times 4 \) matrix, where \( n_1 \) is the number of data points collected throughout calibration and 4 represents the four WBB sensors. Subsequently, an \( n_1 \times 1 \) vector of the known forces or an \( n_1 \times 2 \) vector of positions was created and the left divide function was used to find a \( 1 \times 4 \) matrix of calibration constants. The calibration matrix was used to properly scale all future outputs from the WBB.

In the first calibration (October 2012), the WBB was found to be most accurate in the range of 40 to 90 kg (392 to 882 N). The calibration coefficients calculated in this range exhibited a root mean square error of 15.2 N and 1.42 cm in both the x and y-directions. In the second calibration (May 2013), the WBB was again found to be most accurate in the range of 40 to 90 kg. The calibration coefficients calculated during this calibration exhibited a smaller amount of loading error, with a root mean square error of 9.71 N and a similar error in the CoP, with 1.61 cm in both the x and y-directions. Notably, the calibration coefficient matrices themselves were marginally different between the two calibration trials (Table 3.1). Although there is likely human error involved in these differences, it is possible that the WBB sensors drifted over the course of six months of testing.

### 3.3.4 Synchronization to Vicon System

Unlike the WBB, FPs (Kistler Instrumente AG, Switzerland) can be hard-wired into the Vicon System, thus ensuring synchronization between the FP, marker trajectories, and electromyography. The WBB was synchronized to the Vicon system by placing the WBB on top of a FP and delivering a sharp tap to both at the same time. The
Table 3.1: Calibration coefficient matrices for Force and CoP over two calibration trials.

<table>
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<th>Force (N)</th>
<th>X</th>
<th>Y</th>
</tr>
</thead>
<tbody>
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<tr>
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<td>0.0861</td>
</tr>
<tr>
<td>May 2013</td>
<td>2.4162</td>
<td>-0.1078</td>
<td>-0.1036</td>
</tr>
<tr>
<td></td>
<td>2.6351</td>
<td>0.1096</td>
<td>-0.1035</td>
</tr>
<tr>
<td></td>
<td>2.7813</td>
<td>-0.1108</td>
<td>0.1044</td>
</tr>
<tr>
<td></td>
<td>2.4158</td>
<td>0.1046</td>
<td>0.0994</td>
</tr>
</tbody>
</table>

spike produced by the sharp tap in both data sets was aligned, thus ensuring alignment between the WBB and the other components of the Vicon System.

### 3.3.5 Comparison to Pressure Insoles

When measuring CoP and GRFs, it is optimal to take these measurements with a single FP under each foot. This allows one to analyze the CoP and force contribution of the individual feet. Measuring these parameters is particularly important when using the data as an input to an inverse dynamics model. Due to its size, the perturbation platform was able to fit only one WBB and thus collect the global CoP and GRF. However, it is possible to achieve an estimation of the contribution of each foot using the global CoP and the location of the feet as detected by Vicon markers (Figure 3.5). This was achieved based on the assumption that the subject placed equal weighting on each foot. Subsequently, the global CoP was aligned with the middle of the board in order to account for feet that were slightly off-centered. Finally, the CoP was superimposed on to the right foot by finding the midline of the foot using the X components of the first and fifth metatarsal markers. Although this estimation method is not to the same quality as using a laboratory grade FP, the small movement of the CoP in the X direction (perpendicular to platform movement) is minimal and thus does not vary greatly throughout the trial.
To substantiate this estimation, pedar insoles (Novel GmbH, Munich, Germany) were utilized in a validation experiment. The pedar insoles were placed on top of the WBB and the entire apparatus was placed on top of the perturbation platform. A healthy subject was asked to stand directly on top of the pedar insoles while the platform moved through a normal test sequence in both the anterior-posterior and medial-lateral directions. The CoP at the right foot was calculated from both the WBB and pedar systems (Figure 3.6). Although the two systems are certainly not a perfect match with a 12% difference in the X component and a 549.3% difference in the Y component, the general foot placement is similar. The large difference in the Y direction is likely an artifact of the larger range in the WBB Y component (seen in Figure 3.6). It is possible that the range is larger for the WBB because it is more affected by an inertial component than the light pedar insoles and thus one can observe the spikes present in the WBB data. However the comparison gives validity to the estimated x-coordinate of the CoP under the right foot. It should be noted that this comparison was performed with a single subject, and therefore additional trials should be completed to further verify the methodology.
3.4 Accelerometer and Gyroscope Testing

When using the moving platform, it was important to verify that the balance response examined in the lab was similar to balance experiences in everyday life. To accomplish this, three different measurements were compared:

i. Joint angles computed from Vicon markers and clusters recorded while on the moving platform.

ii. Joint angles computed from two accelerometer/gyroscope systems recorded while on the moving platform.

iii. Joint angles computed from two accelerometer/gyroscope systems recorded while riding on the London Underground.

The accelerometer/gyroscope systems (sampling frequency \( f_s = 128 \)) were placed at the lumbar spine and lower thoracic spine (Figure 3.7), allowing for computation of the joint angle at the lower thoracic joint. This particular joint was chosen because it is one of the spinal angles measured in this study and is more easily measured with inertial sensors than the lumbar spine. Calculation of the lumbar spine would require inertial tracking of the pelvis, which can be unreliable due to the difficulty of sensor placement.
CHAPTER 3. EQUIPMENT METHODOLOGY

Figure 3.7: Sensors were placed on both the lower thoracic and lumbar spine segments (black) and secured to the torso with flexible straps (grey).

Angles based on Vicon markers ($f_s = 100$) were computed using a kinematics model (please see Chapter 4). In Matlab, angles based on the accelerometers were calculated using an adapted version of the algorithm developed by Williamson et al [63].

The Williamson et al. technique relies on both an accelerometer and a gyroscope to calculate the angle between two segments. Using this combination of sensors minimizes drift in the signal over long collection periods. Additionally, the combination is useful for measuring activities that are non-cyclic and thus missing a period where the angular velocity is known to be zero. The initial tilt of each segment ($\theta_n(0)$) is calculated using accelerometers attached to the lumbar and lower thoracic segments of the spine and averaging over 50 samples ($k$) (Equation (3.1)). Acceleration in both the x ($a_{x,n}$) and y ($a_{y,n}$) directions were utilized in the calculation below.

$$\theta_n(0) = \frac{\sum_{k=0}^{49} \arctan \frac{a_{y,n}(k)}{a_{x,n}(k)}}{50} \quad (3.1)$$

The zero frequency offset of the rate gyroscope was also determined during the initial rest period. Following the initial rest period, the tilt of the segment ($\theta_n$) was calculated by digitally integrating the signal from the rate gyroscope ($w_n$) and then calculating the angle across the joint ($\phi_n$) (Equations (3.2) & (3.3)).
Unlike Williamson et al, nulling of the rate gyroscopes in this study was done in post-processing due to the non-laboratory data collection environment. To detect points in the data where the gyroscope should be reset, low variance regions of the accelerometer signal were identified using a search algorithm.

Although similarities can be seen between the three angle measurements, due to the nature of each measurement technique the three curves are not perfectly matched (Figure 3.8). The relationship between the angle measurement from the inertial sensor (on the tube), the inertial sensor (in the laboratory), and the Vicon markers (in the laboratory) were further explored by plotting the three variables against each other and modeling linear fits (Figures 3.9 - 3.11). The inertial sensors used on the Tube and in the laboratory were most correlated, producing a linear fit with an $R^2$ of 0.25. Interestingly, the inertial sensor used on the Tube was more correlated with the Vicon markers than was the inertial sensor used in the lab (Table 3.2). As previously stated, due to the nature of accelerometer data collection, the signals are not identical. Importantly, this experiment shows that the perturbation platform delivers a postural disturbance with a similar range of motion in the lumbar spine compared to balance disturbances delivered on public transportation. This ensures that the subjects are receiving a perturbation that is within the range of one experienced in everyday life, which is the aim of this preliminary study.
Figure 3.8: Comparison of lower thoracic joint angles for a single perturbation. A) Data collected with an inertial sensor while riding the Tube, B) Data collected with an inertial sensor on the perturbation platform in-laboratory, C) Data collected using Vicon markers on the perturbation platform in-laboratory.

Figure 3.9: Comparison between angles produced by the inertial sensor on the Tube vs the inertial sensor in the laboratory and the linear fit found between the two data sets.
Figure 3.10: Comparison between angles produced by the inertial sensor in the lab vs the Vicon markers in the laboratory and the linear fit found between the two data sets.

Figure 3.11: Comparison between angles produced by the inertial sensor on the Tube vs the Vicon markers in the laboratory and the linear fit found between the two data sets.
Table 3.2: Statistical Calculations for Inertial Sensor Comparison

<table>
<thead>
<tr>
<th>Comparison</th>
<th>$R^2$</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inertial Sensor (Tube) vs. Inertial Sensor(Lab)</td>
<td>0.25</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Inertial Sensor (Lab) vs. Vicon Markers (Lab)</td>
<td>0.05</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Inertial Sensor (Tube) vs. Vicon Markers (Lab)</td>
<td>0.20</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>
Chapter 4

Computational Modeling

Two seven-segment models were developed using Body Builder (Vicon) and OpenSim [16]. A true gold standard does not yet exist in spinal modeling and thus it was of interest to explore the various advantages and limitations each software offers.

4.1 BodyBuilder Model

A kinematics model was developed in BodyBuilder (Vicon), a modeling package associated with the Vicon Nexus platform, used for analyzing 3-dimensional marker trajectory data. Written in BodyLanguage, BodyBuilder has built-in functions for defining body segments, transforming between coordinate systems, and calculating angles between segments. A BodyBuilder model involves both Static Trial Processing and Dynamic Processing, each requiring a model. The Static Trial gathers anthropometric measurements, while the Dynamic Trial gives output of angles during a specific task. A custom seven-segment unilateral joint kinematics model was developed (Figure 4.1). The model is based on the lower limb model developed by Cleather et al. [13]. Three segments, representing the lumbar, lower thoracic, and upper thoracic portions of the spine, were added to the existing model. Ankle, knee, hip, lumbar, lower thoracic, and upper thoracic joints connected the rigid body segments. Each joint has the ability to move in three degrees of rotational freedom.
To solve for the joint angles, the model relies on input from both individual markers, placed at bony landmarks, and tri-marker clusters that are centrally placed on body segments. Using the individual markers placed at bony landmarks, local landmark frames were calculated for each body segment during a static capture. The orientation of these frames follows the lower limb model developed by Cleather et al [13]. Additionally, the tri-marker clusters were used to track segment orientation using a local cluster frame. Utilizing transformation matrices, the cluster coordinate system was transformed into the anatomical frame for each segment. Subsequently, transformation matrices were used to relate the anatomical frame of each adjacent body segment to one another, thus placing both segments in the same coordinate frame. Lastly, Euler angle decomposition was executed to calculate the flexion/extension, abduction/adduction, and axial rotation angles at each joint.

Of particular importance to the accuracy of skeletal modeling is identification of the hip joint center (HJC) [55]. Unlike the knee or ankle, the HJC is impossible to directly
CHAPTER 4. COMPUTATIONAL MODELING

track with reflective markers and therefore it must be estimated based on other pelvic measurements. Utilizing markers situated at the anterior superior iliac spine (ASIS) and posterior superior iliac spine (PSIS), the HJC was located using linear scaling compared to the Klein Horsmann data [29]. The measurements recorded by Horsman et al. include distances between bony landmarks from the right leg of a 77-year old male cadaver.

Due to the black box functionality of BodyBuilder, it was impossible to input forces not gathered through the Vicon Giganet, such as those collected by the WBB. Therefore an inverse dynamics model was not developed using this software.

4.2 OpenSim Model

The second model was executed in OpenSim, an open-source platform software developed at Stanford University. OpenSim, written in C++, allows research groups to build upon musculoskeletal models in a common platform, thus providing a consistent framework for ease of sharing.

The model consists of seven rigid body segments, representing the right leg and spine, and has 21 degrees of freedom. Of the 21 degrees of freedom, 18 correspond to joint angles and the remaining three specify the position of the pelvis. The model was scaled using 39 markers from the static experimental data collection and the corresponding virtual markers on the model. The scaling factors are calculated based on the distances between pairs of experimental markers in comparison to virtual marker position on the model [16]. In addition, the mass properties of each body segment are scaled appropriately for the overall mass of the subject.

The inverse kinematics (IK) problem is solved, including joint angles and translations, using a least-squares minimization approach. OpenSim aims to minimize the differences between the raw marker locations and the virtual marker locations on the model (Equation (4.1)). This is the squared error between subject experimental data and virtual model markers, where \( x \) corresponds to marker trajectories and \( \theta \) corresponds
to calculated joint angles. $\omega$ values give the appropriate weights to various markers or angles.

$$\text{Sq.Error} = \sum_{i=1}^{\text{markers}} \omega_i (x_{i}^{\text{subject}} - x_{i}^{\text{model}})^2 + \sum_{j=1}^{\text{jointangles}} \omega_j (\theta_{j}^{\text{subject}} - \theta_{j}^{\text{model}})^2 \quad (4.1)$$

To perform inverse dynamics calculations, the CoP and GRF collected by the WBB (and adjusted to the right foot) were inputted into OpenSim. The underlying equation of motion governing the inverse dynamics calculation is reliant upon the position, velocity, and acceleration of the body segments in question (Equation (4.2)). Here, $M$ is the system mass matrix, $a$ is acceleration, $C$ is the Coriolis and centrifugal forces, $G$ is the gravitational forces, and $T$ is the calculated intersegmental force (unknown). The output from inverse dynamics analysis is the moment at each joint of interest.

$$Ma + C + G = T \quad (4.2)$$

### 4.3 Model Comparison

Body Builder cluster-based kinematics and OpenSim optimization techniques are two of the most commonly utilized methods for joint analysis. However, they are based on different assumptions and thus offer an opportunity to examine the potential variability of joint kinematics between software packages. Body Builder operates using the direct method, relying on calculation of a segment-embedded frame from two vectors, and not considering skin movement artifacts. OpenSim operates using a segmental optimization method and thus estimates a segment’s orientation in terms of its transformation matrix, minimizing marker position deformation from its reference shape using a least squares algorithm.

To compare the two methods, joint analysis was completed for a random subset of four subjects using both Body Builder and OpenSim. The variance in kinematics at
each joint between the two models was described by finding the Pearson correlation coefficient ($R^2$). This statistic is able to demonstrate the goodness of fit between two vectors of data, with 0 indicating no fit between the two data sets and 1 indicating a perfect fit. The results of the analysis are in Table 4.1.

Table 4.1: Comparison of Seven-Segment Models in BodyBuilder and OpenSim

<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>3</td>
<td>0.96</td>
<td>0.98</td>
<td>0.99</td>
<td>0.84</td>
<td>0.55</td>
<td>0.25</td>
</tr>
<tr>
<td>13</td>
<td>0.81</td>
<td>0.99</td>
<td>0.87</td>
<td>0.04</td>
<td>0.15</td>
<td>0.01</td>
</tr>
<tr>
<td>14</td>
<td>0.93</td>
<td>0.89</td>
<td>0.80</td>
<td>0.37</td>
<td>0.79</td>
<td>0.47</td>
</tr>
<tr>
<td>18</td>
<td>0.38</td>
<td>0.97</td>
<td>0.83</td>
<td>0.90</td>
<td>0.98</td>
<td>0.95</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td>0.77</td>
<td>0.96</td>
<td>0.87</td>
<td>0.54</td>
<td>0.62</td>
<td>0.42</td>
</tr>
<tr>
<td><strong>St. Dev.</strong></td>
<td>0.23</td>
<td>0.04</td>
<td>0.07</td>
<td>0.35</td>
<td>0.31</td>
<td>0.35</td>
</tr>
</tbody>
</table>

The R-squared analysis indicates that the two models are best matched at the knee and hip joints, an example of which can be seen in Figure 4.3. The largest deviance was found in the spinal joints and there are several possible reasons for this deviation. The OpenSim model relies on the placement of virtual markers to estimate a segment’s position. These are user-selected positions, and therefore are susceptible to error, particularly for the complex spinal segments. Secondly, weighting factors are given to each marker, indicating how well the virtual markers track experimental markers. These weights are also supplied by the user and thus introduce additional variation. Although these methods can be helpful in correcting for marker placement errors, if markers are overall correctly placed during testing, the features of OpenSim may be adding error instead of making corrections.

Additionally, the large number of markers utilized for tracking of the lower limb and spine is greater than any existing OpenSim Model. The total error introduced by marker placement is quite high due to the number of markers and this may be introduced into the optimization of the model. Finally, the spinal segments are not constrained relative to one another, thus allowing two adjacent vertebrae to move in a manner that is not physiologically possible. An example of this issue is demonstrated
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Figure 4.2: Scaling of the spine within OpenSim that resulted in kinematic output that is not physiologically possible. In this example, the lumbar spine is extended while the lower thoracic spine exhibits a more flexed position.

Figure 4.3: Ankle and knee joint kinematics for Subject 3 as calculated by BodyBuilder (blue) and OpenSim (red), with an $R^2$ of 0.96 and 0.98, respectively.

in Figure 4.2, in which the lumbar and lower thoracic spinal segments become seemingly disconnected due to pronounced extension of the lumbar segment and flexion of the lower thoracic segment. Using this information, BodyBuilder was chosen as the primary software for evaluation of joint kinematics in this project. A summary of the strengths and weaknesses of both computational methods are given in Table 4.2.

The overall differences between the two models may also be visualized through the output frames from each software. A comparison of the output frames can be seen in Figure 4.4. Most notably, the BodyBuilder model (frames A-C) includes additional markers due to the use of clusters for tracking each of the segments. OpenSim relies
Table 4.2: BodyBuilder vs. OpenSim Comparison Summary

<table>
<thead>
<tr>
<th></th>
<th>BodyBuilder</th>
<th>OpenSim</th>
</tr>
</thead>
<tbody>
<tr>
<td>Method of Angle Calculation</td>
<td>Cluster-based direct method</td>
<td>Segmental optimization</td>
</tr>
<tr>
<td>Correction for Skin Movement</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Utilization of Weighting Factors</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Visual Display</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Require Static &amp; Dynamic Trials</td>
<td>Yes</td>
<td>Yes</td>
</tr>
</tbody>
</table>

on single markers and uses these to scale pre-designed bone animations. Additionally, the OpenSim model was designed to be unilateral (as seen in frames D-F), while the BodyBuilder model performs bilateral calculations. It should be noted that the OpenSim model can be easy amended for bilateral calculations. Although the tracking methodology and aesthetics are different, the overall body position and joint angles are not significantly different. Once again, although there are strengths and weaknesses to both software packages, for the purposes of this study, BodyBuilder was chosen as the main analysis tool. In the future, and with addition constraints added to the OpenSim model, it may be the software of choice for these types of analyses.
Figure 4.4: A comparison between the output frames from BodyBuilder software (A-C) and OpenSim software (D-F) for a single subject (C17). The left column (A & D) is a frontal view, the middle column (B & E) is a side view during quiet standing, and the right column (C & F) is a side view in response to a perturbation.
Chapter 5

Testing Protocol

5.1 Study Participants

16 healthy volunteers (12 female; 4 male) and 10 volunteers (6 female; 4 male) with a history of LBP (hLBP) were recruited for this study. There was not a significant age difference between the two cohorts (p=0.09). All study participants were over the age of 18 and younger than 70 years old. In addition, all participants were able to walk and stand without assistance. Volunteers were excluded from the study if they had a medical history of neurological disease or balance disorders, uncorrected vision problems, systemic infection, cardiovascular disorders, or severe musculoskeletal deformity. Volunteers were also excluded if they were currently pregnant, had an injury to the lower extremity, or had surgery three months prior to testing. **Volunteers in the healthy group had no history of lower back pain and volunteers in the hLBP group had recurrent episodes of LBP at least once per year. However, no participants were tested while in pain.** This was important both for the comfort of the subject and ensured that the results were not influenced by current pain responses. Previous studies of LBP have often tested non-specific lower back pain (NSLBP) patients that were currently in pain. These results may be confounded by the various levels of discomfort felt by the subjects during testing. Instead, testing
in this project provided a measure of subjects’ innate joint and muscle responses. Unfortunately, it was not possible to obtain accurate rehabilitation statistics from the hLBP cohort. Group participant characteristics may be found in Table 5.1. (A full description of individual participant characteristics and the questionnaire used to obtain participant data may be found in the Appendix.)

Table 5.1: Subject Characteristics

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Control Group</th>
<th>hLBP Group</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>29 ± 9.5</td>
<td>35 ± 7.7</td>
<td>0.09</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>168.3 ± 11.0</td>
<td>172.9 ± 7.49</td>
<td>0.25</td>
</tr>
<tr>
<td>Mass (kg)</td>
<td>63.5 ± 15.8</td>
<td>74.5 ± 12.6</td>
<td>0.07</td>
</tr>
<tr>
<td>BMI</td>
<td>22.2 ± 3.8</td>
<td>24.8 ± 2.6</td>
<td>0.07</td>
</tr>
<tr>
<td>Gender</td>
<td>12 F; 4 M</td>
<td>6 F; 4 M</td>
<td>–</td>
</tr>
<tr>
<td>Frequency of NSLBP</td>
<td>-</td>
<td>76.7 [1 – 365] days/yr</td>
<td>–</td>
</tr>
</tbody>
</table>

5.2 Markers & Clusters

Twenty-four individual reflective markers were utilized in the analysis of lower limb and spinal movement during a balance disturbance (Figure 5.3). In addition, a flat 3-marker cluster was placed on both the calf and thigh to track the orientation of the segment in the capture volume. On the spine, a novel cluster method was used to track the lumbar, lower thoracic, and upper thoracic segments. The clusters placed on the spine consisted of three orthogonal reflective markers (Figures 5.1 & 5.2). These were built with a machined plastic base and nylon 6.6 slot cheese head screws (RS Components).

These markers allowed for body movements to be tracked and recorded by Vicon cameras (Figures 5.4 & 5.5). Specifically, the single markers defined the subjects anatomical frame and marker triads tracked the orientation of each segment in space (Table 5.2). The marker/cluster design was developed from an existing lower limb model utilized at Imperial College. This lower limb model has been validated for gait applications in conjunction with an osteoarthritis study [18]).
Although lower limb kinematics and kinetics models are commonly used in movement research, fewer spinal and trunk models exist. As previously discussed, there are several existing musculoskeletal models of the spine, however many focus on a single region of the spine and thus do not allow for investigation of the interplay between the various regions and the lower limbs. Incorporating this interplay is integral to the understanding of activities such as balance. Other trunk models are too simplistic, treating the trunk as a single segment, when the spine truly consists of 33 individually articulating vertebrae.

Thus, in order to investigate the spinal system within the context of LBP, it was of importance to develop a marker set-up and model that tracked the entire spine while also accounting for variation in movement along the the length of the spine. It is well established that the vertebrae in a similar region typically articulate in synchronization and thus the spine can be broken into segments to approximate movement at various regions. Although this method offers a more realistic measure of spinal movement, it is still a compromise to tracking the individual vertebrae, which is only achievable using bone pins or imaging.

The spine was separated into three regions: lumbar (L1-L5), lower thoracic (T7-T12), and upper thoracic (T1-T6). The thoracic region was split into two segments in order to observe different manifestations in the upper and lower portions. Based on observations of posture, the thoracic is generally considered to be a stiffer region than either the cervical or lumbar regions. By splitting the region into the upper and lower regions, it is possible to observe any flexibility that does occur in this area, such as the lower thoracic region articulating more similarly to the lumbar region. The cervical vertebrae were not relevant to the investigation of lower back pain and thus were not included in this investigation.

One marker was placed at either side of the spinous process of each vertebra of interest, including T1, T6, T7, T12, L1, and L5. These anatomical landmarks were tracked during the static portion of the trial. A 3-marker cluster was also placed at the center of each spinal segment, which was tracked during the dynamic portion of
Figure 5.1: Spinal cluster consisting of three orthogonally placed markers and a flexible strip to fit to different spinal shapes.

the trial. Marker data was sampled at 100 Hz. In post-processing all marker data was filtered using a 4\textsuperscript{th} order, zero lag filter with a cut-off frequency of 10 Hz.
Figure 5.2: The entire spinal cluster plus base is 11.5 cm in length.

Figure 5.3: Schematic of the full marker set, viewed from both the anterior and posterior perspectives.
Figure 5.4: The lower limb marker set (left), including the foot, calf, and thigh segments, and the spinal marker set (right), including the lumbar, lower thoracic, and upper thoracic segments.

Figure 5.5: The full marker set, including the spine, lower limbs, and moving platform.
Table 5.2: Marker Placement

<table>
<thead>
<tr>
<th>Anatomical Segment</th>
<th>Marker Placement</th>
</tr>
</thead>
<tbody>
<tr>
<td>Foot</td>
<td>Calcaneus - Heel bone</td>
</tr>
<tr>
<td></td>
<td>Tuberosity of the fifth metatarsal - Lateral edge of the foot</td>
</tr>
<tr>
<td></td>
<td>Top of foot, running along the line of the big toe</td>
</tr>
<tr>
<td></td>
<td>Joint between the foot and the first metatarsal</td>
</tr>
<tr>
<td>Calf</td>
<td>Lateral malleolus - Distal-most aspect of the fibula</td>
</tr>
<tr>
<td></td>
<td>Medial malleolus - Bony process extending distally off the tibia</td>
</tr>
<tr>
<td></td>
<td>Calf cluster - Mid-way down the lateral edge of the calf</td>
</tr>
<tr>
<td>Thigh</td>
<td>Lateral chondyle - Lateral articular body of the femur</td>
</tr>
<tr>
<td></td>
<td>Medial chondyle - Medial articular body of the femur</td>
</tr>
<tr>
<td></td>
<td>Thigh cluster - Mid-way down the lateral edge of the thigh</td>
</tr>
<tr>
<td>Pelvis</td>
<td>Anterior superior iliac spine - Anterior extremity of the iliac crest</td>
</tr>
<tr>
<td></td>
<td>Posterior superior iliac spine - Posterior extremity of the iliac crest</td>
</tr>
<tr>
<td>Lumbar Spine</td>
<td>L1 &amp; L5; right &amp; left of the spinous processes</td>
</tr>
<tr>
<td></td>
<td>Cluster - Triad of markers placed midway between L1 &amp; L5</td>
</tr>
<tr>
<td>Lower Thoracic Spine</td>
<td>T7 &amp; T12; right &amp; left of the spinous processes</td>
</tr>
<tr>
<td></td>
<td>Cluster - Triad of markers placed midway between T7 &amp; T12</td>
</tr>
<tr>
<td>Upper Thoracic Spine</td>
<td>T1 &amp; T6; right &amp; left of the spinous processes</td>
</tr>
<tr>
<td></td>
<td>Cluster - Triad of markers placed midway between T1 &amp; T6</td>
</tr>
</tbody>
</table>

5.3 Subject Preparation

Reflective markers, 14-mm in diameter, were applied to bony landmarks on the lower limbs, pelvis, and back using double-sided tape according to the landmarks outlined in the previous section.

EMG sensors were applied to the skin to record muscle activation of the lower limb and trunk (Figure 5.6). The skin was prepared using alcohol wipes and 7 wireless sensors were applied to each side of the body using double-sided tape utilizing Seniam Guidelines (http://www.seniam.org/). The muscles investigated include those listed in Table 5.3 and shown in Figure 5.7.
Figure 5.6: Muscle activation was measured using wireless Myon electromyography (EMG) (Baar, Switzerland) in muscles known to integral to the balance response: glutaeus medius (GM), vastus lateralis (VL), soleus (SL), semitendinosus (SM), erector spinae (ES), external obliques (EO), and rectus abdominus (RA).
5.4 Perturbation Platform Protocol

The volunteer stood on the translating platform with safety bars on either side of the platform edge. The volunteer was instructed to maintain his or her balance through a series of 31 anterior-posterior platform movements and 31 medial-lateral movements. The postural disturbances were delivered in four sections, with a 20-second pause between each section (Table 5.4). Three types of platform movement were implemented: low frequency regular timing, high frequency regular timing, and erratic timing. The different frequencies of movement were implemented to investigate the
resulting changes in balance response. This has not been investigated in previous perturbation studies and may have an effect on experimental results. Erratic timing in both the second and fourth sections allows for investigation of learning effects throughout the trial.

Table 5.4: Perturbation Platform Protocol

<table>
<thead>
<tr>
<th>Test Section</th>
<th>Perturbations</th>
<th>Pauses (sec)</th>
<th>Timing (min:sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular, Low Hz</td>
<td>5</td>
<td>6</td>
<td>0:00, 0:06, 0:12, 0:18, 0:24</td>
</tr>
<tr>
<td>Pause</td>
<td>0</td>
<td>-</td>
<td>0:24 - 0:44</td>
</tr>
<tr>
<td>Erratic</td>
<td>10</td>
<td>2-6</td>
<td>0:44, 0:48, 0:50, 0:56, 0:58...</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1:02, 1:06, 1:12, 1:16, 1:18</td>
</tr>
<tr>
<td>Pause</td>
<td>0</td>
<td>-</td>
<td>1:18 - 1:38</td>
</tr>
<tr>
<td>Regular, High Hz</td>
<td>5</td>
<td>2</td>
<td>1:38, 1:40, 1:42, 1:44, 1:46</td>
</tr>
<tr>
<td>Pause</td>
<td>0</td>
<td>-</td>
<td>1:46 - 2:06</td>
</tr>
<tr>
<td>Erratic</td>
<td>11</td>
<td>2-6</td>
<td>2:06, 2:10, 2:12, 2:18, 2:20, 2:24...</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>2:26, 2:30, 2:36, 2:40, 2:42</td>
</tr>
<tr>
<td>Pause</td>
<td>0</td>
<td>-</td>
<td>2:42 - 3:02</td>
</tr>
</tbody>
</table>

5.5 Statistics

All p-values were calculated using the Student t-test.
Chapter 6

Results

6.1 Healthy Case Studies

Following equipment set-up and study design, two pilot subjects were tested to investigate the results that could be ascertained from perturbation platform testing. Both were control subjects without hLBP and each subject was tested two times with slightly different testing protocols. The first test session did not have a defined protocol for platform timing, whereas a defined protocol was implemented in the second test session, as well as additional movements, such as walking and sit-to-stand.

In addition to refining the protocol and gaining experience with the complex equipment set-up, the purpose of the two testing sessions was to understand if a considerable amount of variability could be found between test sessions. The variability may be a result of marker placement, subject comfort and learning, or camera placement. The pilot testing sessions were also useful for understanding inherent differences between the reactions of healthy individuals to a perturbation. Before attempting to piece together the differences between healthy controls and individuals with a hLBP, it was important to establish what constituted a normal range of movement and muscle activation. This was established through a pilot study with two healthy subjects that will be called C1 and C2 (Controls 1 and 2; in the Appendix these are Subjects 1 and 2).
In this study, flexion and extension joint motion was the focus of analyses. Subjects were instructed to stand along the axis of platform movement and thus, the majority of motion was seen in the sagittal plane. A side-view of the various segments and joints can be seen in Figure 6.1. The flexion/extension calculations were performed in this plane through comparison of the segmental coordinate frames, as illustrated in Figure 6.2. The relative difference in each plane was calculated through comparison of the coordinate frames for two connected segments.

For both C1 and C2, output angles in the sagittal plane were obtained and plotted
Figure 6.2: An illustration of the calculations performed to obtain intersegmental angles. (a) The ankle joint, comprised of the calf and the foot and (b) The hip joint, comprised of the pelvis and thigh segments. The black arrows represent the coordinate frames for individual segments while the red shading represents the relative angle between the two segments.

together in order to examine the similarities and differences between their responses to platform movement. The average angle of movement is shown for each subject and for both forward and backwards perturbations (Figures 6.3 - 6.8). Flexion is represented by a positive angle in the sagittal plane and extension is represented by a negative angle in the sagittal plane.

Figures 6.3 - 6.8 illustrate that C2 has a naturally higher degree of flexion or extension in a relaxed posture. However, the amount of movement in response to platform movement or the range of motion is similar between C1 and C2 for the majority of joints. However, Figure 6.3 demonstrates that C2 responds to a platform perturbation with greater flexion in the ankle in comparison to C1. C2 also responds to the perturbation with greater flexion at the lower thoracic joint than C1, who responds with little flexion at the lower thoracic spine (Figure 6.4). At the hip, knee, and remaining spinal joints, the range of motion is closely matched between the two pilot subjects.

In order to gain a sense of the amount of movement at each joint Table 6.1 demon-
CHAPTER 6. RESULTS

Figure 6.3: Flexion at the ankle joint in the sagittal plane (degrees) following a perturbation in the sagittal plane.

Figure 6.4: Flexion at the knee joint in the sagittal plane (degrees) following a perturbation in the sagittal plane.

strates the differences in average range of flexion/extension in response to perturbation between C1 and C2. Table 6.1 gives range of motion for both Trials 1 and 2.

C1 demonstrated an overall smaller range of motion in response to perturbation compared to C2 in both trials. The one exception to this is at the lumbar joint in Trial 1, for which C1 moved through a larger average range of motion (2.14°) compared to C2. However, the larger amount of movement in the lumbar spine of C1 disappears in Trial 2 to 0.71°. Both C1 and C2 have the greatest amount of movement at the hip joint (4.95° and 5.66°, respectively in Trial 1) which is maintained at (3.54° and 6.31°, respectively) in Trial 2. The differences in range of motion between the two subjects likely stems from differences in movement strategy in response to a perturbation.
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Figure 6.5: Flexion at the hip joint in the sagittal plane (degrees) following a perturbation in the sagittal plane.

Figure 6.6: Extension at the lumbar joint in the sagittal plane (degrees) following a perturbation in the sagittal plane.

Figure 6.7: Extension at the lower thoracic joint in the sagittal plane (degrees) following a perturbation in the sagittal plane.
CHAPTER 6. RESULTS

Figure 6.8: Flexion at the upper thoracic joint in the sagittal plane (degrees) following a perturbation in the sagittal plane.

Table 6.1: Comparison of Pilot Subjects C1 & C2 During a Forward Perturbation - Average Range of Flexion/Extension at Six Joints

<table>
<thead>
<tr>
<th>Angle (deg)</th>
<th>Trial 1</th>
<th>Trial 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C1</td>
<td>C2</td>
</tr>
<tr>
<td>Ankle</td>
<td>1.61</td>
<td>4.12</td>
</tr>
<tr>
<td>Knee</td>
<td>2.94</td>
<td>3.86</td>
</tr>
<tr>
<td>Hip</td>
<td>4.95</td>
<td>5.66</td>
</tr>
<tr>
<td>Lumbar</td>
<td>2.14</td>
<td>1.30</td>
</tr>
<tr>
<td>Lower Thoracic</td>
<td>1.74</td>
<td>4.38</td>
</tr>
<tr>
<td>Upper Thoracic</td>
<td>1.45</td>
<td>1.71</td>
</tr>
</tbody>
</table>

However, both C1 and C2 appear to compensate for the balance disturbance primarily using a hip strategy. This strategy will be discussed further in depth in comparison of hLBP subjects to control subjects.

For most angles of measure, the results between the two trials exhibited slight, but not significant differences. Both subjects exhibited similar differences in angle measures between the two trials and it was not significantly different (p=0.93). Both subjects, on the whole, decreased their RoM between trial 1 and trial 2, possibly indicating an increased amount of comfort with the platform and ability to sustain posture control. However, a mentioned previously, C2 had a slightly increased RoM at the hip and upper thoracic joints in trial 2 and C1 had increased RoM at the lower thoracic spine. Though these differences are quite small (Table 6.1), they may be indicative of a change in balance strategy when considering spinal stability and the link to LBP.
CHAPTER 6. RESULTS

However, it can be argued that there are possible sources of error that created differences between the results of the two trials. The marker placement may have varied between trials (conducted on two different days) thus influencing the placement of each body segment in space and the calculation of angles between the segments. This source of error is present in all kinematics studies utilizing bony landmark tracking.

In addition to understanding the kinematics of movement between these two subjects, the muscle activation was also examined through analysis of EMG signals. Figures 6.9 - 6.14 illustrate the activation of the soleus, semitendinosus, vastus lateralis, gluteus medius, external oblique, and erector spinae muscles. Rectus abdominus was also measured but not included in these results due to poor signal recordings. It is hypothesized that this is due to varying amounts of body fat in this region of the body, often impeding proper signal acquisition. In the signals analyzed, the muscle activation is normalized to muscle usage during quiet standing. Therefore, an activation level of 1 would be equivalent to muscle activation during quiet standing.

Figure 6.9 shows soleus activation for both Subjects C1 and C2. Here, C2 demonstrates greater muscle activation in response to platform perturbation in comparison to C1, who appears to activate the soleus only to the level of quiet standing. This indicates a distinct difference in strategy at the soleus muscle in which C2 demonstrates...
Figure 6.10: Activation of the semitendinosus muscle in response to a platform perturbation, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).

Figure 6.11: Activation of the vastus lateralis muscle in response to a platform perturbation, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).
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Figure 6.12: Activation of the gluteus medius muscle in response to a platform perturbation, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).

Figure 6.13: Activation of the external oblique muscle in response to a platform perturbation, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).
greater activation and possibly greater control.

Semitendinosus muscle activation, depicted in Figure 6.10, is similar in amplitude between Subjects C1 and C2. However, it is interesting to examine the timing of muscle activation in this plot because C1 has a delayed activation in comparison to C2. This could be due to a number of factors, including feedback-related differences in motor output due to differences in proprioceptive sensitivity [30] or central nervous system differences in muscle synergies [25]. A muscle synergy is a pattern of muscle co-activation recruited by a single neural command signal. The topic of muscle synergies is also investigated in this study and is discussed further at the end of this chapter.

Two additional differences in muscle activation between C1 and C2 occur at the gluteus medius (Figure 6.12) and erector spinae (Figure 6.14) muscles. For both of these muscles, C1 shows little muscle response to platform movement and instead appears to generally remain at a level of activation equivalent to quiet standing. Alternatively, C2 demonstrates strong muscular activation in order to maintain balance on the platform, often reaching two to four times the level seen during quiet standing.

In Table 6.2, muscle activation is shown for six muscles in reaction to a forward perturbation. The muscle activation is normalized to quiet standing and thus represents
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how many times greater the activation is compared to a static position. Similarly to
the kinematics comparison, a direct match was not found between the two trials. This
is likely due to changes in subject comfort with the platform and thus response, as
well as variability of EMG electrode placement. C1 demonstrated an overall consistent
response between the two trials, except at the vastus lateralis muscle, the activation
of which was higher in Trial 1. C2 had a less consistent response, particularly at the
soleus and gluteus medius muscles. The activation of these muscles are significantly
increased in Trial 2, possibly indicating a slight change in strategy in order to maintain
balance control. Examining the kinematics data (Table 6.1), the higher activation of
the gluteus medius in Trial 2 may be a result of the better hip control demonstrated by
C2 in Trial 2. Additionally, it is likely that the spine and body have a range of strate-
gies for balance maintenance and dynamic stability, thus leading to slightly different
results between two testing sessions.

Table 6.2: Comparison of Pilot Subjects 1 & 2 During a Forward Perturbation -
Average Muscle Activation at Six Muscles

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Trial 1</th>
<th>Trial 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>C1</td>
<td>C2</td>
</tr>
<tr>
<td>Soleus</td>
<td>1.96</td>
<td>1.25</td>
</tr>
<tr>
<td>Semitendinosus</td>
<td>2.90</td>
<td>2.89</td>
</tr>
<tr>
<td>Vastus Lateralis</td>
<td>3.06</td>
<td>3.39</td>
</tr>
<tr>
<td>Gluteus Medius</td>
<td>1.57</td>
<td>3.62</td>
</tr>
<tr>
<td>External Obliques</td>
<td>2.49</td>
<td>4.32</td>
</tr>
<tr>
<td>Erector Spinae</td>
<td>1.97</td>
<td>4.57</td>
</tr>
</tbody>
</table>

These initial insights into the movement of healthy subjects helped inform the anal-
ysis of future subjects. These pilot subjects also showed that differences in response to
a perturbation exist between subjects for both kinematics and muscle activation. How-
ever, they also served to show the great variability present in a healthy population– i.e.
not every healthy person uses the same balance strategies. Finally, the results from
Subjects C1 and C2 served to identify some limitations of this type of study, including
the complexities of repeatability, both on the part of the subject and the researcher.
From the perspective of the subject, it is important that none of the participants have
experience using the platform. Such experience could skew results because a particular subject has developed a strategy through a process of learning. Secondly, from the perspective of the researcher, it is important to take the time to correctly identify the bony landmarks and muscle bellies in order to achieve results with the smallest amount of error possible.

6.2 hLBP Case Studies

After beginning to identify the range of movement in healthy control subjects, the next step in the pilot study was to begin looking at individuals with hLBP. The first subjects with hLBP will be identified here as P1 and P2 (subjects with a history of pain; in the Appendix P1 and P2 are Subjects 5 and 7, respectively). Both subjects had a hLBP, but were not experiencing pain or discomfort at the time of testing, and thus pain had no direct effect on their movement patterns. Similarly to the healthy subjects, the movement of Subjects P1 and P2 is described using joint angle kinematics in Figures 6.15 - 6.20.

Notably, Subjects P1 and P2 were not significantly different in their movement strategies (Table 6.3). However, at the hip joint, P2 demonstrates a greater degree of flexion both during perturbation and rest (Figure 6.17).

Table 6.3: Comparison of Pilot Subjects P1 & P2 During a Forward Perturbation - Average Range of Flexion/Extension at Six Joints

<table>
<thead>
<tr>
<th>Angle</th>
<th>P1</th>
<th>P2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankle</td>
<td>3.10</td>
<td>2.55</td>
</tr>
<tr>
<td>Knee</td>
<td>3.59</td>
<td>2.12</td>
</tr>
<tr>
<td>Hip</td>
<td>2.94</td>
<td>7.53</td>
</tr>
<tr>
<td>Lumbar</td>
<td>2.79</td>
<td>1.45</td>
</tr>
<tr>
<td>Lower Thoracic</td>
<td>1.28</td>
<td>2.19</td>
</tr>
<tr>
<td>Upper Thoracic</td>
<td>1.44</td>
<td>2.10</td>
</tr>
</tbody>
</table>

The most notable region of movement for both cohorts was at the hip joint. For both the healthy controls (C1 and C2) and hLBP pilot subjects (P1 and P2), this was the joint exhibiting the greatest RoM in response to perturbation (Table 6.4).
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Figure 6.15: Flexion at the ankle joint in the sagittal plane (degrees) in subjects with hLBP.

Figure 6.16: Flexion at the knee joint in the sagittal plane (degrees) in subjects with hLBP.

Figure 6.17: Flexion at the hip joint in the sagittal plane (degrees) in subjects with hLBP.
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Figure 6.18: Extension at the lumbar spine joint in the sagittal plane (degrees) in subjects with hLBP.

Figure 6.19: Extension at the lower thoracic spine joint in the sagittal plane (degrees) in subjects with hLBP.

Figure 6.20: Flexion at the upper thoracic joint in the sagittal plane (degrees) in subjects with hLBP.
This is not unexpected because the degree of hip movement is influenced by a number of muscle groups, including the abdominal and spinal muscles, as well as the gluteal muscle groups. Each of these muscles has previously been studied and implicated in the manifestation of LBP [9, 37]. Interestingly, C2 and P2 exhibited substantially larger RoM at the hip joint compared to C1 and P1. Instead, C1 and P1 exhibited a more well-distributed RoM, particularly relying on ankle and knee movement to maintain stability.

Table 6.4: Comparison of Hip Range of Motion Between Healthy and hLBP Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Hip Range of Motion (deg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1 - Healthy</td>
<td>3.54</td>
</tr>
<tr>
<td>C2 - Healthy</td>
<td>6.31</td>
</tr>
<tr>
<td>P1 - hLBP</td>
<td>2.94</td>
</tr>
<tr>
<td>P2 - hLBP</td>
<td>7.53</td>
</tr>
</tbody>
</table>

A second notable area of movement was found in the lumbar spine. Subjects C1 and C2 exhibited relatively little movement in this region (particularly C1), whereas Subjects P1 and P2 demonstrated greater lumbar range of motion. These results are summarized in Table 6.5, where the healthy subject results are taken from Trial 2 because it utilized a protocol identical to that for Subjects P1 and P2. Greater RoM is also seen at the ankle joint in the hLBP case studies. A smaller RoM is seen at the lower thoracic joint in the hLBP case studies when compared to the healthy cases.

Table 6.5: Comparison of Lumbar Range of Motion Between Healthy and hLBP Subjects

<table>
<thead>
<tr>
<th>Subject</th>
<th>Lumbar Range of Motion (deg.)</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1 - Healthy</td>
<td>0.71</td>
</tr>
<tr>
<td>C2 - Healthy</td>
<td>1.19</td>
</tr>
<tr>
<td>P1 - hLBP</td>
<td>2.79</td>
</tr>
<tr>
<td>P2 - hLBP</td>
<td>1.45</td>
</tr>
</tbody>
</table>

EMG measurements were also obtained from Subjects P1 and P2 and are displayed in Figures 6.21 - 6.26. Overall, P1 reacts to platform perturbations with higher muscle activation than P2. In addition to higher average muscle activation, P1 also has a
greater diversity in the strength of muscle activation compared to P2, who has a small range of activation strength. This is illustrated by the maximum and minimum activation curves that accompany the average activation curves in Figures 6.21 - 6.26. An exception to this is at the external oblique muscle, which P2 activates to a much higher level compared to baseline than any other muscle (Figure 6.25). As discussed with respect to the two healthy case studies, this may be a result of differences in muscle synergies or muscles that are activated as a group. It has been suggested that individuals with hLBP have central nervous system changes in muscle activation patterns [30], but it is likely that these differences are not consistent for all people suffering from LBP. This hypothesis is further explored later in this chapter in the Muscle Synergies section.

Collectively, the kinematics and EMG datasets demonstrate that not all people who experience LBP move in the same way, similarly to how two healthy people do not react to balance disturbances in the same manner. Additionally, if one examines the the maximum and minimum curves for both healthy and hLBP subjects, one will see that even the same subject does not react to every perturbation using the same strategy or with the same magnitude. This suggests that consistency in balance maintenance strategy may also provide information about spinal health. Based on these observations from both the pilot control subjects and the first several hLBP subjects, further testing was undertaken to understand the range of strategies utilized by healthy and hLBP subjects. The results from the full trial are discussed in the subsequent sections.
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Figure 6.21: Activation of the soleus muscle in subjects with hLBP, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).

Figure 6.22: Activation of the semitendinosus muscle in subjects with hLBP, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).
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Figure 6.23: Activation of the vastus lateralis muscle in subjects with hLBP, presented as muscle activation normalized to resting muscle readings \( \frac{V_{\text{dynamic}}}{V_{\text{static}}} \).

Figure 6.24: Activation of the gluteus medius muscle in subjects with hLBP, presented as muscle activation normalized to resting muscle readings \( \frac{V_{\text{dynamic}}}{V_{\text{static}}} \).
Figure 6.25: Activation of the external oblique muscle in subjects with hLBP, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).

Figure 6.26: Activation of the erector spinae muscle in subjects with hLBP, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).
6.3 Joint Kinematics within the Full Cohorts

In the previous section, several case studies gave insight into the range of movement strategies that can be used to maintain balance following a perturbation. In order to gain a fuller appreciation for the distinct strategies used by subjects with hLBP, full cohorts of healthy and hLBP subjects were tested and analyzed. In order to understand spinal stability and its connection to hLBP, it is of importance to measure how well system equilibrium is maintained following a postural disturbance by examining the range of motion at each joint.

Similarly to the case studies, the average joint kinematics are plotted for each joint in Figures 6.27 - 6.32. The averaged curve for each cohort is remarkably similar in shape and timing. However, at several joints, including the knee, hip, and lumbar spine the two cohorts exhibit noticeable, though not statistically significant, differences. At the knee, the hLBP cohort has a greater degree of flexion, particularly in the resting posture (Figure 6.28). This may reflect the fact that they assume a more protective stance than their LBP-free counterparts. At the hip, the healthy cohort exhibits an overall greater range of motion from the maximum to minimum curves, demonstrating the wide diversity in range of hip strategies that are utilized. Overall, the healthy cohort utilizes a more flexed hip posture as compared to the hLBP cohort. However, both cohorts exhibit greatest range of motion in the hips, particularly during a forward perturbation (as demonstrated in Figure 6.29). Finally, the hLBP cohort stands with a more extended lumbar posture as compared to their healthy counterparts, perhaps suggesting less abdominal muscle engagement in anticipation of platform movement.

6.3.1 Equilibrium Maintenance

To understand the level of robustness present in the spinal system, it is of interest to examine how well equilibrium is maintained during a balance disturbance. In order to calculate this, the angle of movement produced during the perturbation is measured...
Figure 6.27: Flexion at the ankle joint for healthy and hLBP subjects, with the maximum and minimum kinematics curves describing the variation between subjects within each cohort. The average standard deviation is $1.14^\circ$ (healthy) and $0.88^\circ$ (hLBP).

Figure 6.28: Flexion at the knee joint for healthy and hLBP subjects, with the maximum and minimum kinematics curves describing the variation between subjects within each cohort. The average standard deviation is $2.14^\circ$ (healthy) and $1.50^\circ$ (hLBP).
Figure 6.29: Flexion at the hip joint for healthy and hLBP subjects, with the maximum and minimum kinematics curves describing the variation between subjects within each cohort. The average standard deviation is $1.83^\circ$ (healthy) and $1.95^\circ$ (hLBP).

Figure 6.30: Extension at the lumbar spine joint for healthy and hLBP subjects, with the maximum and minimum kinematics curves describing the variation between subjects within each cohort. The average standard deviation is $1.62^\circ$ (healthy) and $1.63^\circ$ (hLBP).
Figure 6.31: Extension at the lower thoracic spine joint for healthy and hLBP subjects, with the maximum and minimum kinematics curves describing the variation between subjects within each cohort. The average standard deviation is $1.95^\circ$ (healthy) and $1.40^\circ$ (hLBP).

Figure 6.32: Flexion at the upper thoracic spine joint for healthy and hLBP subjects, with the maximum and minimum kinematics curves describing the variation between subjects within each cohort. The average standard deviation is $0.96^\circ$ (healthy) and $1.19^\circ$ (hLBP).
and averaged over the entire testing session (31 perturbations) for all subjects in each cohort (Figures 6.33 & 6.34). No significant differences were found between the two cohorts, however the knee, hip, and lumbar spine were the joints with the greatest differences in range of motion between the two groups. The healthy cohort was found to have a greater range of movement at the knee joint, particularly when the platform was moving backwards. At the hip joint, the hLBP cohort had a greater average RoM. Once again, this was most prominent in response to the backwards perturbation. Although there are noticeable differences between the RoM at these joints, the differences are not statistically significant. Future work may benefit from further in-depth analysis, taking into account resting posture as well as separation of flexion and extension motion.

It was also important to understand how consistent the perturbation response was over time and if any trends existed, particularly during the high and low frequency perturbation sequences. Averages from the two cohorts indicate consistent movement patterns throughout the trial, not significantly affected by the frequency of platform movement (Figures 6.35 & 6.36). Additionally, these results indicate that the learning affect was minimal throughout the trial due to the consistency in average platform response over time seen in both cohorts.
Figure 6.34: Average range of motion for healthy controls (blue) and subjects with hLBP (red) at joints between the major segments of the body following a backwards perturbation. The joint angles demonstrate flexion/extension motion.

Figure 6.35: Average range of motion (in degrees) for healthy controls (black line, one standard deviation in grey) and subjects with hLBP (red line, one standard deviation in pink) at joints between the major segments of the body following a forwards perturbation. These plots show the changes in average response to perturbation over an entire trial.
Figure 6.36: Average range of motion (in degrees) for healthy controls (black line, one standard deviation in grey) and subjects with hLBP (red line, one standard deviation in pink) at joints between the major segments of the body following a backwards perturbation. These plots show the changes in average response to perturbation over an entire trial.

6.3.2 Ternplots

Although the first step in analysis of the kinematics is to look at each joint in isolation, as described in the previous section, it is of interest to understand the function of each joint in relation to the others. In order to examine this, several joints were selected for comparison in ternplots. These plots allow for comparison of three joints in relation to one another. Of particular interest was the comparison between the ankle, knee, and hip joints (Figure 6.37). Here, the ratio of joint movement for healthy controls and subjects with hLBP were plotted and elliptical fitting was applied to each data set using eigenvector-based calculations. In Figure 6.37, the hLBP group is contained to a more centralized region within the ternplot, whereas the healthy control group is scattered over a much wider region. This demonstrates a more equal distribution of movement between the ankle, knee, and hip for hLBP subjects, whereas healthy subjects appear to use a more diverse range of balance maintenance techniques. However, when creating the same plot for comparison of the spinal segments, the difference in ratios between the three segments (lumbar, lower thoracic, and upper thoracic) appears to be greater for the hLBP cohort, suggesting a wider variety of
spinal movement strategies (Figure 6.38).

In addition to analyzing the relative movement between the segment of the spine (lumbar, lower thoracic, and upper thoracic) and the lower limb (ankle, knee, and hip), a comparison was also performed between joints of the lower limb and spine. A ternplot was created for the knee, hip, and lumbar joints in order to identify the relative ranges of motion between three joints arguably situated within the epicenter of LBP (Figure 6.39). This ternplot demonstrates that some healthy subjects use knee-hip-lumbar balance strategies similar to hLBP subjects. However, on the whole, healthy subjects tend to use a greater knee-based strategy than the hLBP subjects, as evidenced by the cluster of blue dots further along the ‘knee’ axis. This is in accordance with earlier results demonstrating greater knee range of motion in the healthy cohort.
Figure 6.38: Ternplot comparison between the lumbar, lower thoracic, and upper thoracic ranges of flexion/extension motion during a forward perturbation.

Figure 6.39: Ternplot comparison between the knee, hip, and lumbar ranges of flexion/extension motion during a forward perturbation.
6.4 Electromyography within the Full Cohorts

In addition to understanding the extent of joint movement in response to a perturbation, it is also of interest to examine the driving force behind such movement, occurring at each muscle. The muscle activation is plotted in Figures 6.40 - 6.45 and demonstrates the average activation in both the healthy and hLBP cohorts. The hLBP cohort has a distinctly higher activation of the soleus and semitendinosus muscles (Figures 6.40 & 6.41), while the healthy cohort demonstrates higher activation of the gluteus medius, external obliques, and erector spinae (Figures 6.43 - 6.45). Activation of the vastus lateralis is remarkable similar between the two cohorts (Figure 6.42), though slightly greater for the hLBP group. These patterns suggest a lower limb-focused strategy for hLBP subjects and reduced use of pelvic and trunk muscles in comparison to the healthy cohort.

6.4.1 Peak Muscle Activation

In addition to seeing the average muscle activation curve, it is also interesting to know the average peak activation and see this comparison in a tabular format. The average peak activation for each muscle was calculated for each subject and subsequently averaged for each cohort. Backward and forward perturbations were examined separately (Figures 6.46 & 6.47). During forward perturbations, soleus was found to be significantly different between cohorts (p=0.04), while both soleus (p=0.04) and semitendinosus (p=0.01) were found to be significantly different during backwards perturbations.

These figures are in line with observations made from observing the muscle activations curves. Based on the significant use of the soleus and semitendinosus muscles by the hLBP cohort, it can be hypothesized that these subjects are employing a lower limb strategy in order to maintain balance on the moving platform. This reliance on the lower limb musculature, coupled with the increased joint range of movement in the
Figure 6.40: Activation of the soleus muscle in healthy and hLBP subjects, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$). The maximum and minimum muscle activation curves are provided to give a sense of the variation between subjects within a cohort. The average standard deviation is 1.24 for healthy and 5.26 for hLBP.

Figure 6.41: Activation of the semitendinosus muscle in healthy and hLBP subjects, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$). The maximum and minimum muscle activation curves are provided to give a sense of the variation between subjects within a cohort. The average standard deviation is 1.54 for healthy and 1.87 for hLBP.
Figure 6.42: Activation of the vastus lateralis muscle in healthy and hLBP subjects, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$). The maximum and minimum muscle activation curves are provided to give a sense of the variation between subjects within a cohort. The average standard deviation is 2.99 for both the healthy and hLBP cohorts.

Figure 6.43: Activation of the gluteus medius muscle in healthy and hLBP subjects, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$). The maximum and minimum muscle activation curves are provided to give a sense of the variation between subjects within a cohort. The average standard deviation is 3.03 for healthy and 1.02 for hLBP.
CHAPTER 6. RESULTS

Figure 6.44: Activation of the external oblique muscle in healthy and hLBP subjects, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$). The maximum and minimum muscle activation curves are provided to give a sense of the variation between subjects within a cohort. The average standard deviation is 3.50 for healthy and 0.68 for hLBP.

Figure 6.45: Activation of the erector spinae muscle in healthy and hLBP subjects, presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$). The maximum and minimum muscle activation curves are provided to give a sense of the variation between subjects within a cohort. The average standard deviation is 3.13 for healthy and 0.61 for hLBP.
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Figure 6.46: Average peak muscle activation during forward platform movement for each cohort. Measurements were taken at the soleus, semitendinosus, vastus lateralis, gluteus medius, external obliques, and erector spinae. Results are presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).

Lumbar spine compared to healthy subjects and reduced use of the trunk musculature, makes a case for the strategy outlined in Figure 6.48. Here, the lumbar spine is not well controlled by the erector spinae and external oblique muscles and the lower limb is held rigid by the soleus and semitendinosus.

6.4.2 Muscle Synergies

In addition to examining the peak muscle activation in response to a balance disturbance, the way in which muscles worked together was also investigated. Based on the proposed strategy in Figure 6.48 it was expected that different muscles may work in concert for healthy and hLBP subjects, such as the soleus and semitendinosus versus the trunk musculature. This was accomplished through calculation of muscle synergies using a nonnegative optimization procedure [57]. This calculation is aimed at identifying the set of muscle synergies that can best predict observed experimental responses. The algorithm extracts both the muscles that contribute to a synergy as well as weighting coefficients for each synergy that can be used to reconstruct the ex-
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Figure 6.47: Average peak muscle activation during backwards platform movement for each cohort. Measurements were taken at the soleus, semitendinosus, vastus lateralis, gluteus medius, external obliques, and erector spinae. Results are presented as muscle activation normalized to resting muscle readings ($V_{\text{dynamic}}/V_{\text{static}}$).

Figure 6.48: Proposed strategy of hLBP cohort based on muscle activation patterns and joint kinematics of the lumbar spine.
peripheral muscle measurements. The optimal number of synergies for the six muscles of interest was found to be three. (This was determined through curve fitting between the synergy-produced responses and the experimental responses.)

Although examining muscle synergies is not the main focus of this project, it is a natural next step in understanding the neural encoding that likely influences the strategies utilized during a balance disturbance. To begin investigating the possibility of muscle synergy being tied to LBP, we return to the original four case studies discussed at the beginning of this chapter: C1, C2, P1, and P2. The three muscle synergies calculated for each of these subjects may be found in Figures 6.49 - 6.52. Muscles are listed along the x-axis, where Sol = soleus, Semi = semitendinosus, VL = vastus lateralis, GM = gluteus medius, EO = external oblique, and ES = erector spinae. The y-axis gives the fraction of activation each muscle contributes to each muscle synergy (0-1).

As proposed in the previous section, the external oblique and erector spinae appear to function synergistically for healthy subjects. This can be visualized in Figures 6.49(c) and 6.50(c). This is suggestive of a higher degree of trunk control because these subjects are capable of synergistically activating these two muscles together. However, Subjects P1 and P2 did not demonstrate the predicted synergy of the semitendinosus and the soleus. In Figure 6.51(a), some degree of synergy can be seen, yet the soleus is most strongly activated as part of a different synergy in (c). P2 (Figure 6.52) shows very little synergistic activation of these two muscles. Although the results presented here are the first to analyze muscle synergies in LBP subjects, the optimization procedure would benefit from measuring additional muscles in the limb and spinal region in order to achieve more reliable synergy groups and fitting between the algorithm outputs and experimental recordings.
Figure 6.49: The three muscle synergies calculated for C1 (healthy).

Figure 6.50: The three muscle synergies calculated for C2 (healthy).

Figure 6.51: The three muscle synergies calculated for P1 (hLBP)

Figure 6.52: The three muscle synergies calculated for P2 (hLBP)
6.5 Overall Results

The findings presented in the above results and discussion begin to form a picture about the differences between people with hLBP and healthy individuals. Importantly, subjects tested in this study were not currently in pain, but did report a history of LBP. This is unique from other studies where the subjects were currently in pain. Therefore, we are seeing true differences in movement patterns between the two cohorts as opposed to movement affected by daily pain levels. The results demonstrated several distinct differences between the healthy and hLBP subjects, including a difference in balance strategy.

Overall, the healthy cohort utilized an upper body-focused strategy for balance control. Specifically, this involved activation of the erector spinae and external oblique muscles, thus resulting in a significantly smaller range of motion at the lumbar spine compared to the hLBP group. The hLBP cohort, as a whole, implemented a lower limb-focused strategy, relying on activation of the semitendinosus and soleus muscles. The upper limb strategy implemented by the healthy subjects could also be identified in the muscle synergies in which Subjects C1 and C2 utilized the erector spinae and external obliques synergistically. Ternplots were also implemented to begin understanding the relative movement between the joints or the ratio of adjacent joint movement. These results identified a wider spread of lower limb strategies for healthy subjects as opposed to the more tightly centered ratio of hip-knee-ankle joint movement seen in hLBP subjects. This may be a result of the lower limb-focused strategy used by this cohort, leading to a more even distribution of joint movement between the three joints of the lower limb. Conversely, ternplots of the spinal segments show a greater spread of movement ratios for the hLBP group as compared to the healthy cohort. This suggests greater variability in the relative movement within the different section of the spinal column for the hLBP group.

In addition to identifying strategies that each cohort adopted to maintain balance, it was also interesting to understand if any differences could be seen during high or
low frequency perturbations or if learning was present throughout the perturbation exercises. Minimal evidence of learning was identified and changes in balance strategy were not identified between the high and low frequency perturbations. This was an unexpected result because one would assume learning would occur over the 30 perturbations. It is possible that learning did in fact occur, but it was too small to be detected in this analysis.

These results begin to form a more cohesive picture surrounding control strategies in LBP. However, further work is required to reach a full understanding of these movement programs in both healthy and hLBP subjects. In particular, it would be interesting to have a side-by-side comparison of healthy, hLBP, and acute LBP subjects using the above analysis methods in order to better understand the progression of movement changes. The limitations of this work and future directions for this research will be explored further in the Discussion section.
Chapter 7

Discussion

It is challenging to compare the movement of individuals in response to a balance disturbance. Each response is unique and has characteristics that allow for postural maintenance, with varied degrees of optimization. For example, one subject may move into flexion in response to a forward perturbation, whereas another individual may move into extension. Additionally, it is possible to see little range of motion for two individuals, but for different underlying reasons. One participant may have a highly optimized muscular response, thus requiring little engagement of the muscles, leading to high stability and little joint movement. A second participant may have a poorly optimized muscular response requiring very high muscle activation, also leading to little joint movement. The spinal stability systems of these two subjects are very different, often indicative of different levels of spinal health. The goal of this thesis was to begin teasing these aspects apart in preparation for further study of spinal stability as it relates to hLBP. The spine is a complex structure that is part of a complex system and thus the body has many strategies to maintain functional dynamic stability. Due to these aspects of spinal control, it is challenging to identify the precise problems in LBP as many strategies can be used to compensate for system deficiencies. It is hypothesized that through exploration of these compensation strategies treatment can be targeted to need rather than the current global approach that assumes one
treatment method works for all patients.

This study made several steps toward achieving this goal. A system was developed for testing postural stability and the body’s response to perturbation, through the incorporation of a perturbation platform, Vicon System, EMG monitors, WBB instrumentation, marker set-up, and testing protocol. In addition, an analysis approach was developed using Matlab, BodyBuilder, and OpenSim. The primary mode of analysis was a novel spinal model in BodyBuilder. Finally, based on the challenges encountered during testing and analysis in this study, a new perturbation platform was developed in conjunction with an undergraduate student, clinical fellow, and post-doctoral fellow.

As introduced above, this study developed a novel marker/cluster set-up that offers a compromise between complex/invasive set-ups and simple single trunk segment models. Current models are either too complex, introducing a great deal of marker error, or are too simplified, treating the spine as a single segment. The single published OpenSim model to attempt modeling the spine [11] requires experimental tracking of individual vertebrae, which would be challenging, if not impossible, to achieve without the use of bone pins. Other models have treated the spine as a single trunk segment, such as Henry et al [25]. This study modeled the spine as a single segment articulating about the lumbar-pelvic joint. The model and marker set-up developed in this study are simple enough to be implemented in human subject testing and complex enough to offer a true sense of spinal movement. A similar model [50], also considers the spine as three segments (lumbar, lower thoracic, and upper thoracic) and is utilized as a point of comparison for the results obtained during the present study.

Secondly, the combination of kinematics and EMG analysis for the two cohorts found that the healthy cohort used an upper body strategy, utilizing the erector spinae and external obliques more strongly than the hLBP group. Trunk muscle contraction contributed to the smaller range of motion at the lumbar spine seen in the healthy cohort compared to the hLBP group. Alternatively, the hLBP cohort implemented a lower limb-focused strategy, as evidenced by the increased activation of the soleus and semitendinosus muscles compared to the healthy cohort in response to a balance
disturbance.

It is challenging to directly compare these results to other studies because the methodology, marker placement, and subject characteristics vary widely. However, when considering the Preuss et al [50] study previously mentioned, it is possible to make a more direct comparison because both studies utilize a three-segment spinal model and a perturbation platform as the means of delivering the perturbation. Unlike the present study, Preuss et al delivered the perturbations in a diagonal direction, thus sending their subjects anterior-right or posterior-left, instead of directly ahead. Examining results presented for intersegmental joint angles with the spine, the measure of flexion/extension in the sagittal plane correlates well with the findings of this study. Both studies show 1-2 degrees of flexion/extension when analyzing averaged sagittal plane traces. However, it is difficult to ascertain the exact angles measured based on the plots provided in the 2008 paper. Additionally, the results collected by Preuss et al [50] were obtained from a healthy population and did not include any subjects with a history of LBP.

Similarly to the present study, Jacobs et al [30] explored the effect of hLBP on EMG activation in response to a postural perturbation. However, one of the main findings of this paper was increased resting muscle activation, which was not identified in the present study (Figure 3.2). This difference may be due to the cohorts selected as hLBP subjects. The present study identified subjects with a history of LBP, but absolutely no current pain. Jacobs et al identified subjects that were not having an acute flare-up, but may be in mild pain. This is an important difference, as has been shown by Hodges et al [27], as pain has a prominent affect on human movement strategies. As the present study aimed to identify the underlying features that may contribute to causing recurrent LBP, it is important that the subjects are not experiencing pain that may change movement strategies. Examination of muscle activation during the perturbation response was reported by Jacobs et al to be reduced in both the ankle and trunk muscles in the hLBP cohort relative to the healthy subjects. The reduction in trunk musculature correlates well with findings in the present study where the
healthy cohort demonstrated stronger activation of the trunk muscles relative to the hLBP group. However, the present study found an increase in soleus muscle activation in the hLBP group compared to the healthy cohort. This discrepancy between the two studies may arise from a number of different factors, including subject selection, platform protocol, or analysis methods.

Thirdly, this study is the first, to our knowledge, to compare muscle synergies between a healthy cohort and subjects with hLBP. Although this must be further explored in future experiments, the results offer an interesting starting point for this type of work. Looking at two healthy subjects and two hLBP subjects, it was found that, as predicted by kinematics and EMG results, the external oblique and erector spinae appear to operate synergistically in healthy subjects. This is not true for subjects with hLBP. It is possible that this synergy is one of many that is neurally hardwired in a balance maintenance program. The field of LBP biomechanics would benefit from future research in muscle synergies in order to better understand such centrally programmed responses.

Future applications and directions for this research are extensive and touch the realms of engineering, biomechanics, neurology, and physical therapy. Of particular interest is the use of the wobble board as a training tool. In physical therapy, it is common practice to use wobble boards to retrain sprained ankles in order to regain proprioception. Proprioception is also challenged clinically using eyes open, eyes closed exercises, changing base of support (double leg, single leg activities), changing the support surface (using gym balls and wobble boards) or through using equipment or strapping to increase proprioceptive input into the system. However, this technique has not been widely employed to regain proper proprioception within the spine and possibly avoid future injury. In the past decade, the concept of training LBP patients to obtain a neutral or midrange spine has been investigated. Clinically, it is often noted that LBP patients have difficulty moving into a neutral lumbar posture, suggestive of proprioceptive deficits. O’Sullivan et al [47] have shown this to be true in a study utilizing electromagnetic sensors. However, the study was conducted while
the participant was seated, as have several other studies in the area of neutral spine retraining, as summarized by O’Sullivan et al [46]. Retraining proprioception from a seated posture neglects the importance of retraining both the lower limbs and the spine in concert to properly adjust to everyday balance perturbations. It could be useful to use a perturbation platform or wobble board to retrain a patient’s balance and reaction control in a contained environment, therefore avoiding improper control and, possibly, chronic LBP.

A second future direction of the work presented in this study is a most robust model of the spine that would allow for exploration of trunk muscle activation as well as joint kinematics. The model presented in this project would benefit from additional attention to scaling of the spinal segments and mathematical constraints between the adjacent spinal segments. This would aid in avoiding anatomically inaccurate disconnection between adjacent vertebrae. Additionally, the current models presented in this paper are only appropriate for kinematics calculations and do not incorporate muscles. In OpenSim, there currently is a model of the lumbar spine [11], including both bone and muscle. However, this is inadequate for investigating the entire spine complex, including the muscles such as the erector spinae, which travel the entire length of the spinal column. The availability of such a model would allow researchers to determine features, such as muscle activation and joint torques, that contribute to or prevent LBP in a more efficient method than can be obtained in a laboratory environment.

Although this study made progress towards better understanding of spinal stability, it was limited by a number of factors. Most importantly, the study was limited by moderately-sized numbers, including a control group of 16 subjects and a hLBP cohort of 10 subjects. In a better study, this number would be much larger in order to identify the full spectrum of strategies used by subjects of varying backgrounds, ages, and physical competencies. This is particularly important for LBP, which can be challenging to understand due to its many manifestations in different individuals. However, this study was able to recruit subjects of a variety of ages and backgrounds,
which provides validity in the comparison between the two cohorts. (Full subject information may be viewed in the Appendix.) Secondly, the study faced limitations in equipment functionality. This is primarily centered about the functionality of the perturbation platform that, as discussed within the Equipment Methodology, had very limited velocity and acceleration controls, limited to a single lever and a knob for throttle control. Greater precision and consistency with these factors would improve the quality of the trial. However, the current equipment was adequate for the pilot experiments performed in this study. Additionally, the deficiencies in the current equipment have provided insight into the required specifications for a new platform, also being developed at Imperial College within the McGregor and Phillips lab groups [15].

Within all studies examining kinematics and body movement, there is also inherent error in measurement of bony landmarks through reflective marker proxies and subsequently joint angles. Error is derived both from skin movement, where markers move away from the desired bony landmark, and from tracking error of the Vicon system itself. Skin error was minimized through the use of a cluster tracking method, as opposed to tracking individual markers throughout the trial. While tracking error is particularly difficult to control for when measuring the spine due to the large number of markers used and precision that must be repeated during each trial in placement of the markers, great care was taken while preparing subjects for testing and during the test itself. In addition to marker and system error, one must remember that the kinematics model itself is an estimation of human movement based on markers. While a kinematics model can offer a high quality estimate of joint movement, it is inherently imperfect in the assumptions it makes. For example, the hip joint center is estimated from scaling factors based off of cadaver measurements made by Horsman et al [29]. These measurements were performed on a male cadaver, and yet this study, and many others, impose the same scaling factors on female pelvises, which are generally of different size and shape compared to the male counterpart.

The concepts investigated in this study provide many avenues for future research
and begin to address gaps in the LBP research space. This study has contributed a new way of investigating spinal motion by splitting the spinal column into three portions, instead of treating it as a single element or many individual vertebrae. By doing so, this work strikes a balance between a far too simplified model and one that is too complex. This study also investigates both the upper and lower limb together, allowing for understanding of how the lower limb may contribute to spinal control, stability, and LBP. Finally, this work seeks to understand how muscle synergies allow for spinal control, which is integral to maintenance of spinal health. Though there is still a great deal of work to be done in these areas, this study has introduced new ideas and analysis methods into the area of LBP research.
Chapter 8

Conclusion

The work presented in this study focuses on the measurement and analysis of movement in healthy controls and those with a history of LBP. This project specifically targeted analysis of human movement following a balance disturbance, often regarded as an informative movement because it requires quick and automated muscle activation, resulting in less subject-altered motion. Using a novel seven-segment model to process experimental marker data, it was found that subjects with hLBP exhibited a distinctly different strategy than healthy subjects. Healthy subjects primarily exhibited a trunk-focused balance strategy, utilizing the erector spinae and external obliques more strongly than the hLBP group. Trunk muscle contraction contributed to the smaller range of motion at the lumbar spine seen in the healthy cohort compared to the hLBP group. Alternatively, the hLBP cohort implemented a lower limb-focused strategy, activating the soleus and semitendinosus muscles more strongly than the healthy cohort. These findings suggest that particular reactive movement patterns may be indicative of muscular deficits in subjects with hLBP, either present as a result of the LBP or themselves causes of the LBP. In either case, identification of these deficits may be helpful in the development of specific rehabilitation programs that can provide more effective treatment plans than are currently available. Additionally, it may be interesting to further investigate using the perturbation platform as a tool
for retraining people suffering from chronic LBP, specifically regaining adequate proprioception. By regaining proprioception, it might be possible to activate muscles in the proper sequence and at the appropriate level, thus reducing movements that place strain on the spine and produce pain.
References


REFERENCES


REFERENCES


REFERENCES


Appendix
Back Pain Study - Medical History Form

Date: ________________________  Subject ID: ________________________

Height (m): ____________________  Weight (kg): ________________________

Date of Birth (DD-MM-YY): ________________________

Handedness:  Left  Right  Ambidextrous

Dominant Foot:  Bug – Left  Right  Kick – Left  Right  Diamond – Left  Right

BACK PAIN
1. Do you have a history of back pain?  Y  N
2. Is the back pain recurrent?  Y  N
3. If yes, how often does it recur?  ________________________
4. Do you have a history of neck pain?  Y  N
5. Is the neck pain recurrent?  Y  N
6. If yes, how often does it recur?  ________________________

INJURIES
1. Have you had spinal injuries or surgeries?  Y  N
2. If yes, please describe:  ______________________________________
3. Have you had injuries to the lower legs?  Y  N
4. If yes, please describe:  ______________________________________
5. Have you had an abdominal injury?  Y  N
6. If yes, please describe:  ______________________________________
7. Have you ever had any other surgery?  Y  N
8. If yes, please describe:  ______________________________________

ADDITIONAL HISTORY
1. Have you been told you have scoliosis?  Y  N
2. Have you had previous balance issues?  Y  N
3. If yes, please describe:  ______________________________________
4. Do you wear glasses?  Y  N
5. Do you have other visual disturbances?  Y  N
6. If yes, please describe:  ______________________________________

PROFESSION
1. What is your current profession?
   - Full-time, please describe:  ______________________________________
   - Part-time, please describe:  ______________________________________
   - Full-time Homemaker
   - Student
   - Unemployed
2. Would you describe your work as
   - Physical
   - Mixed activity
   - Sedentary
3. Have you ever performed the following tasks consistently as part of your profession?
   - Heavy lifting
   - Sitting all day
   - Standing all day

If you experience chronic back pain, please continue to the reverse side of this form.
Please indicate where you typically experience back pain by filling in the appropriate squares:

Please describe the sensation of back pain you experience and how frequently (i.e. every month, several times per year, etc.) it occurs:

________________________________________________________________________________________

________________________________________________________________________________________

________________________________________________________________________________________

________________________________________________________________________________________

________________________________________________________________________________________

Thank you for your participation!
The Wii Balance Board code consists of one main code and two subroutines. The codes are presented as a web of graphical nodes representing different built-in functions of LabView.
Figure 8.1: Main Routine

Figure 8.2: Subroutine I - subGetWiiMoteState

Figure 8.3: Subroutine II - subRetreiveBalanceBoardValues
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<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>12-Mar-13</td>
<td>12</td>
<td>26</td>
<td>F</td>
<td>188.0</td>
<td>57.0</td>
<td>1.69</td>
<td>student</td>
<td>mixed activity</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>26-Mar-13</td>
<td>13</td>
<td>24</td>
<td>F</td>
<td>166.5</td>
<td>72.6</td>
<td>2.62</td>
<td>student</td>
<td>physical</td>
<td>sitting all day</td>
<td>L</td>
<td>L</td>
<td>Y, back pain</td>
<td>Y, every 6 months, not currently in pain</td>
<td>N</td>
</tr>
<tr>
<td>27-Mar-13</td>
<td>14</td>
<td>17</td>
<td>F</td>
<td>168.5</td>
<td>68.0</td>
<td>2.32</td>
<td>engagement manager</td>
<td>sedentary</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>Y, every menstrual month</td>
<td>Y, fuzzy vision</td>
</tr>
<tr>
<td>6-Apr-13</td>
<td>15</td>
<td>25</td>
<td>M</td>
<td>163.5</td>
<td>71.2</td>
<td>2.11</td>
<td>student</td>
<td>mixed activity</td>
<td>heavy lifting</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>not currently in pain</td>
<td>Hip sprain</td>
</tr>
<tr>
<td>8-Apr-13</td>
<td>16</td>
<td>42</td>
<td>M</td>
<td>182.5</td>
<td>98.8</td>
<td>2.89</td>
<td>carpenter</td>
<td>physical</td>
<td>heavy lifting</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>Y, every 6 months</td>
<td>N</td>
</tr>
<tr>
<td>10-Apr-13</td>
<td>17</td>
<td>31</td>
<td>F</td>
<td>172.5</td>
<td>59.8</td>
<td>2.01</td>
<td>research associate</td>
<td>mixed activity</td>
<td>heavy lifting all day</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>Y, every 6 months, not currently in pain</td>
<td>N</td>
</tr>
<tr>
<td>11-Apr-13</td>
<td>18</td>
<td>22</td>
<td>F</td>
<td>168.5</td>
<td>56.6</td>
<td>2.31</td>
<td>scientist</td>
<td>mixed activity</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>N</td>
<td>N</td>
<td>N</td>
</tr>
<tr>
<td>19-Apr-13</td>
<td>19</td>
<td>39</td>
<td>M</td>
<td>179.0</td>
<td>86.2</td>
<td>2.78</td>
<td>orthopaedic surgeon</td>
<td>mixed activity</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>Y, left knee surgery</td>
<td>N</td>
</tr>
<tr>
<td>30-Apr-13</td>
<td>20</td>
<td>35</td>
<td>M</td>
<td>165.0</td>
<td>79.0</td>
<td>2.64</td>
<td>firefighter</td>
<td>mixed activity</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>Y, every 6 months</td>
<td>N</td>
</tr>
<tr>
<td>2-May-13</td>
<td>21</td>
<td>35</td>
<td>M</td>
<td>183.0</td>
<td>87.0</td>
<td>2.63</td>
<td>lefthanded</td>
<td>mixed activity</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>Y, back pain</td>
<td>back pain, fracture, knee, hairline fracture</td>
<td>N</td>
</tr>
<tr>
<td>7-May-13</td>
<td>22</td>
<td>48</td>
<td>F</td>
<td>165.0</td>
<td>83.3</td>
<td>2.58</td>
<td>research associate</td>
<td>sedentary</td>
<td>sitting all day</td>
<td>R</td>
<td>R</td>
<td>Y, neck pain</td>
<td>Y, every 6 months</td>
<td>Y, red rim glasses</td>
</tr>
</tbody>
</table>
Macro AXISVISUALISATION(Segment)
ORIGIN#Segment=0(Segment)
AXISX#Segment=\{100,0,0\}*Segment
AXISY#Segment=\{0,100,0\}*Segment
AXISZ#Segment=\{0,0,100\}*Segment
Output(ORIGIN#Segment,AXISX#Segment,AXISY#Segment,AXISZ#Segment)
Endmacro

macro REPLACE4(p1,p2,p3,p4)
{*Replaces any point missing from set of four fixed in a segment*}

{*SECTION FOR INITIALISATION OF VIRTUAL POINTS*}
{*REPLACE4*}
s123 = \[p2,p1-p2,p2-p3\]
p4V1 = Average(p4/s123)*s123
s124 = \[p2,p1-p2,p2-p4\]
p3V1 = Average(p3/s124)*s124
s134 = \[p3,p1-p3,p3-p4\]
p2V1 = Average(p2/s134)*s134
s234 = \[p3,p2-p3,p3-p4\]
p1V1 = Average(p1/s234)*s234

{*SECTION FOR SPECIFICATION OF VIRTUAL POINTS*}
p1 = p1 ? p1V1
p2 = p2 ? p2V1
p3 = p3 ? p3V1
p4 = p4 ? p4V1

endmacro

{*End of macro section*}

{*Define Optional Markers*}

{*OptionalPoints(MJ,RAA,LAA)*}
OptionalPoints(RASIS,LASIS,LPSIS,RPSIS)
OptionalPoints(LFLE,LTh1,LFME,LTh2,LTh3)
OptionalPoints(RFLE,RTh1,RFME,RTh3,RTh2)
OptionalPoints(LFAM,LC1,LTAM,LC2,LC3,LTT)
OptionalPoints(RFAM, RC1, RTAM, RC2, RC3, RTT)
OptionalPoints(LFM2, LFCC, LFMT, LTF)
OptionalPoints(RFM2, RFCC, RFMT, RTF)*

OptionalPoints(RT1, LT1, UTC1, UTC2, UTC3, RT6, LT6)
OptionalPoints(RT7, LT7, LTC1, LTC2, LTC3, RT12, LT12)
OptionalPoints(RL1, LL1, LumC1, LumC2, LumC3, RL5, LL5)
OptionalPoints(RASIS, LASIS, RPSIS, LPSIS)
OptionalPoints(LFLE, LTh1, LFME, LTh2, LTh3)
OptionalPoints(RFLE, RTh1, RFME, RTh3, RTh2)
OptionalPoints(LLC, LC1, LMC, LC2, LC3)
OptionalPoints(RLC, RC1, RMC, RC2, RC3)
OptionalPoints(LMet, LCal, LTube, LExtra)
OptionalPoints(RMet, RCal, RTube, RExtra)

{*Create segments*}

{*Global Segment*}

ORIGINGlobal={0,0,0}
Global=[ORIGINGlobal,{1,0,0},{0,0,1},xyz]

AXISXGlobal=ORIGINGlobal+{100,0,0}
AXISYGlobal=ORIGINGlobal+{0,100,0}
AXISZGlobal=ORIGINGlobal+{0,0,100}

Output(ORIGINGlobal, AXISXGlobal, AXISYGlobal, AXISZGlobal)

{*Pelvis Segment*}

REPLACE4(RASIS, LASIS, RPSIS, LPSIS)

PelvisORIGIN=(RASIS+LASIS+RPSIS+LPSIS)/4
SACR=(RPSIS+LPSIS)/2
PELF = (LASIS+RASIS)/2
Pelvis=[PELF,(LASIS-RASIS),(PELF-SACR),xzy]

If $Static == 1 Then

$SACR_PELFDist = DIST(SACR,PELF)
$LASIS_RASISDist = DIST(LASIS,RASIS)
$RASIS_RFLE = DIST(RASIS,RFLE)
$LASIS_LFLE = DIST(LASIS,LFLE)

Endif

PARAM($SACR_PELFDist)
PARAM($LASIS_RASISDist)
PARAM($RASIS_RFLE)
PARAM($LASIS_LFLE)

{*Thigh Segments*}

{*parameters for HJC*}

AP_Scale = 149.95
ML_Scale = 262.4
SI_Scale = 487.61

Dist2=$SACR_PELFDist/AP_Scale*(-38.76)
Dist1=(($LASIS_RASISDist)/ML_Scale*89.70)
Dist3=(($LASIS_LFLE)/SI_Scale*(-87.29))

$RVect = {-Dist1,-Dist2,Dist3}
$LVect = {Dist1,-Dist2,Dist3}
PARAM($RVect,$LVect)

RHJC_G=$RVect*Pelvis
LHJC_G=$LVect*Pelvis

output(RHJC_G,LHJC_G)

If  $Static == 1 Then

%LHJC=LHJC_G/Pelvis
%RHJC=RHJC_G/Pelvis

EndIf

PARAM(%LHJC,%RHJC)

LHJC=%LHJC*Pelvis
RHJC=%RHJC*Pelvis

REPLACE4(LFLE,LTh1,LTh2,LTh3)
REPLACE4(RFLE,RTh1,RTh2,RTh3)
REPLACE4(LFME,LTh1,LTh2,LTh3)
REPLACE4(RFME,RTh1,RTh2,RTh3)

LThighCluster = [LTh2,(LTh2-LTh1),(LTh3-LTh2),zxy]
RThighCluster = [RTh2,(RTh2-RTh1),(RTh3-RTh2),zxy]

LKJC=(LFLE+LFME)/2
RKJC=(RFLE+RFME)/2
LThigh = \{LHJC, (LHJC - LKJC), (LFME - LFLE), zyx\}  
RThigh = \{RHJC, (RHJC - RKJC), (RFLE - RFME), zyx\}  

If $Static == 1$ Then  
$LThighClusterAngle = \langle LThighCluster, LThigh, xyz \rangle$  
OUTPUT($LThighClusterAngle$)  
Endif  
PARAM($LThighClusterAngle$)  

$LThighCluster = ROT(LThighCluster, LThighCluster(1), -$LThighClusterAngle(1))$  
$LThighCluster = ROT(LThighCluster, LThighCluster(2), -$LThighClusterAngle(2))$  
$LThighCluster = ROT(LThighCluster, LThighCluster(3), -$LThighClusterAngle(3))$  

If $Static == 1$ Then  
$RThighClusterAngle = \langle RThighCluster, RThigh, xyz \rangle$  
OUTPUT($RThighClusterAngle$)  
Endif  
PARAM($RThighClusterAngle$)  

$RThighCluster = ROT(RThighCluster, RThighCluster(1), -$RThighClusterAngle(1))$  
$RThighCluster = ROT(RThighCluster, RThighCluster(2), -$RThighClusterAngle(2))$  
$RThighCluster = ROT(RThighCluster, RThighCluster(3), -$RThighClusterAngle(3))$  

{*Shank Segments*}  
REPLACE4(LLC, LC1, LC2, LC3)  
REPLACE4(RLC, RC1, RC2, RC3)  
REPLACE4(LMC, LC1, LC2, LC3)  
REPLACE4(RMC, RC1, RC2, RC3)  

$LShankCluster = [LC2, (LC2-LC1), (LC2-LC3), xyz]$  
$LShankCluster = [LC2, (LC2-LC1), (LC2-LC3), xyz]$  

LAJC = (LLC + LMC)/2  
RAJC = (RLC + RMC)/2  

$LShank = [LAJC, (LKJC - LAJC), (LMC - LLC), zyx]$  
$RShank = [RAJC, (RKJC - RAJC), (RLC - RMC), zyx]$  

If $Static == 1$ Then
$LShankClusterAngle=<LShankCluster,LShank,xyz>
OUTPUT($LShankClusterAngle)

Endif

PARAM($LShankClusterAngle)

LShankCluster=ROT(LShankCluster,LShankCluster(1),-$LShankClusterAngle(1))
LShankCluster=ROT(LShankCluster,LShankCluster(2),-$LShankClusterAngle(2))
LShankCluster=ROT(LShankCluster,LShankCluster(3),-$LShankClusterAngle(3))

If $Static == 1 Then

$RShankClusterAngle=<RShankCluster,RShank,xyz>
OUTPUT($RShankClusterAngle)

Endif

PARAM($RShankClusterAngle)

RShankCluster=ROT(RShankCluster,RShankCluster(1),-$RShankClusterAngle(1))
RShankCluster=ROT(RShankCluster,RShankCluster(2),-$RShankClusterAngle(2))
RShankCluster=ROT(RShankCluster,RShankCluster(3),-$RShankClusterAngle(3))

{*Foot Segments*}

REPLACE4(LMet,LCal,LLC,LMC)
REPLACE4(RMet,RCal,RLC,RMC)

LFoot = [LAJC,LCal-LMet,LCal-LExtra,yxz]
RFoot = [RAJC,RCal-RMet,RCal-RExtra,yxz]

{*Lumbar Segment*}

REPLACE4(RL1,LumC1,LumC2,LumC3)
REPLACE4(LL1,LumC1,LumC2,LumC3)
REPLACE4(RL5,LumC1,LumC2,LumC3)
REPLACE4(LL5,LumC1,LumC2,LumC3)

LumCluster = [LumC2,(LumC2-LumC1),(LumC2-LumC3),zyx]
LumJC=(RL5+LL5)/2
UpperLum = (RL1+LL1)/2
Lumbar=[LumJC,(UpperLum-LumJC),(RL5-LL5),zyx]

If $Static == 1 Then
$\text{LumClusterAngle}=\langle \text{LumCluster, Lumbar, xyz}\rangle
\text{OUTPUT}(\text{LumClusterAngle})
$

\text{Endif}$

\text{PARAM}(\text{LumClusterAngle})$

\text{LumCluster} = \text{ROT}(\text{LumCluster}, \text{LumCluster}(1), -\text{LumClusterAngle}(1))$
\text{LumCluster} = \text{ROT}(\text{LumCluster}, \text{LumCluster}(2), -\text{LumClusterAngle}(2))$
\text{LumCluster} = \text{ROT}(\text{LumCluster}, \text{LumCluster}(3), -\text{LumClusterAngle}(3))$

\{\* Lower Thoracic Segment *\} 

\text{REPLACE4}(\text{RT7}, \text{LTC1}, \text{LTC2}, \text{LTC3})$
\text{REPLACE4}(\text{LT7}, \text{LTC1}, \text{LTC2}, \text{LTC3})$
\text{REPLACE4}(\text{RT12}, \text{LTC1}, \text{LTC2}, \text{LTC3})$
\text{REPLACE4}(\text{LT12}, \text{LTC1}, \text{LTC2}, \text{LTC3})$

\text{LThorCluster} = [\text{LTC2}, (\text{LTC2-LTC1}), (\text{LTC2-LTC3}), \text{xyz}]$
\text{LTJC} = (\text{RT12}+\text{LT12})/2$
\text{UpperLThor} = (\text{RT7}+\text{LT7})/2$
\text{LThor} = [\text{LTJC}, (\text{UpperLThor-LTJC}), (\text{RT12}-\text{LT12}), \text{zyx}]$

\text{If } \text{Static} == 1 \text{ Then}$
\$\text{LThorClusterAngle}=\langle \text{LThorCluster, LThor, xyz}\rangle
\text{OUTPUT}(\text{LThorClusterAngle})$
\text{Endif}$

\text{PARAM}(\text{LThorClusterAngle})$

\text{LThorCluster} = \text{ROT}(\text{LThorCluster}, \text{LThorCluster}(1), -\text{LThorClusterAngle}(1))$
\text{LThorCluster} = \text{ROT}(\text{LThorCluster}, \text{LThorCluster}(2), -\text{LThorClusterAngle}(2))$
\text{LThorCluster} = \text{ROT}(\text{LThorCluster}, \text{LThorCluster}(3), -\text{LThorClusterAngle}(3))$

\{\* Upper Thoracic Segment *\} 

\text{REPLACE4}(\text{RT1}, \text{UTC1}, \text{UTC2}, \text{UTC3})$
\text{REPLACE4}(\text{LT1}, \text{UTC1}, \text{UTC2}, \text{UTC3})$
\text{REPLACE4}(\text{RT6}, \text{UTC1}, \text{UTC2}, \text{UTC3})$
\text{REPLACE4}(\text{LT6}, \text{UTC1}, \text{UTC2}, \text{UTC3})$

\text{UThorCluster} = [\text{UTC2}, (\text{UTC2-UTC1}), (\text{UTC2-UTC3}), \text{xyz}]$
\text{UTJC} = (\text{RT6}+\text{LT6})/2
UpperUThor=(RT1+LT1)/2

UThor=[UTJC,(UpperUThor-UTJC),(RT6-LT6),zyx]

If $Static == 1 Then

$UThorClusterAngle=<UThorCluster,UThor,xyz>
OUTPUT($UThorClusterAngle)

Endif

PARAM($UThorClusterAngle)

UThorCluster=ROT(UThorCluster,UThorCluster(1),-$UThorClusterAngle(1))
UThorCluster=ROT(UThorCluster,UThorCluster(2),-$UThorClusterAngle(2))
UThorCluster=ROT(UThorCluster,UThorCluster(3),-$UThorClusterAngle(3))

{*Output Virtual Markers*}

Output(ORIGINGlobal,PelvisORIGIN,RHJC,LHJC,RKJC,LKJC,RAJC,LAJC,LumJC,LTJC,UTJC)

{* Find the general progression direction of the subject *}
{*==============================================================*}

PelvisDirection = AVERAGE( SACR-PELF )
If $Static == 1 Then
  Anatomy = [0(Global),PelvisDirection,3(Global),zyx]
Else
  Anatomy = [0(Global),-Progress,3(Global),xyz]
EndIf

Fwd = 3(<Anatomy,Global>)

{*align to +X*} Anatomy = Global
  If (Fwd > -135) AND (Fwd <= -45) Then {*align to -Y*}
    Anatomy = ROT(Anatomy, 3(Anatomy),-90)
  ElseIf (Fwd > 45) AND (Fwd <= 135) Then {*align to +Y*}
    Anatomy = ROT(Anatomy, 3(Anatomy),90)
  ElseIf (Fwd > 135) OR (Fwd <= -135) Then {*align to -X*}
    Anatomy = ROT(Anatomy, 3(Anatomy),180)
  EndIf

{*Visualise AXIS*}

AXISVISUALISATION(Pelvis)
AXISVISUALISATION(LThigh)
AXISVISUALISATION(RThigh)
AXISVISUALISATION(LThighCluster)
AXISVISUALISATION(RThighCluster)
AXISVISUALISATION(LShank)
AXISVISUALISATION(RShank)
AXISVISUALISATION(LShankCluster)
AXISVISUALISATION(RShankCluster)
AXISVISUALISATION(LFoot)
AXISVISUALISATION(RFoot)
AXISVISUALISATION(Anatomy)
AXISVISUALISATION(UTHor)
AXISVISUALISATION(LThor)
AXISVISUALISATION(Lumbar)

{*Angle Calculations*}

PelvisAngle=<Anatomy,Pelvis,xyz>
LHipAngle=<1(PelvisAngle),2(PelvisAngle),3(PelvisAngle)>
RHipAngle=<Pelvis,RThigh,xyz>

LHipAngle=<1(LHipAngle),-2(LHipAngle),-3(LHipAngle)>
RHipAngle=<1(RHipAngle),2(RHipAngle),3(RHipAngle)>

LKneeAngle=<LThigh,LShank,xyz>
LAnkleAngle=<1(LKneeAngle),-2(LKneeAngle),-3(LKneeAngle)>

RKneeAngle=<RThigh,RShank,xyz>
RAnkleAngle=<1(RKneeAngle),2(RKneeAngle),3(RKneeAngle)>

LFootProgressAngle = <Anatomy,LFoot,xyz>
LAnkleAngle=<1(LAnkleAngle),-2(LAnkleAngle),-3(LAnkleAngle)>

RFootProgressAngle = <Anatomy,RFoot,xyz>
RAnkleAngle=<1(RAnkleAngle),2(RAnkleAngle),3(RAnkleAngle)>

LumAngle=<Lumbar,Pelvis,xyz>
LThorAngle=<1(LThorAngle),2(LThorAngle),3(LThorAngle)>

UTHorAngle=<1(UTHorAngle),2(UTHorAngle),3(UTHorAngle)>

OUTPUT(PelvisAngle,LHipAngle,RHipAngle,LKneeAngle,RKneeAngle,LAnkleAngle,RAnkleAngle,LumAngle,LThorAngle,UTHorAngle)
Macro AXISVISUALISATION(Segment)
  ORIGIN#Segment=O(Segment)
  AXISX#Segment={100,0,0}*Segment
  AXISY#Segment={0,100,0}*Segment
  AXISZ#Segment={0,0,100}*Segment
  Output(ORIGIN#Segment,AXISX#Segment,AXISY#Segment,AXISZ#Segment)
Endmacro

macro REPLACE4(p1,p2,p3,p4)
  {*Replaces any point missing from set of four fixed in a segment*}
  s123 = [p2,p1-p2,p2-p3]
  p4V1 = Average(p4/s123)*s123
  s124 = [p2,p1-p2,p2-p4]
  p3V1 = Average(p3/s124)*s124
  s134 = [p3,p1-p3,p3-p4]
  p2V1 = Average(p2/s134)*s134
  s234 = [p3,p2-p3,p3-p4]
  p1V1 = Average(p1/s234)*s234
Endmacro

macro FORCEVECTOR(FP)
  If ExistAtAll( FP )
    F_#FP = FP(1)
    M_#FP = FP(2)
    C_#FP = FP(3)
    if ( ABS ( F_#FP ) > 10 )
\[ P\_\text{#FP} = C\_\text{#FP} + \{ -M\_\text{#FP}(2)/F\_\text{#FP}(3), M\_\text{#FP}(1)/F\_\text{#FP}(3), -C\_\text{#FP}(3) \} \]

else
\[ P\_\text{#FP} = C\_\text{#FP} \]
endif

\[ F\_\text{#FP} = F\_\text{#FP} + P\_\text{#FP} \]

OUTPUT ( P\_\text{#FP}, F\_\text{#FP } )

EndIf
endmacro

*End of macro section*

*Define Optional Markers*

OptionalPoints(RASIS,LASIS,LPSIS,RPSIS)
OptionalPoints(LFLE,LTh1,LFME,LTh2,LTh3)
OptionalPoints(RFLE,RTh1,RFME,RTh3,RTh2)
OptionalPoints(LLC,LC1,LMC,LC2,LC3)
OptionalPoints(RLC,RC1,RMC,RC2,RC3)
OptionalPoints(LMet,LCal,LTube,LExtra)
OptionalPoints(RMet,RCal,RTube,RExtra)

*Create segments*

*Global Segment*

ORIGINGlobal={0,0,0}
Global=[ORIGINGlobal,{1,0,0},{0,0,1},xyz]

AXISXGlobal=ORIGINGlobal+{100,0,0}
AXISYGlobal=ORIGINGlobal+{0,100,0}
AXISZGlobal=ORIGINGlobal+{0,0,100}

Output(ORIGINGlobal,AXISXGlobal,AXISYGlobal,AXISZGlobal)

ORIGINAnatomy={0,0,0}
Anatomy=[ORIGINAnatomy,{1,0,0},{0,0,1},xyz]

AXISYAnatomy=ORIGINAnatomy+{100,0,0}
AXISXAnatomy=ORIGINAnatomy+{0,100,0}
AXISZAnatomy=ORIGINAnatomy+{0,0,100}

Output(ORIGINAnatomy,AXISXAnatomy,AXISYAnatomy,AXISZAnatomy)

*Pelvis Segment*

If $Static == 1 Then
\[ \text{InterASISDist} = \text{RASIS}(2) - \text{LASIS}(2) \]
Endif

REPLACE4(RASIS,LASIS,RPSIS,LPSIS)

PelvisORIGIN=(RASIS+LASIS+RPSIS+LPSIS)/4
SACR=(RPSIS+LPSIS)/2
PELF = (LASIS+RASIS)/2
Pelvis=[PELF,(LASIS-RASIS),(PELF-SACR),xyz]

{"Thigh Segments*}

LHJC=%LHJC*Pelvis
RHJC=%RHJC*Pelvis

REPLACE4(LFLE,LTh1,LTh2,LTh3)
REPLACE4(RFLE,RTh1,RTh2,RTh3)
REPLACE4(LFME,LTh1,LTh2,LTh3)
REPLACE4(RFME,RTh1,RTh2,RTh3)

LThighCluster = [LTh2,(LTh2-LTh1),(LTh3-LTh2),xyz]
RThighCluster = [RTh2,(RTh2-RTh1),(RTh3-RTh2),xyz]

LKJC=(LFLE+LFME)/2
RKJC=(RFLE+RFME)/2

LThigh=[LHJC,(LHJC-LKJC),(LFME-LFLE),zyx]
RThigh=[RHJC,(RHJC-RKJC),(RFLE-RFME),zyx]

{"LThighCluster=ROT(LThighCluster,LThighCluster(1),-$LThighClusterAngle(1))
LThighCluster=ROT(LThighCluster,LThighCluster(2),-$LThighClusterAngle(2))
LThighCluster=ROT(LThighCluster,LThighCluster(3),-$LThighClusterAngle(3)){*}

RThighCluster=ROT(RThighCluster,RThighCluster(1),-$RThighClusterAngle(1))
RThighCluster=ROT(RThighCluster,RThighCluster(2),-$RThighClusterAngle(2))
RThighCluster=ROT(RThighCluster,RThighCluster(3),-$RThighClusterAngle(3))

{"Shank Segments*}

REPLACE4(LLC,LC1,LC2,LC3)
REPLACE4(RLC,RC1,RC2,RC3)
REPLACE4(LMC,LC1,LC2,LC3)
REPLACE4(RMC,RC1,RC2,RC3)

LShankCluster = [LC2,(LC2-LC1),(LC2-LC3),xyz]
RShankCluster = [RC2,(RC2-RC1),(RC2-RC3),xyz]
LAJC = (LLC + LMC) / 2
RAJC = (RLC + RMC) / 2

LShank = [LAJC, (LKJC - LAJC), (LMC - LLC), yxz]
RShank = [RAJC, (RKJC - RAJC), (RLC - RMC), yxz]

{*LShankCluster = ROT(LShankCluster, LShankCluster(1), -$LShankClusterAngle(1))
LShankCluster = ROT(LShankCluster, LShankCluster(2), -$LShankClusterAngle(2))
LShankCluster = ROT(LShankCluster, LShankCluster(3), -$LShankClusterAngle(3)) *}*

RShankCluster = ROT(RShankCluster, RShankCluster(1), -$RShankClusterAngle(1))
RShankCluster = ROT(RShankCluster, RShankCluster(2), -$RShankClusterAngle(2))
RShankCluster = ROT(RShankCluster, RShankCluster(3), -$RShankClusterAngle(3))

{*Foot Segments*}

REPLACE4( LMet, LCal, LLC, LMC )
REPLACE4( RMet, RCal, RLC, RMC )

LFoot = [LAJC, LCal - LMet, LCal - LExtra, yxz]
RFoot = [RAJC, RCal - RMet, RCal - RExtra, yxz]

{*Lumbar Segments*}

REPLACE4( RL1, UTC1, UTC2, UTC3 )
REPLACE4( LL1, UTC1, UTC2, UTC3 )
REPLACE4( RL5, UTC1, UTC2, UTC3 )
REPLACE4( LL5, UTC1, UTC2, UTC3 )

LumCluster = [LumC2, (LumC2 - LumC1), (LumC2 - LumC3), xyz]
LumJC = (RL5 + LL5) / 2
UpperLum = (RL1 + LL1) / 2
Lumbar = [LumJC, (UpperLum - LumJC), (RL5 - LL5), yxz]

LumCluster = ROT(LumCluster, LumCluster(1), -$LumClusterAngle(1))
LumCluster = ROT(LumCluster, LumCluster(2), -$LumClusterAngle(2))
LumCluster = ROT(LumCluster, LumCluster(3), -$LumClusterAngle(3))

{*Lower Thoracic Segments*}

REPLACE4( RT7, LTC1, LTC2, LTC3 )
REPLACE4( LT7, LTC1, LTC2, LTC3 )
REPLACE4( RT12, LTC1, LTC2, LTC3 )
REPLACE4( LT12, LTC1, LTC2, LTC3 )

LThorCluster = [LTC2, (LTC2 - LTC1), (LTC2 - LTC3), xyz]
LTJC=(RT12+LT12)/2
UpperLThor = (RT7+LT7)/2
LThor=[LTJC,(UpperLThor-LTJC),(RT12-LT12),zyx]
LThorCluster=ROT(LThorCluster,LThorCluster(1),-$LThorClusterAngle(1))
LThorCluster=ROT(LThorCluster,LThorCluster(2),-$LThorClusterAngle(2))
LThorCluster=ROT(LThorCluster,LThorCluster(3),-$LThorClusterAngle(3))

{*Upper Thoracic Segments*}
REPLACE4(RT1,UTC1,UTC2,UTC3)
REPLACE4(LT1,UTC1,UTC2,UTC3)
REPLACE4(RT6,UTC1,UTC2,UTC3)
REPLACE4(LT6,UTC1,UTC2,UTC3)
UThorCluster = [UTC2,(UTC2-UTC1),(UTC2-UTC3),xyz]
UTJC=(RT6+LT6)/2
UpperUThor=(RT1+LT1)/2
UThor=[UTJC,(UpperUThor-UTJC),(RT6-LT6),zyx]
UThorCluster=ROT(UThorCluster,UThorCluster(1),-$UThorClusterAngle(1))
UThorCluster=ROT(UThorCluster,UThorCluster(2),-$UThorClusterAngle(2))
UThorCluster=ROT(UThorCluster,UThorCluster(3),-$UThorClusterAngle(3))

{*Output Virtual Markers*}
Output(ORIGINGlobal,PelvisORIGIN,RHJC,LHJC,RKJC,LKJC,RAJC,LAJC,LumJC,LTJC,UTJC)

{* Find the general progression direction of the subject}
{*==============================================================*}
PelvisDirection = AVERAGE( SACR-PELF )
If $Static == 1 Then
  Anatomy = [0(Global),PelvisDirection,3(Global),zyx]
Else
  - PELF[-7]- PELF[-6]- PELF[-5]- PELF[-4]- PELF[-3])/5
  Anatomy = [0(Global),-Progress,3(Global),xyz]
EndIf
Fwd = 3(<Anatomy,Global>)

{*align to +X*} Anatomy = Global
  If (Fwd > -135) AND (Fwd <= -45) Then {*align to -Y*}
    Anatomy = ROT(Anatomy, 3(Anatomy),-90)
  ElseIf (Fwd > 45) AND (Fwd <= 135) Then {*align to +Y*}
Anatomy = ROT(Anatomy, 3(Anatomy),90)
ElseIf (Fwd > 135) OR (Fwd <= -135) Then {*align to -X*}
    Anatomy = ROT(Anatomy, 3(Anatomy),180)
EndIf*

{*Visualise AXIS*}

AXISVISUALISATION(Pelvis)
AXISVISUALISATION(LThigh)
AXISVISUALISATION(RThigh)
AXISVISUALISATION(LThighCluster)
AXISVISUALISATION(RThighCluster)
AXISVISUALISATION(LShank)
AXISVISUALISATION(RShank)
AXISVISUALISATION(LShankCluster)
AXISVISUALISATION(RShankCluster)
AXISVISUALISATION(LFoot)
AXISVISUALISATION(RFoot)
AXISVISUALISATION(Anatomy)
AXISVISUALISATION(LThor)
AXISVISUALISATION(LThor)
AXISVISUALISATION(UThor)

{*Angle Calculations*}

PelvisAngle=<Anatomy,Pelvis,xyz>
PelvisAngle=<1(PelvisAngle),2(PelvisAngle),3(PelvisAngle)>
LHipAngle=<Pelvis,LThighCluster,xyz>
LHipAngle=<1(LHipAngle),-2(LHipAngle),-3(LHipAngle)>
RHipAngle=<Pelvis,RThighCluster,xyz>
RHipAngle=<1(RHipAngle),2(RHipAngle),3(RHipAngle)>
LKneeAngle=<LThighCluster,LShankCluster,xyz>
LKneeAngle=<-1(LKneeAngle),-2(LKneeAngle),-3(LKneeAngle)>
RKneeAngle=<RThighCluster,RShankCluster,xyz>
RKneeAngle=<-1(RKneeAngle),2(RKneeAngle),3(RKneeAngle)>
LAnkleAngle=<LShankCluster,LFoot,xyz>
LAnkleAngle=<1(LAnkleAngle),-2(LAnkleAngle),-3(LAnkleAngle)>
RAnkleAngle=<RShankCluster,RFoot,xyz>
RAnkleAngle=<1(RAnkleAngle),2(RAnkleAngle),3(RAnkleAngle)>
LFootProgressAngle = <Anatomy,LFoot,xyz>
LFootProgressAngle = <1(LFootProgressAngle),2(LFootProgressAngle),
3(LFootProgressAngle)>
RFootProgressAngle = <Anatomy,RFoot,xyz>
RFootProgressAngle =
<1(RFootProgressAngle),-2(RFootProgressAngle),-3(RFootProgressAngle)>
LumAngle=<Lumbar,Pelvis,xyz>
LumAngle=<1(LumAngle),2(LumAngle),3(LumAngle)>
LThorAngle=<LThor,Lumbar,xyz>
LThorAngle=<1(LThorAngle),2(LThorAngle),3(LThorAngle)>
UThorAngle=<UThor,LThor,xyz>
UTHorAngle=<1(UTHorAngle),2(UTHorAngle),3(UTHorAngle)>

OUTPUT(PelvisAngle, LHipAngle, RHipAngle, LKneeAngle, RKneeAngle, LAnkleAngle, RAnkleAngle, LFootProgressAngle, RFootProgressAngle, LumAngle, LThorAngle, UThorAngle, UThorAngle)

ClusterAngleL=<LThighCluster, Global, yxz>
ClusterAngleR=<RThighCluster, Global, yxz>

OUTPUT(ClusterAngleL, ClusterAngleR)
OpenSim code is available upon request. (Greater than 50 pages.)