AN INVESTIGATION OF ELECTROMYOGRAPHIC ACTIVITY DIFFERENCES IN PARTICIPANTS RECOVERED FROM A LOW BACK INJURY

by

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Submitted in partial fulfilment of the requirements

for the degree of Master of Science

at

Dalhousie University

Halifax, Nova Scotia

October 2017

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ABSTRACT

Introduction:
The purpose of this analysis was to investigate for differences in EMG activity between a recovered LBI military and non-military population during a controlled task. Further, differences in EMG activity were also investigated based upon PIT (positive vs. negative responders) as well as re-injury (reinjured vs. non-reinjured) status.

Methods:
A secondary analysis was preformed on EMG data collected from a comprehensive set of muscle sites during the hand transition phase of a standardized dynamic transfer task on group of 32 military as well as 32 non-military recovered LBI subjects.

Results:
Significant differences in EMG activation were not detected based upon group (military vs. non-military) or re-injury status. Differences in EMG activity were detected based upon PIT status including a qualitative clustering of variable 2 ratio scores around 50% for those who tested positive during the PIT compared to those who tested negative.

Conclusions:
As expected, both recovered LBI groups elicited similar neuromuscular responses during hand transition as indicated by the lack of differences in EMG activity. Differences in variable ratio scores including a qualitative clustering of scores in those who tested positive during the PIT may represent a common neuromuscular strategy to the flexion moment during hand transition in comparison to participants who tested negative during the PIT. This study also provides preliminary objective evidence supporting the theoretical framework of the PIT as well as it's generalizability among different LBI populations.

LIST OF ABBRIEVIATIONS USED

LBP	Low back pain
LBI	Low back injury
EMG	Electromyography
PIT	Prone instability test
CPR	Clinical prediction rule
HT	Hand transition
RHT	Right hand transfer
LHT	Left hand transfer
RA	Rectus abdominus
EO	External oblique
MT	Lumbar multifidus
QL	Quadtratus lumborum
TrA	Transversus abdominis
IO	Internal oblique
ES	Erector spinae
EO1	Anterior fibers of external oblique
EO2	Lateral fibers of external oblique
EO3	Posterior fibers of external oblique
URA	Upper rectus abdominus
LRA	Lower rectus abdominus
CSA	Cross sectional area
DFV	Digital fluoroscopic video
PAIVMs	Passive accessory intervertebral motion tests
PA	Posterior-anterior

mCPR	Modified clinical predication rule
MVIC	Maximum voluntary isometric contraction
OA	Osteoarthritis
ICC	Intraclass correlation
FOB	Flock of Birds
PCA	Principal component analysis
PC	Principal component
VAS	Visual analogue scale
RMQ	Roland Morris questionnaire
RMS	Root mean square
PCS	Pain catastrophizing scale
NRMS	Normalized root mean square
BMI	Body mass index
ANOVA	Analysis of variance

ACKNOWLEDGMENTS

I would like to start by thanking my supervisor Dr. John Kozey for his insight, dedication, and unwavering support throughout this entire process. His timely advice and expertise was crucial to the completion of this thesis and I am forever grateful for his guidance. I would also like to thank my supervisory committee including Dr. Cheryl Hubley-Kozey and Dr. Janice Moreside for their support and patience as well as their significant contributions to this project through previous works. A sincere thank-you to Dr. Anne Fenety for allowing me to pursue the completion of my studies through her role as my external examiner. A special thank you to Adam for your extensive lab knowledge and selfless assistance with data gathering and processing. You are an invaluable asset to the Neuromuscular Functions Lab. I would also like to sincerely thank Dan for allowing me to assist with data collections and helping me to become proficient with the laboratory protocols as well as your general assistance throughout this project. Lastly, I would like to thank my family with love and admiration. To my wife Amy, your calming support and encouragement as well as enduring patience throughout this journey has forever strengthened our bound. You inspire me every day, I love you. Our family has certainly grown since this journey began. To Cameron, Emily, and Owen; Daddy loves you very dearly and thank you for always reminding me about the things in life that matter most.

CHAPTER 1 INTRODUCTION

Low back pain (LBP) is currently one of the major concerns in healthcare (O'Sullivan, 2005). In the majority of cases LBP is non-specific, meaning that less than 10% of individuals experiencing LBP can be assigned a specific diagnosis based on anatomical pathologies such as nerve root compression, vertebral fracture, tumor, inflammatory disease, infection, spondylolithesis or spinal stenosis (Delitto, Erhard, & Bowling, 2012). Consequently, this large, heterogeneous group (90%) of individuals with LBP have been classified as having "non-specific" or "mechanical" LBP. In order to improve treatment outcomes is growing evidence suggesting that the homogeneous sub-classification of patients with non-specific LBP may be possible based upon similar signs and symptoms as well as patho-anatomical, psychological and social characteristics (Frymoyer, Rosen, Clements, & Pope, 1985; O'Sullivan, 1997; Nachemson, 1999; Skouen, Grasdal, Haldorsen, & Ursin, 2002; Fritz, Delitto, & Erhard, 2003; Stuge, Laerum, Kirkesola, &Vollestad, 2004). One sub-classification is lumbar spine instability, which has been used to describe individuals with non-specific LBP, is based upon documented changes in trunk neuromuscular control. However, spinal instability is poorly defined, and concepts linking spinal instability and LBP have been controversial (Panjabi, 2003).

The control of joint motion and movement coordination is complex. It requires contribution and input from structures surrounding a joint, combined with additional sensory information provided by visual, vestibular, and somatosensory systems (Riemann, 2002a; van Dieen, Selen, & Cholewicki, 2003). Spinal stability has been most commonly described as the ability of the stabilizing system of the spine to maintain the intervertebral neutral zone within the physiological limits so that there is no neurological dysfunction, no major deformity and no incapacitating pain (Panjabi, 1992a). The neutral zone has been previously defined as a region of

intervertebral motion around the neutral posture where little resistance is offered by the passive subsystem as defined by Panjabi's model of spinal stability (Panjabi, 1992a). This zone appears to be a clinically important measure of spinal stability as it's size may increase with injury to the spinal column resulting in impending spinal instability and subsequent spinal injury. Unlike other proposed models of spinal stability, Panjabi's more clinically focused definition regards spinal stability as a continuously variable phenomenon in which the system can be more or less stable (Bergmark, 1989; Cholewicki & McGill, 1996; Panjabi, 1992a). According to Panjabi et al., the responsibility of maintaining joint stability can be partitioned into three functionally interdependent subsystems (passive, active, and neural) that possess the functional abilities to contribute to joint stability (Panjabi, 1992a; Panjabi, 1992b; Panjabi, 2003). Briefly, the passive subsystem including joint structures such as the vertebrae, facet articulations, intervertebral discs, spinal ligaments, and joint capsules as well as the passive mechanical properties of the spinal muscles produce passive stiffness (force) which increases toward the end ranges of spinal motion (Panjabi, 1992a). The active subsystem, comprised of trunk muscles surrounding the spine, has been shown to be a major contributor to spinal stability through synchronous levels of co-activity resulting in increased active stiffness and motion control (McGill, 2003; Cholewicki & Reeves, 2004). The neural control subsystem, through direct or indirect innervations with motor neurons, interacts with the other two subsystems and is responsible for the timely planning and delivery of all requirements for spinal stability and the generation of appropriate muscle responses (Panjabi, 2006). Appropriate neuromuscular responses are the result of precise interplay among all three sub-systems, demonstrating the inability of any one subsystem to act in isolation (Panjabi, 1992a; Riemann, 2002a; Hodges, 2003). Previous modeling work on spinal stability has shown the importance of coordinated motor activation patterns, suggesting that only one muscle eliciting an

inappropriate response could be significant enough to disrupt lumbar spine stability (McGill, 2003; Cholewicki et al, 2004).

Documented changes in any one or all three subsystems have been linked to decreased lumbar spine stability and subsequent non- specific LBP. Specifically, alterations in the active and neural control subsystems have been demonstrated using surface electromyographic (EMG) recordings from various LBP populations. Changes in activation amplitudes, timing and muscle activation patterns during expected and unexpected spinal perturbations as well as during a variety of dynamic tasks have been documented in chronic LBP individuals as well as in recurrent LBP individuals or individuals in the sub-acute phase of a low back injury (LBI) during symptom remission (Hodges & Richardson, 1999; Hubley-Kozey & Vezina, 2002b; MacDonald, Moseley, & Hodges, 2010; MacDonald, Dawson, & Hodges, 2011; Moreside, Quirk, & Hubley-Kozey 2014; Radebold, Cholewicki, Panjabi, & Patel, 2000; van Dieen et al., 2003). Growing evidence suggests that EMG can accurately differentiate those with and those without LBP promoting the use of EMG as an objective measure of LBP (Geisser et al, 2005). Butler et al. and MacDonald et al. have also shown the ability of EMG recordings to differentiate between those individuals who have been deemed recovered from a LBI and healthy controls (Butler, Hubley-Kozey, & Kozey 2013; MacDonald et al., 2010; MacDonald et al., 2011). In a follow-up study to Butler and colleagues, Hubley-Kozey et al. demonstrated a difference in EMG activity levels during a dynamic functional task in those who had a recurrent LBI and those who did not (Hubley-Kozey, Moreside, & Quirk, 2014). Additionally, evidence has also demonstrated the potential of EMG measures to predict those who will sustain a first-time LBI (Cholewicki et al., 2005; Heydari, Nargol, Jones, Humphrey, & Greenough, 2010). Collectively, these studies illustrate common

neuromuscular control adaptations utilized by various LBP and LBI populations as well as the ability of EMG measurements to detect such adaptations.

Although lumbar spine instability has been considered a significant contributor to non-specific LBP, this subgroup remains controversial due to the complexity of the pathomechanical consequences of spinal instability and minimal evidence linking these consequences with severity of clinical signs and symptoms (Dupuis, Yong-Hing, Cassidy, & Kirkaldy-Wills, 1985; Paris, 1985; Panjabi, 1992a; Fritz, Erhard, & Hagen, 1998). Additionally, given the vast number of proposed anatomical contributors to spinal dysfunction resulting in decreased spinal stability and the documented compensations among the three stabilizing subsystems, lumbar spine instability is a challenging condition to diagnose clinically. Common subjective and objective clinical descriptors (Maigne, Lapeyre, Morvan, & Chateellier, 1976; Paris, 1985; O'Sullivan, 2000; Hicks, Fritz, Delitto, & Mishock, 2003; Cooks et al., 2006; Demoulin, Crielaard, & Vanderthommen, 2007) as well as special clinical tests (Dilitto, Erhard, & Bowling, 1995; Magee, 1997; Kasai, Morishita, Kawakita, Kondo, & Uchida, 2006) have been used to diagnose this condition. It has been generally assumed that clinical measures of spinal instability challenge the passive subsystem through some form of static clinical test or dynamic movement assessment resulting in pain or aberrant movement in those suspected of deficient spinal stabilizing function (Panjabi, 2003; Hicks, Fritz, Delitto, & McGill, 2005). However, most clinical measures have been documented on symptomatic populations bringing into question the effect of test-induced pain and avoidance of test-induced painful stimuli on positive test outcomes.

Currently, the prone instability test (PIT) is arguably the most reliable clinical measure of lumbar spine instability (Hicks et al., 2003; Fritz, Piva, & Childs, 2005a; Schnieder et al., 2008). The test, originally published by Magee (1997), is comprised of two progressive interdependent

components (passive and active) that theoretically challenge all three spinal stabilizing subsystems. A positive result is noted by a reduction in pain (induced by a manual posterior-anterior spinal force) following a bilateral leg lift maneuver that increases active subsystem contribution to spinal stability (active phase). Investigations have shown the PIT to have moderate to good interrater reliability with kappa values ranging from 0.54-0.87 (Hicks et al., 2003; Fritz et al., 2005a; Schnieder et al., 2008). In the absence of a true diagnostic standard for identifying lumbar spine instability the link between LBP/LBI and lumbar instability is often confirmed upon a favorable response to lumbar stabilization exercises. In a study by Hicks and colleagues, investigators established a preliminary multivariate clinical prediction rule (CPR) that included four predictors for lumbar stabilization exercise success including age<40 years, average straight leg raise > 91°, aberrant movement present and a positive PIT (Hicks et al., 2005). Of these predictors, only a positive PIT and aberrant movement were significant univariate predictors of stabilization success. These findings were supported by Rabin and colleagues who developed a modified CPR which included only the PIT and aberrant movement as predictors of stabilization success (Rabin, Shashua, Pizem, Dickstein, & Dar, 2014).

Previously documented special tests (Dilitto et al., 1995; Magee, 1997; Kasai et al., 2006) as well as common subjective and objective clinical descriptors used to diagnose lumbar spinal instability have been largely based upon observations made from symptomatic populations. Further, all but one previous investigation (Hicks et al., 2003) of the PIT studied subjects from a single population or recruitment source with similar population characteristics resulting in potentially poor generalizability of the study results. Theoretically, the PIT should elicit the same clinical responses in all LBI populations with deficient passive subsystem contribution to spinal stability regardless of differing demographic characteristics as well as presence or absence of

symptoms. In fact, the PIT is the only specific clinical test of spinal instability that is expected to produce the same result in LBI populations who are both symptomatic and asymptomatic at the time of examination (Magee, 1997; Rabin et al, 2014). In considering the theoretical basis of the PIT, assessment of the passive stabilizing structures of the spine during the passive component negates all active and neural subsystem contribution to spinal stability and should elicit a positive response in all individuals devoid of sufficient passive stiffness regardless of demographic factors. This concept speaks to the expected generalizability of the clinical measure when comparing across LBI populations with different demographic characteristics. Similarly, because of it's generally simplistic application, the PIT has been considered the most practical clinical measure of spinal instability (Hicks el al., 2003) also perhaps relating to it's documented moderate to good interrater reliability (Hicks et al., 2003; Fritz et al., 2005a; Schnieder et al., 2008).

Although objective investigation of the PIT as well as other specific clinical tests for spinal instability has proven difficult, a previous study detected minor differences in back extensor EMG activation patterns during a highly-controlled transfer task between those who tested positive and negative during the PIT in a group of 32 military workers deemed recovered from a LBI (Trudel, 2014). However, the author concluded that the findings were not clinically relevant and that the PIT failed to separate the group such that each subgroup had distinct muscle activation patterns that could impact clinical decision making (Trudel, 2014). In an earlier analysis, Butler and colleagues used EMG measures captured during a symmetrical bilateral lifting task to demonstrate altered neuromuscular patterns in a LBI group deemed ready to resume regular activities when compared to a non-LBI group (Butler et al., 2013). Additionally, modest evidence has been presented suggesting the ability to predict LBI reoccurrence in a recovered LBI group using EMG data captured during the same transfer task used by Trudel (Hubley-Kozey et al., 2014). This

previously documented standardized task has been developed and shown to create a highly controlled transfer from the right to left side of the body resulting in continuously changing flexion and lateral flexion moments around the spine created primarily by the external load (Butler, Hubley-Kozey, & Kozey, 2010). Upon comparison, demographic characteristics may be different between the military population used by Trudel et al. and the non-military population used by Hubley-Kozey and colleagues (Hubley-Kozey et al., 2014; Trudel, 2014). Despite the fact that a comparison between those who tested positive and negative during the PIT was not an objective investigated by Hubley-Kozey et al., the test was performed as part of a standardized physiotherapy assessment conducted on all subjects enrolled in the study. In contrast to a largely PIT positive study population used in the investigation by Trudel and colleagues (n=23, 72%), the study population used by Hubley-Kozey et al. demonstrated a much lower percentage of LBI individuals who tested positive during the PIT (n=7, 23%) (Hubley-Kozey et al., 2014; Trudel, 2014).

An important aspect of the abovementioned investigations of differing LBI populations is the definition of recovery. These previously investigated populations were within the subacute phase of a LBI (between 4 and 12 weeks post LBI) and were deemed recovered based upon multiple factors including reduction of pain and disability as well as restoration of normal activities (Hubley-Kozey et al., 2014; Trudel, 2014). It has been suggested that a minimum pain-free period of 30 days may be used as an indicator of recovery (de Vet et al., 2002). This criterion has been challenged as it represents a single factor (pain) in a potential multi-factorial phenomenon. Therefore, over-and-above pain, additional factors including measures of low back-related disability and function may be required to develop a more robust criterion for evaluating recovery (Stanton, Latimer, Maher, & Hancock, 2009). Further, despite mounting efforts to determine

minimally important clinically (MIC) differences when assessing the efficacy of treatments for LBP, agreement regarding appropriate outcome criteria for defining recovery is still lacking (Mehling et al., 2011). Two of the more commonly used self-reported measures of low back pain and disability are the Visual Analog Scale (VAS) and the Roland Morris Disability Questionnaire (RMQ), respectively. Both measures have been shown to be valid and reliable for detecting reduction of pain (VAS) and an increase in overall function (RMQ) (Resnik & Dobrzykowski, 2003; Roland & Fairbank, 2000). However, a low VAS score does not clearly distinguish between those who view themselves as recovered from those who do not (Hush et al., 2009). In addition, cut-offs for MCI differences vary widely from 0-20mm on the VAS to 0-8 points on the RMQ making interpretation difficult (Hilfeker et al., 2007; Kent & Keating, 2008; Fritz, Hebert & Koppenhaver, 2009). In considering these challenges as well as the previous recommendation of combining pain with other important determinants of recovery, the definition of recovery used in this analysis was consistent with the definition used by Hubley-Kozey and colleagues as well as Trudel which included i) a pain level of less than 20mm on the VAS ii) a score of less than 8 on the RMQ and iii) resumption of normal activities (Hubley-Kozey et al., 2014; Trudel, 2014). Allowing for a more objective definition of recovery, this criterion also discourages misguided interpretation of recovery resulting from a cause and effect relationship between decreased pain (VAS) and dysfunction (RMQ) resulting from lack of usual activity.

Similar to recovery, an operational definition of re-injury or recurrence must be outlined for the completeness of this secondary analysis. Defining recurrence presents similar challenges to those related to the definition of recovery. Specifically, failure to differentiate recurrence or re-injury from persistence of low grade LBP as well as aggravation of an original episode following a LBI will result in overestimation of low back reinjuries (Stanton et al., 2009). Therefore, as

previously defined by Hubley-Kozey et al., re-injury was defined in this study as re-injury episode to the low back resulting in lost time from work and/or normal activities or requiring medical attention during a one-year follow-up period (Hubley-Kozey et al., 2014). The concept of recovery is critical in this definition as previously defined indicating that a participant has both i) fully recovered from the original episode and ii) subsequently experiences a new and separate LBI episode.

The intent of this secondary analysis was to investigate for potential differences in these previously investigated military and non-military populations by comparing EMG activity during a portion of the abovementioned transfer task. Specifically, due to the presumably similar effect of the flexion moment generated during the PIT and the hand-transition (HT) phase of the transfer task, only EMG data captured during HT were used in this investigation for all comparisons. In addition, EMG comparisons between all participants who tested positive and negative during the PIT were also conducted. Confirmation of differences in EMG activity detected based upon PIT status (positive vs. negative) in a diverse group of recovered LBI subjects could add preliminary evidence supporting the test as an appropriate clinical measure of neuromuscular control that is generalizable among various LBI populations. Comparisons in EMG activity were also conducted between recovered LBI individuals who had sustained a reinjury during a one-year follow-up period and individuals who had not reinjured over the same timeframe. Although not a main objective of the study, PIT status was qualitatively assessed between those who reinjured and those who did not reinjure. Confirmation of discrepancies in PIT status between those who reinjured and those who did not reinjure could add preliminary evidence supporting the PIT as a potential factor associated with re-injury. Therefore, the overall goal of this thesis was to use a documented highly-controlled functional task and the objective physiologically-relevant EMG measures

captured during the task to compare between two recovered LBI groups that have been previously investigated and sampled from different LBI populations (Hubley-Kozey et al., 2014; Trudel, 2014). In addition, the effect of PIT as well as re-injury status (reinjured vs. non-reinjured) on EMG activity was also investigated.

1.1 PURPOSE

The purpose of this study was to expand current clinical knowledge by comparing EMG activity captured during a previously documented standardized functional task with a specific clinical test for spinal instability. This was accomplished by performing a secondary analysis of EMG data captured from a comprehensive set of muscle sites from two previously documented study groups recruited from presumably different recovered LBI populations.

1.2 OBJECTIVES

The main study objective was to:

1. To determine if differences in trunk muscle EMG activity could be detected during a standardized functional task between two groups recruited from different recovered LBI populations (military and non-military). Two sub objectives were to:

    1.1. determine if differences in trunk muscle EMG activity could be detected between recovered LBI participants based upon a positive or negative response during the PIT.

    1.2. determine if differences in trunk muscle EMG activity could be detected between recovered LBI participants based upon the presence or absence of a re-injury.

1.3 HYPOTHESIS

The main hypothesis of this investigation is that significant differences in EMG activity will not be detected during the HT phase of the transfer task based upon group status (military vs. non-military). It is assumed that both groups will have similar neuromuscular responses to the flexion moment generated during HT resulting in minimal differences in EMG activity. Additionally, because the PIT theoretically tests the ability of the active subsystem to adequately compensate in the presence of a deficient passive subsystem, it is hypothesized that PIT status will detect differences in EMG activity during HT due to a potential increase in overall trunk muscle activation in PIT positive participants in response to the increased flexion moment during HT. Lastly, it is hypothesized that the re-injury status (reinjured vs. non-reinjured) will also detect differences in EMG activity during HT. It is believed that those who re-injure will also have overall increased EMG activation of trunk muscle sites compared to non-reinjured participants as a response to the increasing flexion moment.

1.4 ASSUMPTIONS

The assumptions of this secondary analysis are:

- That electrode placement was consistent during each collection.
- That minimal movement occurred between the electrodes and skin during EMG collections
- That no learning effect occurred between trials.
- That fatigue was not present during the testing.
- That the subjects were able to exert their maximal voluntary isometric contraction during the normalization exercises.

CHAPTER 2 LITERATURE REVIEW

An extensive body of literature has examined non-specific LBP for the purpose of gaining knowledge pertinent for LBP prevention as well as the treatment and recovery of LBP. This literature review will attempt to focus on relevant concepts that apply directly to the objectives and hypothesis of this study. Specifically, the purpose of this literature review is to analyse theoretical concepts as well as objective experimental evidence related to a subgroup of non-specific LBP that has been associated with changes in trunk muscle neuromuscular control. The concept of spinal stability will be defined and a review of important documented changes in neuromuscular control that have been linked to spinal instability as well as non-specific LBP and LBI will be presented. Additionally, an important consideration for this project is the clinical diagnosis of lumbar spine instability and therefore, current concepts surrounding clinical measures of lumbar spine instability will be discussed. Particular attention will be given to a special clinical test for lumbar spine instability, the prone instability test. Finally, because of its significance to the methodology of this study as well as its relevance to the LBP/LBI literature, this literature review will examine applicable concepts surrounding electromyography (EMG).

2.1 Sub-classification of Low Back Pain

LBP has been referred to as an epidemic related problem with a reported one-year incidence rate as high as 36% (Delitto et al., 2012) with up to 62% of episodic LBP individuals experiencing a recurrence of LBP within a one-year timeframe (Maetzel & Li, 2002). More recently, the challenges related to the proper diagnosis and subsequent management of LBP has become abundantly clear to all invested in the epidemic including both researchers and clinicians. With a limited percentage of LBP cases receiving structural diagnoses based upon validated diagnostic measures, diagnostic validity is questioned for the remainder of LBP diagnoses that are often made

clinically in a large majority of LBP cases (O'Sullivan, 2005). Adding to the complexity of non-specific LBP, it has been well established that non-specific LBP is a multi-dimensional problem consisting of pathoanatomical, neurophysiological, physical and psychosocial factors (McCarthy, Arnall, Strimpakos, Freemont, & Oldham, 2004; Waddell, 2004).

As a result of these inherent complexities, the identification of proper management options and effective treatment strategies for individuals diagnosed with non-specific LBP remains a great challenge. In an attempt to improve treatment outcomes there has been growing evidence suggesting the sub-classification of patients with non-specific LBP could promote more efficient and tailored treatments (Frymoyer et al., 1985; O'Sullivan, Twomey, Allison, Sinclair, Miller, & Knox, 1997; Nachemson, 1999; Skouen et al., 2002; Fritz et al., 2003; Stuge et al., 2004). Several systems have been used to classify non-specific LBP patients into homogenous sub-groups including patho-anatomical (Bernard & Kirkaldy-Willis, 1987; Newton, Curtis, Witt, & Hobler, 1997), signs and symptoms (Delitto et al., 1995), prognosis (Engel & von Korff MKaton, 1996; Dionne et al., 1997), psychosocial (Keefe & Williams, 1990; Main, Wood, Hollis, Spanswick, & Waddell, 1992; Waddell, 2004) and mechanism (MacKenzie, 1981; Sahrmann, 2001) based models. The shift from viewing LBP as a patho-anatomical disorder, to viewing LBP as a multifactorial bio-psycho-social disorder is now well accepted.

One such subgroup of non-specific LBP has been categorized based upon the presence of lumbar spinal instability as demonstrated by changes in trunk neuromuscular control (van Dieen et al, 2003; Hides et al., 2009; Hodges et al, 2001) as well as specific clinical findings (Alqarni, 2011; Demoulin et al., 2007; Hicks el al., 2003). Clinical prediction rules have been established to identify those individuals with LBP most likely to benefit from stabilization exercises (Hicks et al., 2005, Robin et al, 2014). The ability to detect individuals with altered trunk neuromuscular

control clinically enables prescription of effective neuromuscular control rehabilitative strategies conducive to complete recovery from episodic LBP. Recently, the clinical importance of identifying alterations in trunk neuromuscular control has been demonstrated further as investigators have shown persistent trunk neuromuscular alterations in individuals deemed recovered from a subacute LBI based on self-reported remission of symptoms and resumption of normal activities (Butler et al., 2013; Hubley-Kozey et al., 2014, Moreside et al., 2014).

2.2 Defining Spinal Stability – Panjabi's Model

Bergmark described the presence of two muscle systems that share the responsibility of maintaining spinal stability (Bergmark, 1989). The "global" muscle system including the rectus abdominus (RA), the external obliques (EO), and the thoracic portion of the iliocostalis are capable of producing large amounts of torque and provide general trunk stabilization. The "local" muscle system, having direct attachments to the lumbar vertebrae, is responsible for providing segmental stability or motion control between adjacent segments (Bergmark, 1989). The lumbar multifidus (MT), psoas major, quadratus lumborum (QL), transversus abdominis (TrA), diaphragm, posterior fibers of the internal obliques (IO), and lumbar portions of the iliocostalis and longissimus are all muscles defined as being part of the local system (Bergmark, 1989). Cholewicki and McGill reported the importance of coordination between the global and local systems during functional activities to ensure spinal stability (Cholewicki & McGill, 1996). Further, these authors have suggested the lumbar spine is more apt to become unstable during low load activities requiring low force illustrating the importance of increased activation (stiffness) of the local muscles during such activities (Cholwicki & McGill, 1996). It has also been proposed that co-activation of local system muscles such as the TrA/IO and lumbar MT has an intersegmental stiffening effect resulting in a stable base in which the global muscles can optimally perform (Wilke, Wolf, Claes,

Arand, & Wiesend, 1995; Hodges & Richardson, 1996). A limitation to Bergmark's model is that it based largely on static equilibrium which fails to consider the neuromuscular demands of dynamic stabilization.

According to Panjabi's definition, the responsibility of maintaining joint stability can be partitioned into three functionally interdependent subsystems (passive, active, and neural) that possess individual abilities that contribute to joint stability (Panjabi, 1992a; Panjabi, 1992b; Panjabi, 2003). Specifically, Panjabi defined spinal stability as "the ability of the stabilizing system of the spine to maintain the intervertebral neutral zones within the physiological limits so that there is no neurological dysfunction, no major deformity, and no incapacitating pain" (Panjabi, 1992a). This model relies on precise interplay between all three subsystems whereby deficiencies in a single subsystem or combination of subsystems could result in spinal dysfunction and subsequent spinal injury. Unlike other proposed models of spinal stability, Panjabi's model may be more clinically relevant in that it regards spinal stability as a continuously variable phenomenon in which the system can be more or less stable (Bergmark, 1989; Cholewicki & McGill, 1996; Panjabi, 1992a). This section will review the contributions made by each subsystem to spinal stability and some of the important documented changes in these subsystems that have been linked to LBI and LBP.

2.2.1 The Passive Subsystem

The passive subsystem represents passive stiffness generated by joint structures such as the vertebrae, facet articulations, intervertebral discs, spinal ligaments, and joint capsules as well as the passive mechanical properties of the spinal muscles (Panjabi, 1992a). Components of the passive subsystem are responsible for developing reactive forces required to resist spinal motion

towards end range of spinal motion (Panjabi, 1992a). The passive tissues can be loaded in a neutral position where initial strain lengthens the tissue with minimal resistance. This region of minimal passive resistance is known as the "neutral zone" of a tissue (Panjabi, 1992b). In the case of LBP and LBI, it is hypothesized that this "neutral zone" becomes larger due to pathological changes in the passive spinal structures. Once the passive tissues are challenged beyond the "neutral zone" they apply a stress (force per cross sectional area) that is proportional to the amount of tissue strain (or deformation) (Solomonow, 2011). The passive stiffness generated is represented by the stress-strain relationship of the passive tissues. The general response of passive tissue to generate stiffness is rather complex and non-linear, and subjected to several time-sensitive phenomena including creep, tension-relaxation, strain rate and hysteresis (Solomonow 2009, 2011).

2.2.2 The Active Subsystem

Over the past few decades, an important aspect of joint stability has been introduced placing emphasis on the contribution of the musculature associated with a joint. Preparatory and reflexive activation of muscles has been shown to substantially add to the stability of a joint with the antagonistic muscles contributing significantly to joint stiffness through generation of compressive and posteriorly directed forces as well as increased intra-abdominal pressure through a mode known as co-activation (or co-contraction) with the agonist muscles (Cholewicki, Juluru, Radebold, Panjabi, & McGill, 1999; Solomonow, 2011). The osteoligamentous lumbar spine (in vitro spine with all non-passive structures removed) has been shown to have an insignificant load-carrying capacity of 90N (<20lbs) (Morris et al., 1961). When considering the active subsystem contribution to generation of spinal stiffness, the load-carrying capacity of the lumbar spine has been estimated to be as high as 1500N (Nachemson & Morris, 1964). The active subsystem is comprised of the muscles and tendons that contribute to spinal stability (Panjabi, 1992a). The

trunk musculature surrounding the spine can generate the appropriate tension responses to changing external moments during dynamic tasks resulting in increased spinal stiffness and motion control. Compared to passive stiffness generated by the passive subsystem, the active subsystem is also capable of modulating joint stiffness in the neutral zone.

Several trunk muscles have important documented contributions to lumbar spine stability. The RA and the erector spinae (ES) work to control sagittal plane spinal motion with the RA being the main agonist controlling spinal flexion and the ES being the main agonist controlling spinal extension. The lumbar components of the ES (iliocostalis lumborum and pars lumborum) connect to the mamillary, accessory, and transverse processes of the lumbar vertebrae and originate over the posterior sacrum and medial aspect of the iliac crest (McGill, 2007). The line of action of these muscles has a posterior and caudal direction that cause them to generate posterior shear forces along with extensor moment on the superior vertebrae. The lumbar ES contribute to active stiffness through generation of posterior shear forces that stiffen the spine adding resistance to any anterior reaction shear forces of the superior vertebrae that are produced during spinal flexion (McGill, 2007). This role is altered with increasing spinal flexion as these muscles lose their oblique line of action and reorient to the compressive axis of the spine (McGill et al., 2000).

Researchers have focused significantly on the TrA and its role in lumbar spine stability due to its belt-like containment of the abdomen creating a spinal "corset" as well as its ability to tension the thoracolumbar fascia and increase intraabdominal pressure (IAP) (Hodges & Richardson, 1996; Richardson & Jull, 1995). Previous work has shown that lateral forces generated by the TrA and the IO are transmitted to the thoracolumbar fascia via their attachments to the lateral border adding tension to the fasica resulting in increased spinal stiffness (Gracovetsky, Farfan, & Lamy, 1981). The TrA and the inferior portion of IO have been shown to have similar fiber orientation

as well as a high degree of coupling in a wide variety of nonballistic extertions suggesting synergistic function (Hodges & Richardson, 1996; Junker, McGill, & Kropf, 1998). The EO is responsible for controlling trunk rotation and also the EO and IO have been shown to increase activity under pure axial compressive loads further suggesting a role in lumbar spine stability (McGill, 1991; Junker et al., 1998). The QL is another important spinal stabilizer as it attaches each lumbar vertebra (transverse process) to the rigid pelvis and rib cage effectively buttressing the adjacent vertebrae bilaterally (McGill, 2007). The QL has also been shown to undergo minimal changes in length during spinal motion suggesting a role in generating active stiffness through isometric contraction (McGill, 1991).

There is strong evidence that the lumbar MT is responsible for controlling spinal segmental motion (McGill, 1991; Bogduk, Macintosh, & Pearcy, 1992; Richradson &Jull, 1995, O'Sullivan et al., 1997). It has been documented that the MT contributes approximately two thirds of total stiffness at L4/L5 (Wilke et.al, 1995) and studies have also shown MT activity to increase intervertebral stiffness at an injured lumbar segment (MacDonald et al., 2006). The MT has been previously divided into deep and superficial fibers based on anatomical (Macintosh, Valencia, Bogduk, & Munro, 1986) and biomechanical (Bogduk et al., 1992) discrepancies. Fibers of the MT that cross two spinal levels and insert onto the lamina, mamillary process and joint capsule have been referred to as the deep fibers of MT (Macintosh et al., 1986). The superficial fibers make up the greatest muscle mass of the MT crossing more than two spinal levels and inserting caudally onto the mamillary process, lamina and posterior superior iliac spine and dorsal sacrum (Macintosh et al., 1986). Biomechanical models, based on anatomical data, suggest the superficial fibers produce sufficient torque to create posterior sagittal rotation (extension) of the lumbar spine in combination with intervertebral compression resulting in enhanced lumbar spine stiffness

(Macintosh et al., 1986; Bogduk et al., 1992). In contrast, the deep fibers of the MT are near the predicted axis of rotation of the lumbar segments primarily generating compressive forces with minimal associated torque (Bogduk et al., 1992) resulting in enhanced segmental stabilization of the lumbar spine (Macintosh et al., 1986; Kay, 2000; Jemmett, Macdonald, & Agur, 2004). Previous modeling work on spinal stability has demonstrated the importance of these trunk muscles acting in a coordinated manner to maintain spinal stability suggesting that only one muscle eliciting an inappropriate contraction could be significant enough to disrupt lumbar spine stability (McGill, Grenier, Kavcic, & Cholewicki, 2003; Cholewicki et al, 2004).

2.2.3 *The Neural Control Subsystem*

The neural control subsystem is responsible for the timely evaluation and determination of all requirements for spinal stability and the generation of appropriate muscle responses (Panjabi, 2006). Spinal stability is a result of dynamic, highly coordinated muscle activation patterns involving many muscles (McGill et al., 2003). In order to achieve spinal stability at any instance in time, the neural control subsystem must make complex decisions on how to redistribute muscle tensions following changes in posture or external loads while taking into consideration masses, inertias, and accelerations associated with a given task (Panjabi, 1992a; Panjabi, 2006). Both cortical responses and subcortical reflex responses to spinal perturbations are mediated by the neural control subsystem through direct or indirect innervations with motor neurons that function to coordinate the level of tension of various trunk muscles (McGill et al., 2003). In response to a spinal perturbation leading to tissue deformation, mechanoreceptors or transducers positioned in muscles (Riemann, 2002a; Brown and McGill, 2009) and passive tissues (ligaments, joint capsules, and the annulus fibers of the vertebral disc) (Riemann, 2002a, Solomonow, 2009) quantitatively transduce mechanical events in their host tissues into neural signals resulting in

generation of an inhibitory or excitatory reflex response to the motor unit (Riemann, 2002a). These appropriate responses are generated based upon the afferent input from the peripheral mechanoreceptors resulting in the recruitment of motor units in the correct sequence, at the correct time, and with the correct amplitude to achieve joint stability (Riemann, 2002a; Hodges & Moseley, 2003).

Generation of a muscular response to an unexpected joint perturbation to enhance active stiffness and joint stability, as discussed above, can be termed a feedback mediated response (Riemann, 2002a). Conversely, preparatory responses to predictable disturbances, or feedforward mediated responses, have been described as anticipatory muscle activation occurring in advance of limb movements to prepare the body for perturbations caused by the movement (Hodges, 2001). Using sensory information to appropriately adjust muscle tension prior to a given joint perturbation can actively stiffen the joint in preparation for the force application resulting in reduced joint motion (Hodges and Richadson, 1999; Hodges, 2001). Feedforward mediated responses elicited by superficial trunk muscles (RA and ES) (Bouisset and Zattara 1981; Aruin and Latash, 1995) as well as deep trunk muscles (TrA and MT) (Hodges and Richardson, 1997; Moseley, Hodges, & Gandevia, 2002) have been well documented in the literature prior to upper extremity movements. The responsibility of the neural subsystem to anticipate (feedforward) and respond (feedback) to spinal perturbations with timely and efficient neuromuscular control has a direct effect on spinal stability. Spinal stiffness may be compromised with even one trunk muscle eliciting an insufficient contraction to counter balance moments generated from a spinal perturbation. This decrease in neuromuscular control may result in a LBI (McGill et al., 2003; Cholewicki et al, 2004).

2.2.4 Spinal Instability: Dysfunction of the Spinal Stabilizing System

Spinal instability is considered to be one of the important causes of LBP but is poorly defined and concepts linking spinal instability and LBP have been controversial (Panjabi, 2003). A number of researchers have used clinical findings (Fritz et al, 1998; Paris, 1985) as well as theoretical (Frymoyer et al, 1985; Bergmark, 1989; Panjabi, 1992a; Panjabi, 1992b;) and modeling work (Cholewicki & McGill, 1996) to link changes in trunk neuromuscular control to lumbar spine instability and LBP. There is convincing evidence of decreased muscle strength (Suzuki & Endo, 1983; Mannion, 1999) and altered neuromuscular control (Hodges & Richardson, 1999; Hubley-Kozey & Vezina, 2002; MacDonald et al., 2010; MacDonald et al., 2011; Moreside et al., 2014; Radebold et al., 2000; van Dieen et al., 2003) as well as trunk muscle histochemical and morphological changes (Mattila et al., 1986; Zhu et al., 1989; Mannion, 1999; Hides et al., 1994) in chronic LBP populations. Further, these morphological (D'hooge et al., 2012) and neuromuscular (Butler et al, 2013; Hides, Richardson, & Jull, 1996; MacDonald, Moseley, & Hodges, 2009; MacDonald et al., 2010; Macdonald et. al, 2011; Hubley-Kozey et al., 2014) changes have been shown to be present in episodic LBP individuals during symptom remission as well as in individuals deemed recovered from a LBI.

It has been suggested that the spinal column has two important functions: structural and transducer (Panjabi, 2006). The passive subsystem is partially responsible for both roles including generation of passive stiffness (structural) as previously discussed as well as providing the sensory information needed for proper neuromuscular control (transducer) via mechanoreceptors present in the spinal column ligaments, facet capsules, and intervertebral disc annulus (Panjabi, 2006). It has been hypothesized that any disruption of these mechanoreceptors positioned in the passive subsystem, such as spinal column degeneration and injury, facet joint injury, changes in the

structure and material of the endplates (schmoral nodes) and intervertebral discs could lead to corruption of transducer signals required for optimal spinal neuromuscular control (Panjabi, 2006). Predisposing traumas, whether a single traumatic event or the result of repetitive motions over a long duration may lead to subfailure ligamentous injuries (stretching the tissue beyond its physiological limit but less than its failure point) and subsequent mechanoreceptor injury initiating the cascade of spinal dysfunction (Panjabi, 2006). Corrupted muscle responses resulting from corrupted afferent information from the mechanoreceptors may result in high stresses and strains in the spinal components of the passive subsystem and over time result in spinal inflammation and subsequent LBP (Panjabi, 2006).

The size of the intervertebral neutral zone (NZ) has been suggested to be an important determinant of spinal stability and optimal spinal function. As previously discussed, the NZ is the range of physiological intervertebral motion, measured from the neutral spinal position, within which spinal motion is produced with minimal internal resistance (Panjabi, 1992b). It is a region of high flexibility or laxity around the neutral position whereby minimal resistance is offered by the passive spinal column. The NZ appears to be a clinically important measure of spinal stability that has been documented to increase in size as a result of spinal column injury (Panjabi, 1992b). Specifically, structural changes of the intervertebral disc (IVD), most commonly degenerative in nature, has been considered one of the most important contributors to lumbar spine instability (Fujiwara et al., 2000). Disc degeneration develops early in life and histological studies of age-related changes in the IVD have revealed reduced endplate blood supply resulting in the breakdown of the nucleus pulposus (inner gel-like core of the IVD) by the second decade of life (Lorio, Jakoi, & Singla, 2010; Boos et al., 2002). Degeneration of the IVD space results in a reduction of the normal disc height and subsequent abnormal transmission of forces across

adjacent vertebrae resulting in an abnormal distribution of common physiological spinal loads (Adams & Hutton, 1983). Two main features of IVD degeneration have been associated with the development of lumbar spine instability (Fujiwara et al., 2000). Specifically, a reduction in IVD height and volume resulting from reduced water and proteoglycan content has been associated with the generation of slackness in the associated intervertebral ligaments (Horst & Brinkmann, 1981). In addition, vertebral endplate damage (a common sequalae of degenerative changes to the spinal column) has been associated with intradiscal pressure changes resulting in a further reduction in disc height as well as creating slack in the annulus fibrosis (collagenous outer layer of the IVD) (Kettler et al., 2011, Rijsbergen et al., 2017). Together, the generation of slackness in associated spinal ligaments and the IVD annulus result in a larger NZ as well as associated changes to the normal stress-strain relationship of the healthy IVD.

Morphological alterations affecting the active subsystem have been documented in LBP populations including changes in muscle fiber type and size. These back muscle structural alterations can lead to decreased strength and fatigue resistance, two impairments that have been linked to recurrent LBP (Lariviere, Arsenault, Gravel, Gagnon, & Loisel, 2003). Paraspinal muscles differ from most other skeletal muscles due to their predominance of relatively large type I (slow-twitch oxidative) fibers (Jorgensen, 1997; Mannion et al, 1997; Ng, Kippers, & Richardson, 1998a), which benefit their function as postural muscles (Mannion et al, 1997). Mannion et al. demonstrated changes in paraspinal muscle fiber type associated with LBP in which LBP patients were shown to have a significantly higher ($p<0.05$) portion of type IIX (fast twitch glycolytic) fibers, at the expense of type I fibers, compared with the muscles of matched-control subjects (Mannion, 1999). This is consistent with the lower paraspinal endurance noted in LBP populations (Suzuki & Endo, 1983; Mannion et al., 1997). Conversion of type I to type II fibers

has been associated with physical deconditioning or immobility (Haggmark, Eriksson, & Jansson, 1986) and these changes have been correlated with symptom duration, so that the longer duration of pain, the more glycolytic the paraspinal fiber composition supporting a role for physical deconditioning (Mannion et al., 2000). Regardless of the origin, whether it is in response to a LBP episode or a predisposition to developing LBP, a conversion of type I fibers to type II fibers would invariably lead to a compromise in paraspinal fatigue resistance. Mannion et al. also reported a more frequent appearance of non-specific pathological changes in fibers (such as moth-eaten or coretargetoid fibers) in the muscles of LBP patients supporting similar reports by Zhao and Yoshihara and colleagues (Mannion et al., 1997; Yoshihara, Shirai, Nakayama, & Uesaka, 2001; Zhao, Kawaguchi, Matsui, Kanamori, & Kimura, 2000). Suggested mechanisms responsible for these non-specific pathological changes include denervation, ischemia, and altered muscle use due to pain (Mannion et al., 1997; Yoshihara et al., 2001). Consequently, the degree to which the fiber structure is changed may be dependent upon the age of the subjects investigated, the duration of LBP symptoms, the level of paraspinal conditioning prior to the onset of LBP, and the extent of immobilization or inactivity (Mannion et al., 1997). Several studies have also documented paraspinal MT muscle wasting in LBP populations (Mattila et al., 1986; Hides, Stokes, Saide, Jull, & Copper, 1994; Danneels, Vanderstraeten, Cambier, Witvrouw, & De Cuyper, 2000).

The MT muscle is strongly developed in the lumbar region and plays an important role in stabilization of this area of the spine (Wilke et al., 1995; Hides, Gilmore, Stanton, & Bohlscheid, 2008). Changes in the morphological characteristics of the MT muscle resulting from low back injury could have important consequences on its ability to generate active stiffness. Many studies investigating morphological changes in the MT have examined subjects with nerve root impairments resulting from lumbar disc injuries (Suzuki & Endo, 1983; Mattila et al., 1986; Zhu

et al., 1989; Yoshihara, 2001). However, Hides and colleagues demonstrated a localized (isolated vertebral level) decrease in cross sectional area (CSA) of the MT on the symptomatic side of subjects suffering from their first episode of unilateral subacute LBP (<3 months duration) using diagnostic ultrasound (Hides et al., 1996). Similarly, localized MT atrophy has been shown among chronic (>3 months duration) LBP populations (Danneels et al., 2000; Hides et al., 2008). Results from a previous investigation comparing MT size and symmetry among chronic LBP and healthy asymptomatic subjects demonstrated significantly smaller (p= 0.001) MT CSA at L4 and L5 in subjects with unilateral chronic LBP (Hides et al., 2008). The stabilizing role of the lumbar MT at the segmental level serves to maintain the spine in a neutral position (normal lumbar lordosis) controlling intersegmental motion throughout the entire range of spinal motion (Wilke et al., 1995; O'Sullivan, 2000; McGill, 2007). The ability of MT to contribute to segmental stability has been confirmed in an in vivo investigation into the architectural design of the MT which confirmed a high CSA and a low fiber length-to-muscle length ratio demonstrating an ability to generate large stabilizing forces (Ward et al., 2009). Further, absence of spontaneous recovery of the MT muscle after remission of symptoms from a LBI has been shown based on a persistent decrease in muscle CSA (Hides et al., 1996). For these reasons, documented cases of morphological tissue changes and atrophy of the MT could be expected to have direct implications on lumbar spine stability. The deep fibers of the MT have also been shown to be less active during predictable and unpredictable loading in subjects who experience episodic LBP compared to healthy controls despite the remission of symptoms (MacDonald et al., 2010). In general, alternative neuromuscular control strategies have been observed in people with LBP including decreased activation of deep local back muscles and increased activation of the more superficial global back muscles in people with low back pain compared to healthy controls during trunk movements (Van

Dieen et al., 2003). This proposed adapted control strategy appears to limit tensile forces and motion of injured or painful structures in the back while compromising segmental control or the ability to 'fine-tune' segmental motion during spinal perturbations (Wilke et al., 1995; Van Dieen et al., 2003)

Changes in activation amplitudes and muscle activation patterns during expected and unexpected spinal perturbations as well as during a variety of dynamic tasks illustrate alterations in the neural control subsystem that have been linked to chronic LBP as well as recurrent LBP and individuals within the sub-acute phase of a LBI during symptom remission (Hodges & Richardson, 1999; O'Sullivan et al., 1997; Newcomer, Laskowski, Yu, Larson, & An, 2000a; Radebold et al., 2000; Hubley-Kozey & Vezina, 2002; van Dieen et al., 2003; Lee, Cholewicki, Reeves, Zazulak, & Mysliwiec, 2010; MacDonlad et al., 2011; Moreside et al., 2014). A previous investigation showing an association between proprioception deficits in multiple planes of motion and low back injuries ($p<0.05$) has resulted in subsequent investigations into neuromuscular control dysfunction in LBP populations associated with proprioceptive deficits (Parkhurst & Burnett, 1994). The association between LBP and proprioceptive deficits has been investigated by comparing postural sway in various positions (standing, seated and single leg stance) and conditions (eyes open vs. eyes closed) (Mientjes & Frank, 1999) as well as with movement based assessments such as motion perception thresholds (Lee et al., 2010), reproduction of submaximal isometric trunk muscle exertions (Descarreaux et al., 2004) and repositioning error (Newcomer et al., 2000; O'Sullivan et al., 2003, Lee et al., 2010). Lee and colleagues investigated the association between proprioception impairments and LBP in various positions (seated, supine and side-lying) using both motion perception threshold and repositioning tests (Lee et al., 2010). The results of the investigation showed a significantly smaller motion perception threshold in the healthy control group compared

to the LBP group (0.8±0.6° vs. 1.3±0.9°) ($p<0.01$), suggesting the healthy controls were more perceptive of trunk motion (Lee et al., 2010). Conversely, the researchers failed to show a significant difference between LBP and healthy control groups in the repositioning tasks. These results differ from a previous investigation in which repositioning error was found to be significantly greater in subjects with lumbar segmental instability compared to asymptomatic subjects matched for age, weight and height ($p=0.02$) (O'Sullivan, 2003). The results of a pilot study by Newcomer et al. examining repositioning error in LBP populations demonstrated an insignificant difference between individuals with LBP and asymptomatic controls (Newcomer et al., 2000a). However, the authors performed a similar experiment with subjects in a supported standing position (subjects stood with unrestricted pelvis and lower extremities in the pilot investigation) and found a significant higher reposition error in LBP subjects compared to control subjects in flexion ($p=0.036$) (Newcomer, Laskowski, Yu, Johnson, & An, 2000b). The conflicting findings in all of these experiments may be explained by heterogeneous LBP populations as well as variation in methodologies such as testing positions and planes of motion tested. In addition to deficits in proprioception, alterations in preparatory and reflex muscle responses have been demonstrated during expected and unexpected perturbations in LBP and LBI populations.

Feedforward control (anticipatory postural adjustment) results in activation of trunk muscles prior to a known perturbation in order to counterbalance the pending displacement of the body center of gravity and disturbance to trunk equilibrium. The onset and duration of activation of each trunk muscle is critical for optimal spinal function during dynamic tasks as together they minimize the displacement of center of gravity and prevent excessive and potential harmful spinal loads (Metha, Cannella, Smith, & Silfies, 2010). Reactive or feedback strategies employed by the

neural control subsystem occur after spinal disturbances and benefit from input of sensory information to the system triggering corrective automatic muscle responses (Silfies, Mehta, Smith, & Karduna, 2009; Mehta et al., 2010). Selective trunk muscles (TrA, IO and MT) have been shown to act in a feedforward manner with activity onset occurring prior to or in conjunction with an upper extremity self-perturbation (Hodges & Richardson, 1999; Moseley et al., 2002). Theoretically, feedforward activation of these local muscles contributes to control of spinal segmental motion and establishes a stable base for contraction of the larger trunk and upper limb musculature while also dampening moments created by the perturbation. Upper as well as lower extremity self-perturbation tasks have been used to examine differences in the response of trunk muscles to changing external moments in subjects with and without chronic LBP (Hodges & Richardson, 1996; Hodges, 1999; Hodges, 2001; Hubley-Kozey & Vezina, 2002). Investigations have demonstrated a lack of TrA and IO feedforward activation in subjects with chronic LBP regardless of upper limb movement speed (Hodges & Richardson, 1999) or direction (Hodges, 1996). Further, in a comparison between two mechanical LBP subgroups, Silfies et al. showed a significant later onset of the lumbar MT ($p=.005$) and ES ($p=.001$) as well as a significantly less number of trunk extensors acting in a feedforward manner ($p=.017$) during an upper extremity self-perturbation task in an instability subgroup compared to a noninstability subgroup of mechanical LBP (Silfies et al., 2009). Reduction in feedforward activation of the deep fibers of MT has also been documented on the previously painful ($p= .042$) and the non-painful ($p<.033$) sides in subjects during symptom remission from episodic LBP compared to healthy controls during predictable upper limb loading (MacDonald et al., 2010). During an investigation comparing temporal EMG waveforms between those with chronic LBP and healthy controls while performing a bilateral leg lifting task shown to challenge spinal stability, Hubley-Kozey and

Vezina also demonstrated impaired feed-forward responses (EO and LRA) as well as a lack of coordination among abdominal and extensor muscles for LBP profiles (Hubley-Kozey & Vezina, 2002). Collectively, these studies demonstrate an impaired feed-forward mechanism prior to a known perturbation suggesting that inappropriate preparatory muscle responses may be a component of or an initiating factor in suboptimal spinal function and subsequent lumbar instability.

It has been well established that individuals with LBP exhibit delayed trunk muscle reflex responses compared to healthy controls following sudden loads (Cholewicki et al., 2005; Radebold et al., 2000; Reeves, Cholewicki, & Milner, 2005). Additionally, sudden loading incidents such as slips and trips as well as excessive spinal movements (flexion and rotation) while lifting have been linked to LBI (Frymoyer et al., 1983; Manning et al., 1984). In an experiment using a quick-release method in four directions of isometric trunk exertions, Radebold et al. showed significant greater latencies (reaction times) for agonistic muscles shutting off (70msec) and antagonistic muscles switching on (83msec) in LBP subjects compared to sex and age matched healthy controls ($p<0.01$) (Radebold et al., 2000). The healthy controls turned their agonistic muscles off before activating their antagonistic muscles compared to the LBP group who demonstrated a pattern of co-activation with agonist remaining active (3.4 out of 6 muscles switched off) while antagonists switched on (5.3 out of 6 muscles). Co-activation of agonistic and antagonistic as well as local and global muscles has been previously shown to stiffen and subsequently enhance spinal stability (Bergmark, 1989; Cholewicki & McGill 1996; Gardner & Stokes, 1998). Van dieen et al. also observed increased levels of co-activation in subjects with LBP and concluded that this increased co-activation strategy may be used to compensate for other neuromuscular control deficiencies such as impaired reflex dynamics (long reflex delays and reduced reflex amplitudes) (Van dieen

et al., 2003). Perhaps more significant, increased agonist-antagonist co-activation has been documented during highly controlled dynamic tasks in various LBP populations. Additionally, these findings have been confirmed in individuals deemed recovered from a LBI (Butler et al., 2013; Hubley-Kozey et al., 2014; Moreside et al., 2014). While a co-activation strategy may be a reasonable temporary solution to prevent further tissue aggravation and pain, a long-term co-activation strategy may have consequences related to increased compressive loading and reduced spinal motion (Butler et al., 2013).

Delayed reflex responses in subjects with LBP could be interpreted as either a compensatory mechanism secondary to previous osteoligamentous damage or as a predisposing factor for developing a LBI. Recent evidence supports flexor-extensor co-activation as a precursor for the development of LBP (Nelson-Wong & Callahan, 2010). Following a 2-year follow-up prospective study, Cholewicki et al. (2005) concluded that delayed muscle reflex latencies in response to a quick force release significantly increases the odds of sustaining a low back injury ($p=.042$). The researchers showed an average of 14msec longer latency in those who sustained a low back injury in comparison to those who did not sustain a low back injury (Cholewicki et al., 2005). It has been hypothesized that such low back injuries may result from an overreaction of the neural control subsystem (excessive generation of trunk muscle forces to stabilize the spine) leading to damaging compressive and shear forces placed on the spine (Radebold et al., 2000) resulting in mechanical derangement of osteoligamentous structures and a subsequent decrease in passive stiffness (Cholewicki et al., 2005; Radebold et al., 2000). Regardless of mechanism, these impairments in neuromuscular control may compromise spinal stability and optimal spinal function rendering a person vulnerable to acquire a LBI or exacerbate an existing LBI in the case of recurrent LBP.

2.3 Diagnosis of Spinal Instability

Although lumbar spine instability has gained notoriety as a subgroup of LBP over the past two decades, there is still a lack of reliable and valid diagnostic procedures used to identify spinal instability. The diagnostic standard for lumbar spine instability has traditionally been excessive sagittal translation (>4.5mm or greater than 15% of the vertebral body width) or rotational movement between lumbar vertebrae on lateral flexion and extension radiographs (White & Panjabi, 1990; Fritz, 1998). However, establishing standardized radiographic diagnostic criteria has been complicated by high false-positive rates (Hayes, Howard, Gruel, & Kopta, 1989) and high variation between asymptomatic individuals (Hicks et al., 2003). Further, the use of flexion and extension radiographic findings has been criticized as traditional radiographs have been assessed statically at end-range motion while neglecting the neuromuscular control demands of dynamic and mid range motion (within the "neutral zone") where the spinal dysfunction has been theorized to occur (Hicks et al., 2003; Cook, Brismee, & Sizer, 2006; Demoulin et al., 2007; Teyhen et al., 2007). Generally speaking, it is plausible that alterations in neuromuscular control (increased active subsystem contribution) resulting from deficiencies in the passive subsystem may be undetected in a static measure such as end-range static radiographs. A previous digital fluoroscopic video (DFV) investigation of sagittal-plane flexion and extension (return to upright posture) demonstrated aberrant segmental motion during mid-range postures in a sub-group of LBP subjects (clinical instability as defined by Hicks et al., 2005) compared to age and body mass index matched controls (Teyhen et al., 2007). The authors concluded that disruptions in the sequence and timing of how the segmental motion occurred in subjects with LBP could be viewed as alterations in neuromuscular control of segmental motion (Teyhen et al., 2007). Consequently, lumbar spine instability has been previously classified into two categories, (1) radiological lumbar

instability and (2) clinical or functional lumbar instability (Cook et al., 2006; Demoulin et al., 2007). The former reflects marked disruption of the passive osteoligamentous constraints leading to joint laxity detectable on radiographic examination while the latter commonly demonstrates subtle quantifiable clinical features in the absence of findings during traditional radiographic analysis (Demoulin et al., 2007).

Regardless of the type of lumbar spinal instability, this subclassification of LBP remains controversial because the pathomechanical consequences of lumbar spine instability are poorly defined and there is minimal evidence to link these consequences with severity of clinical signs and symptoms (Dupuis et al., 1985; Paris, 1985; Panjabi, 1992a; Fritz et al., 1998). Further, there is no confirmed true reference standard for confirmation of lumbar spine instability and so much of the current research has been conducted on the predictive validity of clinical measures to identify LBP populations that will respond favorably to spinal stabilization exercises (Hicks et al., 2005; Fritz, Whitman, & Childs, 2005b; Teyhen et al., 2007; Kumar, 2011; Robin et al., 2014). Studies examining the validity of clinical measures used to diagnose lumbar spine instability are still elusive as a result of these diagnostic pitfalls. Despite this lack of evidence, several researchers have suggested common subjective and objective clinical findings within this subgroup of LBP (Maigne, Lapeyre, Morvan, & Chatellier, 1976; Paris, 1985; O'Sullivan, 2000; Hicks et al., 2003; Cooks et al., 2006; Demoulin et al., 2007).

Many common subjective and objective clinical descriptors for lumbar spine instability have been suggested based upon clinical findings elicited during active trunk flexion and return from a flexed position, a movement commonly associated with symptoms in people considered to have lumbar instability (Dilitto et al., 1995; O'Sullivan, 2000; Hicks et al., 2003; Cook et al, 2006; Demoulin et al, 2007; Alqarni et al., 2011). Researchers have described subjective reports of a

painful arc during return from a forward flexed position (Kirkaldy-Willis & Farfan, 1982; O'Sullivan, 2000; Hicks et al., 2003; Cook et al, 2006; Demoulin et al, 2007; Alqarni et al., 2011) as well as patient's inability to return from such a position without using their hands to climb their thighs (Gower sign) (O'Sullivan, 2000; Hicks et al; 2003; Demoulin et al, 2007). A painful arc can be defined as pain throughout a percentage of an entire range of motion. For an example, on return from a forward flexed position onset of pain may occur at 90° of lumbar flexion and then dissipate at 60° at which time the remaining range of motion would be pain free. Further, changes in pain intensity upon transitional movements such as from standing to sitting and from sitting to standing have been reported (Maigne et al., 1976; Paris, 1985; Cook et al., 2006). Common subjective reports of "giving way", "locking", or sensations of "slipping out" during normal demands of spinal mobility (Kirkaldy-Willis & Farfan, 1982; O'Sullivan, 2000, Cooket al., 2006) are fitting with documented frequent episodes of LBP resulting from minimal spinal perturbations as well as favorable outcomes with temporary bracing (Kirkaldy-Willis & Farfan, 1982; Dilitto et al., 1995). Reports of a necessity to "twist the back into position" or self manipulate (Paris, 1985, Cook et al, 2006) are supported by the documented short-term symptom relief following spinal manipulative therapy (SMT) in subjects suspected of lumbar spine instability (Dilitto et al, 1995). Conversely, there have also been reports of poor outcomes with SMT and mobilization-based treatments (Kirkaldy-Willis & Farfan, 1982; O'Sullivan, 2000). Researchers have also demonstrated that a combined SMT and lumbar spine stabilization exercise approach may be more effective than exercise alone to manage LBP (Childs et al., 2004). These conflicting results illustrate the challenge of tailoring appropriate treatment regimes as well as developing reliable and valid CPR with respect to patients suspected of having lumbar spine instability.

Cook et al. used a Delphi survey instrument to determine if consensual, specific subjective and objective identifiers for lumbar instability could be recognized from surveying 168 physical therapists identified as Orthopaedic Clinical Specialists. Feelings of "giving way" or back "giving out" were documented as the subjective factor that is most related to lumbar spine instability followed by self-manipulation and frequent bouts or episodes of LBP (Cook et al., 2006). A previous investigation examining the interrater reliability of clinical examination measures for the identification of lumbar spine instability showed significant agreement (κ = .69 and κ =.61) in the subjective findings of a painful arch in flexion and return from flexion respectively (Hicks et al., 2003). Alternatively, the same investigation demonstrated poor to fair interrater reliability (κ = 0.25) for Gower sign (Hicks et al., 2003). Objective clinical descriptors have also been presented based on observations during sagittal-plane flexion and extension in individuals suspected of lumbar spine instability (Dilitto et al., 1995; O'Sullivan, 2000; Hicks et al., 2003; Cook et al, 2006; Demoulin et al, 2007; Alqarni et al., 2011).

Examiners have described common aberrant movement patterns during active trunk flexion among patients diagnosed with lumbar spinal instability including an "instability catch" (O'Sullivan, 2000; Hicks et al., 2003; Cook et al., 2006; Demoulin et al, 2007; Alqarni et al., 2011), a reversal of lumbopelvic rhythm (Hicks et al., 2003; Demoulin et al, 2007) and Gower sign (O'Sullivan, 2000; Hicks et al; Demoulin et al, 2007). An instability catch has been defined as any sudden acceleration or deceleration of trunk movement or movement occurring outside the primary plane of motion such as lateral bending or rotation during active trunk flexion (Hicks et al., 2003). In addition, a reversal of lumbopelvic rhythm is noted upon returning from a forward flexed position when the subject bends the knees and shifts the pelvis anteriorly before returning back to an upright neutral position (Hicks et al., 2003). Hicks and colleagues during the same

study investigating interrater reliability, demonstrated poor to fair reliability for objective observations associated with active trunk flexion including Gower sign ($\kappa = 0.00$) and an instability catch ($\kappa = 0.25$) (Hicks et al., 2003). However, low reliabilities were associated with low prevalence of these observations in the sample and following the grouping of all observations into a single category ("aberrant movement during trunk flexion") the reliability increased to $\kappa = 0.60$ (Hicks et al., 2003). Detection of specific aberrant movement patterns during active trunk range of motion can be arbitrary and judgements are made based upon clinical anecdotal evidence requiring significant clinical experience and level of expertise. In addition to aberrant movement patterns during trunk range of motion and subjective signs associated with lumbar spinal instability, the diagnosis of LBP resulting from spinal instability has been confirmed with supporting evidence during assessment of spinal intersegmental motion.

The assessment of passive intervertebral or intersegmental mobility has been used to clinically identify hypermobile (excessive intervertebral motion) or hypomobile (restricted intervertebral motion) lumbar segments in comparison to adjacent segments. Assessments of intervertebral mobility aid in the clinical decision-making process for proper management strategies such as spinal mobilization (hypomobile segments) or exercise induced segmental stabilization (hypermobile segments). Passive accessory intervertebral motion tests (PAIVMs) have been included in the diagnostic cluster of clinical findings associated with lumbar spine instability (Abbott, 2005; Fritz et al., 2005b; Cook et al., 2006). PAIVMs are performed by directing a firm and gradual PA (posterior to anterior) force application into the spinus process of each lumbar segment in order to categorize passive intersegmental motion (relative to the segments above and below) as being normal, hypomoblie, or hypermoblie. The passive structures of the spine resist the PA forces applied by the examiner during PAVIMs with appropriate passive

stiffness. Reduced stiffness may be indicative of segmental instability whereas increased stiffness may be suggestive of segmental hypomobility (Kumar, 2011). A secondary purpose of PAIVMs, and perhaps a more reliable application, is identification of symptomatic segments or segments that reproduce similar LBP as experienced in previous LBP episodes (Hicks et al, 2003; Fritz et al., 2005b). Previous investigations examining intersegmental mobility using PAIVMs have shown poor to fair (ICC range .03-.37) interrater reliability (Maher & Adams, 1994; Binkley, Stratford, & Gill, 1995; Hicks et al., 2003). In contrast, the interrater reliability for pain provocation during PAIVMs appears to be higher (Maher & Adams, 1994; Hicks et al., 2003). Hicks et al. proposed difficulty in identifying the segment level as an explanation for poor reliability of segmental mobility testing. The authors attempted to address this problem by defining the presence of at least one hypermoblie lumbar segment as an indicator for lumbar spine instability (Hicks et al., 2003). While examining the relationship between clinical variables and the presence of radiographic instability, Fritz et al. showed a positive likelihood ratio of 9 for a lack of hypomobility with intervertebral motion testing as the best individual clinical test for instability (Fritz et al., 2005b). In the absence of hypomobile segments, identification of precise levels of excessive intersegmental mobility (hypermobility) may not be as important considering the global effect of lumbar stabilization exercises.

Special clinical tests, or tests specifically designed for the diagnosis of lumbar spine instability have also been included in the diagnostic sequelae of lumbar spine instability (Dilitto et al., 1995; Magee, 1997; Kasai et al., 2006). The posterior shear test, originally described by Delitto, involves a segmental PA force application throughout each lumbar segment with the patient in a standing upright position (Delitto et al., 1995). The test is considered positive upon generation of LBP symptoms similar to previous LBP experiences during the PA force application

(Delittio et al, 1995). In addition, the passive lumbar extension test, developed by Kasai and colleagues is performed with the patient in the prone position while the examiner elevates both lower extremities about 30cm from the table while maintaining full knee extension with gentle traction (Kasai et al., 2006). The test is considered positive upon generation of LBP symptoms similar to previous LBP experiences during elevation of the lower extremities followed by a reduction of LBP symptoms after the lower extremities have been returned to the starting position (Kasai et al., 2006). Both clinical tests have been developed specifically to induce symptom provocation resulting from excessive intersegmental motion following an induced external spinal load. The posterior shear test has been shown to have low sensitivity (57%) and specificity (48%) (Fritz et al., 2005a) as well as poor inter-rater reliability (κ=.22) (Hicks et al, 2003). The only investigation examining the passive lumbar extension test is the original publication of the test in which the investigators compared the clinical measurement to flexion and extension radiographic findings and demonstrated a high sensitivity (84.2%) and specificity (90.4%) as well as a positive likelihood ratio of 8.84.

Most clinical measurements of lumbar spine instability are finite (positive or negative) requiring a highly experienced assessment and have not been substantiated by simultaneous diagnostic measurement (Hicks et al., 2003; Hicks et al., 2005). Presently, there is no validated bio-physical measure of lumbar spine instability. Investigative studies have compared clinical measures of lumbar spine instability to flexion and extension radiographic findings which have also been criticized for reasons previously discussed. Both clinical and radiographic findings are not specific and therefore, the diagnosis of lumbar spine instability should be based on the understanding of theoretical and modeling framework of spinal stability, relevant radiographic findings and the link between these and clinical history and examination findings.

2.4 The Prone Instability Test

The PIT is a special clinical test used to assess lumbar spine stability and is arguably the most reliable (Hicks et al., 2003; Fritz et al., 2005a; Schnieder et al., 2008) and practical (Hicks et al. 2003) clinical measure of lumbar spine instability. The test, originally published by Magee, is comprised of two progressive interdependent components (passive and active) that together challenge all three subsystems (passive, active, and neural control) of the spinal stabilizing system (Magee, 1997). The passive component is conducted with the patient in the prone position on an examination table with the legs over the edge of the table and the feet resting on the floor (Figure 2.1a). While resting in this position, the examiner applies a posterior-anterior (PA) force directly into the spinus process of each successive lumbar segment (L1-L5) using the hypothenar eminence. Upon application of force, the patient reports any provocation of pain similar to that experienced during previous LBP episodes (familiar pain). Following the passive component of the PIT, the patient then lifts both legs off the floor (table holding is permitted to maintain position) and the examiner reapplies a similar PA force into provocative segments identified during the passive component of the test (Figure 2.1b). This constitutes the active component of the PIT. The test is considered positive if familiar pain is present during force application in the resting position (passive component) but subsides during the bilateral leg lifting task (active component) (Hicks et al, 2003; Hicks et al., 2005).

(A) Passive component **(B) Active component**

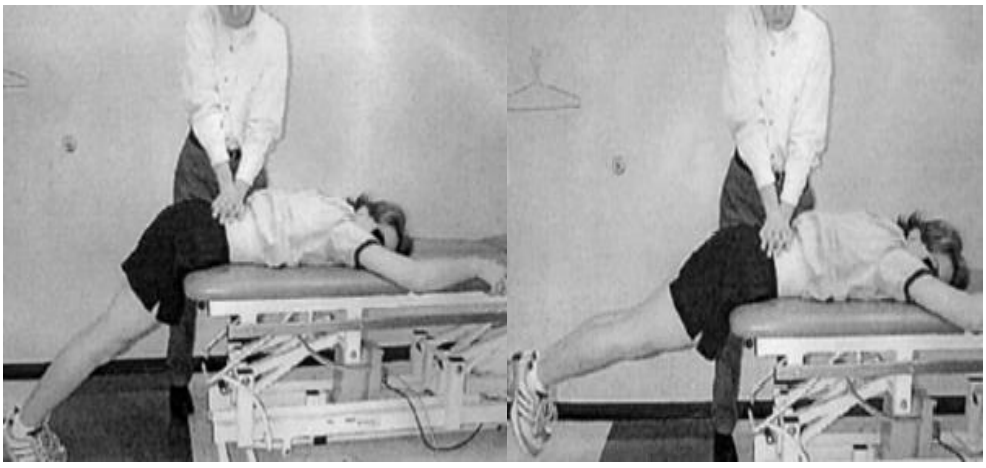


Figure 2.1: Prone Instability Test: active and passive components (Hicks et al, 2003)

The theoretical framework supporting the PIT is largely based upon Panjabi's model of segmental stability including the passive, active, and neural control subsystems. During the passive component of the PIT the subject provides passive resistance (no muscle activity) to an external spinal perturbation applied by the examiner. A major assumption during the passive component of the PIT is that the spinal perturbation is unaffected by trunk muscle activity and is therefore, assessing the passive subsystem. Theoretically, positive responders are differentiated from negative responders based on the onset of low back discomfort resulting from excessive posterior-anterior intersegmental motion (shear motion) due to a decrease in passive spinal stiffness. Identification of dysfunctional segments using PAIVMs based on pain provocation has been shown to be reliable (Maher & Adams, 1994; Hicks et al., 2003) compared to using PAVIMs to classify intersegmental mobility as being hypermoblie in relation to segments above and below (Maher & Adams, 1994; Binkley et al., 1995; Hicks et al., 2003). The passive subsystem, as previously defined, represents spinal stiffness generated by joint structures such as bones,

ligaments, joint capsules and intervertebral discs. Osteoligamentous injury may result in decreased passive stiffness and subsequent pain provocation during the passive component of the PIT.

Following the passive component of the PIT the subject completes the active component during a bilateral leg lifting task promoting activation of the trunk extensors. Positive responders are differentiated from negative responders based on the reduction or absence of pain during a similar spinal perturbation following the bilateral leg lift task. Theoretically, the active subsystem can modulate joint stiffness through generation of trunk muscle activity creating tension which stiffens or splints the symptomatic spinal segment identified during the passive component of the PIT. The neural control subsystem, through direct or indirect innervations with motor neurons, coordinates the level of tension of various muscle fibers and muscle groups required to stiffen the symptomatic segment. Specifically, anatomical distributions of the lumbar components of the iliocostalis and longissimus as well the multifidus support their role as direct contributors to this splinting effect. The line of action of the iliocostalis and longissimus is oriented in a posterior and caudal direction allowing them to generate posterior shear forces together with extensor moment on the superior vertebrae relative to the vertebrae below. Theoretically, the posterior shear forces generated during the bilateral leg lifting task support any anterior reaction forces that are produced during the PA force application resulting in decreased inter-segmental mobility and a reduction in pain provocation. As previously described, the lumbar portion of the MT has been divided into superficial and deep fibers. The MT line of action is parallel to the compressive axis of the spine or in certain cases runs anteriorly and caudal in an oblique direction. Since the MT fibers span between two and four segments their forces act only locally and therefore provide moment support at specific joints enhancing segmental stability (McGill, 2007). Contributions from both the superficial and deep fibers are required for optimal inter-segmental function. The superficial fibers

have been shown to produce sufficient torque to create posterior sagittal rotation (extension) of the lumbar spine in combination with intervertebral compression. Combined with deep fiber generation of compressive forces with minimal associated torque, contributions from both sections of the MT enhance active stiffness (Macintosh et al., 1986, Bogduk et al., 1992) during the active component of the PIT. In a study by Hebert et al, investigators demonstrated a significant relationship between a positive PIT response and lumbar MT activation levels (Herbert et al., 2010). Using diagnostic ultrasound, they showed positive PIT responders to have lower submaximal MT activation (8.51% MVIC) compared to negative PIT responders (14.99%) during a contralateral weighted arm exercise ($p=0.018$).

Researchers investigating clinical measures of lumbar spine instability have consistently shown the PIT to have moderate to good inter-rater reliability (Hicks et al., 2003; Fritz et al., 2005a; Schnieder et al., 2008). The PIT was shown to have good reliability ($\kappa = 0.87$) in a study investigating the interrater reliability of clinical examination measures for identification of lumbar segmental instability (Hicks et al, 2003). This study also supported the generalizability of the PIT to a wide range of clinicians with varying degrees of experience as the reliability coefficient was high with relatively narrow confidence intervals (.80-.94) (Hicks et al, 2003). Similarly, Schneider et al. and Fritz et al. demonstrated moderate inter-rater reliability ($\kappa = 0.54$ and $\kappa = 0.69$, respectively) for the PIT while examining clinical measures for lumbar spine instability (Fritz et al, 2005a; Schnieder et al., 2008). During the same investigation, Fritz et al. failed to show a significant univariate relationship between the PIT and radiographic instability (Fritz et al., 2005a). However, the authors also failed to show a significant relationship between radiological instability and measures that have been previously determined reliable including aberrant movements during

trunk range of motion and pain provocation during inter-segmental motion testing (Maher & Adams, 1994; Hicks, 2003; Schneider et al, 2008).

In absence of a true diagnostic standard for identifying lumbar spine instability the link between LBP and lumbar instability is often confirmed upon a favorable response to lumbar stabilization exercises. Clinical prediction rules (CPR) (Hicks et al, 2005; Rabin et al., 2014) and treatment based classification systems (Delitto et al., 1995; Fritz, Cleland, & Childs, 2007) have been developed to identify LBP populations that will most likely benefit from lumbar stabilization programs. The PIT has been included in CPR as well as classification systems indicative of those who will respond favorably to lumbar stabilization exercises. In a study by Hicks et al., investigators established a preliminary CPR for determining which individuals with LBP would respond to a stabilization exercise program (Hicks et al., 2005). Four predictors of success with stabilization were included in the multivariate CPR (age<40, average straight leg raise > 91°, aberrant movement present, positive PIT) of which the presence of 3 or more of the 4 variables resulted in a positive likelihood ratio (LR) of 4 (Hicks et al, 2005). Further, a positive PIT and the presence of aberrant movement were the only significant univariate predictors of lumbar stabilization success ($p = .034$ and .05). This finding was supported in a validation study by Rabin et al. who used a modified version of the CPR (mCPR), containing only a positive PIT and presence of aberrant movement as variables for predictors of exercise success, to show subjects receiving lumbar stabilization exercises with a positive mCPR status experienced significant less disability ($p = 0.02$) following an 8-week treatment protocol compared to those with a negative mCPR status (Rabin et al., 2014). In addition to inclusion of the PIT in several CPR for identifying those with LBP who will respond favorably to exercise prescription, the special test has also been included in treatment-based LBP classification systems (Delitto et al., 1995; Fritz et al., 2007).

Fritz et al. combined the PIT with a battery of other special tests identified by Stuge and Colleagues (posterior pelvic pain provocation test, active straight leg raise test, provocation of long dorsal sacraliliac ligament, provocation of the pubic symphysis with palpation, and the modified Trendelenburg test) (Stuge et al., 2004) while developing a set of special tests to identify a homologous LBP population requiring lumbar stabilization exercises as part of a successful treatment regime (stabilization classification) (Fritz et al., 2007). However, the tests identified by Stuge and colleagues were adapted for postpartum women experiencing posterior pelvic girdle pain and may not be generalizable to other LBP populations.

In comparison to other clinical measurements used to identify lumbar spine instability, previous investigations have shown the PIT to be reliable as well as predictive with respect to identifying individuals with LBP who respond favorably to lumbar stabilization exercises. It has been suggested that the next logical step with added knowledge of PIT test-retest reliability and predictive validity for lumbar stabilization success is to validate the clinical measure as being truly diagnostic of lumbar spine instability (Hicks et al., 2003).

2.5 Electromyography

EMG is a technique used to gain information about the electrical signals generated by a muscle during activation whereby voltage-measuring electrodes attached to the surface of the skin are used to measure the summation (spatial and temporal) of all motor unit action potentials generated across muscle fibers within the pick-up region of the electrode configuration (Soderberg, 1992; Staudenmann, Roeleveld, Stegeman, & van Dieen, 2010). This technique provides useful detail of the motor patterns used to recruit muscles that make mechanical contributions throughout specific tasks. The myoelectric signal is highly variable and dependent upon important properties

of the electrode-muscle tissue interface including electrode placement (with respect to location and fiber orientation of the target muscle), tissue characteristics and skin preparation procedures (Rutherford, Hubley-Kozey, & Stanish, 2011). When signals are processed to provide data to compare between trials (requiring reapplication of electrodes), subjects and muscle sites guidelines suggest some form of signal amplitude normalization be employed (Knutson, Soderberg, Basllantyne, & Clarke, 1994; Burden, 2010).

Amplitude normalization is accomplished by expressing absolute EMG values as a percentage of a physiologically relevant EMG value obtained during a calibration maximal or submaximal contraction. The most commonly used reference EMG value is the percent of maximum voluntary isometric contraction (%MVIC) whereby subjects are requested to exert a maximal effort during a specific isometric exercise shown to isolate a desired muscle group of experimental significance (Knutson et al, 1994; Vera-Garcia, Moreside, & McGill, 2009). The MVIC values are then used to normalize all experimental trial data. Normalization techniques have recently been the subject of a literature review that has established good to excellent test-retest reliability for MVIC normalization (Burden, 2010) supporting the conclusion from Knutson et al. stating that MVIC normalization should be the standard method of normalization in both healthy and orthopaedic populations (Knutson et al, 1994). The validity of using MVIC normalization has been debated by many researchers questioning the ability of individuals who are experiencing pain to produce MVIC amplitudes during normalization exercises. However, while evidence is still elusive in the LBP literature, an investigation into quadriceps femoris weakness and activation failure in subjects with symptomatic knee osteoarthritis (OA) failed to show a significant difference (p=0.233) in voluntary activation between the OA group and a group of age-matched controls (Lewek, Rudolph, & Mackler, 2004).

EMG amplitude and temporal characteristics captured during specific tasks can be used to assess trunk neuromuscular control. EMG amplitude measures have been shown to have good to excellent within and between-day reliability for LBP subjects with intraclass correlation (ICC) ranging from 0.65-0.98 (Dankaerts, O'Sullivan, Burnett, Straker, & Danneels, 2004; Lariviere et al., 2003). Normalized EMG activity level is a direct measure of how active a particular muscle is throughout a particular exertion. This measure is not an indicator of muscle tension, but merely a measure of the degree of muscle activation solicited from the muscle (Soderbergh, 1992). However, estimates of muscle force are frequently based on EMG measurements. The relationship between EMG and force has been studied extensively and many spinal investigations have concentrated on the trunk extensors. The trunk muscle EMG-Force relationship has more consistently been shown to be non-linear (Stokes, Hides, & Nassiri, 1997; Thelen, Schultz, Fassois, & Ashton-Miller, 1994) although linear relationships have also been demonstrated (Dolan & Adams, 1993; Anders, Brose, & Hofmann, 2008). Factors present during dynamic contractions complicate the EMG-muscle force relationship such as changes in muscle fiber length (Anders et al., 2004) and velocity (Solomonow, 2009). However, interpretations regarding the association between normalized EMG activity and muscular force output may be made with proper control of these factors (Brown & McGill, 2008). Recently, the effect of agonist-antagonist co-activation on the EMG-force relationship has been given consideration. It has been hypothesized that such co-activation may alter the perceived EMG-force relationship as the agonistic force production will be underestimated as a function of the comparable amount of antagonist co-activation (Brown & McGill, 2008). Brown and McGill reported that more linear relationships were found between trunk extensor activity and moments produced when accounting for the additional muscle force

generated by the antagonist muscle groups compared to a non-linear relationship when antagonist activity was ignored during isometric flexion and extension contractions (Brown & McGill, 2008).

Interpretation regarding the association between normalized EMG activity and muscular force output is also complicated by the signal-contaminating phenomenon known as crosstalk. Crosstalk is the result of myoelectric contribution to the captured EMG signal by muscles other than the muscle of interest. The quantity of myoelectric activity contribution from neighbouring muscles has been controversial with estimated values ranging from 5% and 15% (Deluca & Merletti, 1988; Solomonow et al., 1994). However, it has been suggested the effect of crosstalk may be negligible with proper EMG application, electrode size, and interelectrode distance by minimizing the pick-up volume of surface electrodes (Fuglevand, Winter, Patla, & Stashuk, 1992; Solomonow et al., 1994). Electrode placement maximizing distance from adjacent muscle boundaries may also minimize crosstalk and conformation through manual muscle testing may identify contributions from muscles that would not be active during the specific muscle test (Soderbergh, 1992).

As previously discussed, the theoretical basis supporting most clinical tests for spinal instability, including the PIT, resides in the test's ability to challenge a deficient passive subsystem resulting in some form of pain provocation. Additionally, most clinical indicators of poor neuromuscular control including subjective and objective clinical descriptors as well as documented aberrant movement patterns associated with spinal instability are often symptom based and lack usefulness in screening asymptomatic populations. Further, in the absence of a gold standard test from which to compare, clinical tests for spinal stability have lacked construct validity. Conversely, EMG is an objective, physiologically-relevant measure that has been shown to be able to accurately differentiate those with and without LBP (Geisser et al, 2005) as well as

differentiate between those individuals who have been deemed recovered from a LBI and healthy controls (MacDonald et al., 2010; MacDonald et al., 2011) in the absence of LBP symptoms.

Several EMG investigations have been conducted on individuals with no previous history of a LBI as well as recovered LBI individuals using a highly-controlled standardized dynamic transfer task with the intent of identifying physiological alterations in the trunk musculature as an objective marker of recovery as well as potentially adding insight to re-injury mechanisms (Butler et al., 2010; Hubley-Kozey et al., 2014). Collectively, these investigations have used this dynamic transfer task to demonstrate the ability of EMG measures to differentiate between individuals who had a LBI that were deemed recovered and individuals who did not experience a LBI (Butler et al., 2010; Hubley-Kozey et al., 2014). In addition, modest differences in EMG data collected during the transfer task were demonstrated between a group of individuals who sustained a LBI reoccurrence and a group who did not reinjure (Hubley-Kozey et al., 2014). In a more recent investigation of a similar recovered LBI group recruited from a military population, the dynamic transfer task was used to demonstrate minor differences in EMG motor patterns between those who tested positive and negative during the PIT (Trudel, 2014). The transfer task was specifically designed to analyse neuromuscular responses to changing external moments while minimizing the potential effects of trunk motion including changes in muscle length, velocity, and acceleration on these neuromuscular responses. Acceleration and trunk motion control through a standardized motion count (4 seconds from lift and replace), a mid-thoracic tactile feedback sensor, and quantitative motion check via a Flock of Birds (FOB) magnetic motion system have all been implemented during the transfer task to ensure minimal effects of trunk motion on neuromuscular responses (Butler et al., 2010). As a result, the transfer task provides a highly-controlled standardized task that is dynamic and can be used to assess neuromuscular responses to continually

changing flexion and lateral flexion moments on the trunk primarily created by external load and not truck motion. Further, these trunk motion control mechanisms as well as negligible contribution from axial moments (due to minimal acceleration forces) during the transfer task have been validated during a previous investigation comparing the neuromuscular responses between men and women (Hubley-Kozey, Butler, & Kozey, 2012).

In keeping with the modeling evidence that all trunk muscles are important for spinal stability and function (McGill, 2003; Cholewicki et al., 2004) as well as the differing functions of specific sections within a single muscle group (Butler, Hubley-Kozey, Kozey, 2009b), previous EMG investigations have used a comprehensive set of muscle sites to capture neuromuscular patterns during functional tasks (Butler et al.,2009b; Butler et al., 2010; Hubley-Kozey et al., 2012, Butler et al., 2013; Hubley-Kozey et al., 2014). Using such an inclusive set of muscle sites may be advantageous as it negates the necessity to subjectively 'choose' which muscles may be important during the EMG analysis of a particular functional task providing a more complete and objective investigation. As a consequence, large data sets are created presenting logical issues with respect to data analysis which have been previously managed through various data reduction techniques (Butler et al., 2010; Hubley-Kozey et al., 2012, Butler et al., 2013; Hubley-Kozey et al., 2014). Specifically, Principal Component Analysis (PCA), which is based on pattern recognition techniques, has been previously shown to effectively manage large quantities of data by reducing the number of variables used for statistical analysis while still maintaining important features of the data including temporal synergies or the coordination among muscles (Moreside et al., 2014). This technique is used to analyze the entire normalized EMG waveform and subsequently produce principal component (PC) scores that represent the variation in the data from which relevant features can be identified (Jackson, 2003).

The practice of using PCA to examine the coordination of muscles during dynamic tasks has gained popularity in the EMG literature. However, interpretation can be difficult when attempting to associate physiological meaning to the mathematical patterns generated by the analysis. Other common EMG data reduction techniques have been previously employed such as averaging muscles from different sites as well as calculating amplitude ratios (Granata & Orishima, 2001; Marras and Davis, 1998) but have been criticized for loss of important information related to interactions between different muscle sites (Butler et al., 2009b). However, a recent investigation has demonstrated high correlations between amplitude ratios involving abdominal and back extensor muscle sites shown to be important to spinal stability and PC scores generated from EMG activity captured during a symmetrical bilateral lift and replace task similar to the HT phase of the transfer task in a group of recovered LBI individuals as well as a group of healthy controls (Moslehi, Hubley-Kozey, & Quirk, 2014). In considering the previously documented high correlation with PC scores, discrete measures from the normalized EMG amplitude data may serve as a viable reduction technique that reflects similar amplitude and temporal EMG characteristics as those detected by PC scores. In addition to the discrete measure demonstrated by Moslehi (VAR2) two other variables (VAR 1 and 3) were investigated in this study that have not been previously documented. The first, VAR1, was selected with the intent to capture the overall ratio of averaged abdominal and back extensor amplitude during the previously mentioned transfer task. In considering the theoretical framework of the PIT including the importance of the more medial back extensor sites during the test application, the third VAR (VAR 3) was selected with a bias for the more medial back extensor sites. The purpose of generating these discrete measures was to develop more simple measures (compared to PC scores) that have physiological meaning. All three VAR will be defined in the methodology section of this paper.

In summary, the clinical diagnosis of spinal instability has proven to be a challenge. Various clinical measures, both subjective and objective, have been implemented in attempt to identify individuals with suboptimal neuromuscular control. The PIT is a special clinical test for spinal stability that has been shown to be reliable and regardless of the physical characteristics such as sex, mass, and occupation; the PIT has been thought to be both practical and generalizable across all LBP/LBI populations. Specifically, the test has been developed to exploit a deficient passive subsystem as described by Panjabi's three-subsystem model of spinal stability as well as subsequently measure the ability of the neural and active subsystems to generate compensatory active spinal stiffness through appropriate coactivation of the trunk musculature. Previous objective EMG studies have supported similar neuromuscular control strategies during standardized functional tasks. In addition, EMG discrepancies have been demonstrated between recovered LBI individuals and healthy controls as well as recovered LBI individuals who have reinjured and not reinjured. Similarly, the PIT is one of the only clinical measures of neuromuscular control that is applicable to individuals with minimal pain and dysfunction. Combining objective EMG evidence with results from the PIT in a diverse group of recovered LBI subjects could add preliminary evidence validating the PIT as an appropriate and generalizable clinical measure of neuromuscular control. Further, higher reinjury rates in those who test positive during the PIT could support the PIT as being associated with re-injury.

CHAPTER 3 METHODOLOGY

3.1 RESEARCH DESIGN

This cross-sectional comparative study was conducted through a secondary analysis of data comparing differences in EMG activity between a group of military and non-military participants deemed recovered from a LBI based upon self-reported reduction in pain and reported increase in function as well as return to work status. Specifically, the study group included participants within the sub-acute phase of a LBI (between 4 and 12 weeks post LBI) who had been deemed recovered. The definition of recovery was based upon previous investigation and included self-reported remission of symptoms defined as a Visual Analogue Scale (VAS) of less than 20mm and a Roland Morris Questionnaire (RMQ) score of less than 8 as well as a return to normal activities or within one week of returning to normal activities (Butler et al., 2013). Previously documented electromyographic data from each group (military and non-military) were combined and the main objective as well as sub-objectives were addressed by investigating the effects of group status (military vs. non-military) as well as PIT (positive vs. negative) and re-injury status (reinjured vs. non-reinjured), respectively on EMG activity during the HT phase of the transfer task. For the purpose of this secondary analysis, the same standardized methodologies were used to capture data for both previously documented recovered LBI groups (Hubley-Kozey et al., 2014; Trudel, 2014).

3.2 SUBJECTS

The study population consisted of two previously documented recovered LBI groups. The first group consisted of 32 LBI participants between the ages of 20-55 years that were recruited using advertisements and from local physiotherapy clinics (non-military) (Butler et al., 2013). The second group was comprised of 32 LBI participants between the ages of 18-55 years that were

recruited from a local military hospital (military) (Trudel, 2014). The remainder of inclusion criteria for both groups were the same and included (Butler et al, 2013; Trudel, 2014):

- a reported episode of pain between the lower ribs and gluteal folds resulting from a mechanical event causing a low back injury
- within the sub-acute phase of a LBI defined as greater than 4 weeks and less than 12 weeks post-injury
- considered recovered based upon subjective reporting of symptom remission and participation in normal daily activities or within a one-week range of returning to normal activities

LBI participants were excluded in the presence of any neurological, cardiovascular or musculoskeletal condition that would put the participant at risk or prevent proper task completion as well as a history of spinal surgery or presence of structural deformities (scoliosis, spondylolithesis) and/or other diseased processes such as fracture, tumor or infection. Inclusion and exclusion were initially determined through a telephone administered health-screening questionnaire and confirmed during a standard physiotherapy assessment.

3.3 PROCEDURE

The study groups were assessed and completed the study protocol during separate sessions. During the introductory session, a verbal overview outlining the contents of the consent form (Butler et al, 2013; Moreside et al., 2014) was conducted and the participants read and signed the consent form approved by the Health Sciences Research Ethics Board, Dalhousie University. Descriptive variables were recorded including age, sex, mass, occupational level (rated 0-4 based upon occupation activity demand) as well as objective measures of low-back-related disability

(RMQ) (Roland and Morris, 1983), perceptions of pain (Pain Catastrophizing Scale (PCS)) (Sullivan, Bishop, & Pivik, 1995) and current perceived levels of pain (VAS) (Wewers & Lowe, 1990). These measures were taken to ensure low pain levels (VAS), minimal functional limitations (RMQ) and absence of pain behaviors (PCS) supported perception of recovery in the LBI group (Butler et al., 2013). A standard physiotherapy assessment was conducted during the first session in conjunction with the objective questionnaires to determine study eligibility (Hubley-Kozey et al, 2014; Trudel, 2014). Separate registered physiotherapists conducted a postural assessment (including scoliosis and kyphosis), neurological testing including reflex (patellar and achilles tendon, hamstrings), myotomes and dermatomes as well as clinical tests for spinal instability on each group. For the purpose of this secondary analysis, both previous researchers conducted the PIT for clinical spinal instability on all LBI participants in both study groups (Figure 5).

During the second session, a previously documented standardized protocol was used to capture trunk EMG data during a standardized dynamic transfer task (Butler et al, 2009b). Participants were prepared for EMG recordings by placing silver/silver chloride (Ag/AgCl) single use disposable surface electrodes (10mm diameter, Red Dot, 3M, London, Ontario, Canada) in a bipolar configuration (30mm inter-electrode distance) at locations over 12 back and 12 abdominal muscle sites in line with the muscle fibers (figure 3.1) (Butler et al., 2009b). Prior to electrode placement, the skin was prepared for electrode application by shaving hair if necessary and abrading the skin with alcohol swabs in to improve signal conduction (Vezina & Hubley-Kozey, 2000). The abdominal sites used for during the collections included (Butler et al, 2009b): the lower (LRA- midpoint between the umbilicus and pubis) and upper (URA- midpoint between the sternum and the umbilicus) rectus abdominis (Gilleard & Brown, 1994); anterior (EO1 - over the 8th rib) (Ng, Kippers, & Richardson, 1998a), lateral (EO2 - approximately 15 cm lateral to the

umbilicus at a 45° angle) (McGill, 1991) posterior fibers (EO3 - halfway between the iliac crest and the lower border of the ribcage) (Nouwen, Van Akkerveeken, & Versloot, 1987) of external oblique and internal oblique (positioned at the center of a triangle formed by the inguinal ligament, lateral border of the rectus sheath and the line between the two anterior superior iliac spines) (Ng, Richardson, Kippers, & Parnianpour, 1998b). A total of 6 back extensor sites were used and included: lumbar erector spinae at L1 and L3 measured 3 and 6 cm from midline representing the longissimus and iliocostalis muscle sites, respectively (L13, L16, L33, L36) (Vink, van der Velde, & Verbout, 1987); quadratus lumborum at L4 approximately 8 cm from midline (L48); and multifidus at L5 approximately 1-2 cm from midline (L52) (Kavcic, Grenier, & McGill, 2004). Adjustments to electrode placements where made where necessary based upon palpation and specific validation exercises specific for each muscle site including trunk flexion and abdominal hollowing (RA and IO), isometric axial rotation and lateral flexion (EO) and isometric trunk extension (longissimus and iliocostalis) (Butler et al., 2009b). For all EMG recordings, A desired skin impedance of below 200KΩ was confirmed with a multi-meter (Fluke 77) prior to testing.

Myoelectric signals were recorded from a total of 24 muscle sites on the back and abdomen using three surface EMG systems (8 channel, Bortec Inc., Calgary, Alberta). Raw EMG signals will be pre-amplified (200x) and then further amplified using three AMT-8 systems (Bandpass 10-1000Hz; input impedance > 10GΩ; CMRR 115dB, Bortec Inc., Calgary, Alberta). The analogue signal was sampled at 2000Hz using a 16-bit analogue-to-digital (A/D) converter (National instruments, CA-1000) using LABVIEW and was stored on a personal computer for subsequent processing.

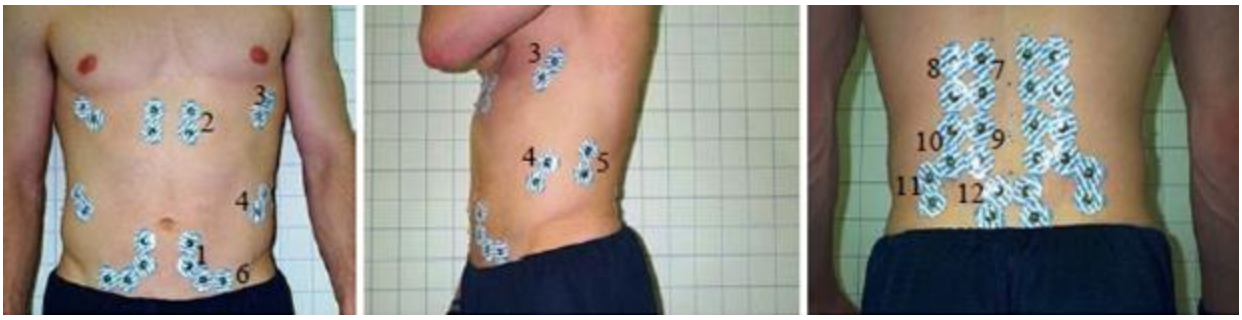


Figure 3.1: **Electrode Placement**: 1=lower rectus abdominis; 2=upper rectus abdominis; 3=anterior fibres of the external oblique; 4=lateral fibres of the external oblique; 5=posterior fibres of the external oblique; 6=internal oblique; 7=longissimus at L1; 8=iliocostalis at L1; 9=longissimus at L3; 10=iliocostalis at L3; 11=quadratus lumborum; 12=multifidus.

3.4 NORMALIZATION PROCEDURES

EMG signal characteristics may vary based on electrode location, electrode or muscle fiber orientation, tissue characteristics and skin preparation procedures and to account for these variables, differential EMG amplitude normalization techniques are considered appropriate (Rutherford et al., 2011; Winter et al., 1994). Therefore, participants were asked to perform a series of exercises that require brief (3sec) maximum isometric voluntary contractions (MVICs) (Butler, Hubley-Kozey, Kozey, 2010). The purpose of these MVICs was to provide a physiological reference in which to make more valid comparisons across muscles and subjects. MVIC normalization has been shown to be effective and reliable (Burden & Bartlett, 1999) and for the trunk muscles, a series of exercises have been recommended (Kavcic et al., 2004). During these normalization exercises, participants were provided with verbal encouragement to increase the likelihood of obtaining a MVIC that is as close as possible to a true maximum (Ng, Parnianpour, Kippers, & Richardson, 2003). Movement during the MVIC exercises was minimized through the use of non-elastic straps to ensure participant's safety and manual resistance was provided in the opposite direction of the intended motion to enforce proper task performance.

A total of 8 exercises previously used for normalization (Butler et al., 2010) were performed to recruit MVICs from all muscle sites. A supine sit-up was used to maximally recruit the rectus abdominus sites (Vezina & Hubley-Kozey, 2000). Side-lying lateral flexion (right and left coupled with ipsilateral hip hike) and seated axial rotations (right and left) was used to maximally recruit the oblique muscle sites. Back extension (McGill, 1991) and back extension coupled with axial rotation (right and left) (Butler et al., 2010) was performed in the prone position to maximally recruit the back musculature. All normalization exercises were repeated twice and held for 3 seconds with a 2-minute rest interval between each contraction for a total of 16 trials. Baseline muscle activity (subject bias) was recorded after the normalization trials for 3 seconds while the subject was lying in a supine and relaxed position. The system bias was recorded for 1 second at the end of the full experimental session and along with the subject bias, was used to correct the EMG data during processing. Post-processing the actual MVIC for each muscle was determined regardless of the actual exercise performance. These trials were checked for each participant. A 500 msec moving window was used on the normalization trials to determine the maximum root mean square (RMS) amplitudes for each of the 24 muscle sites (Vezina & Hubley-Kozey, 2000).

3.5 TEST PROCEDURE

Both study groups involved in this secondary analysis participated in a highly control right-to-left transfer task, described in previous studies (Butler et al., 2010; Hubley-Kozey et al., 2012). This task was developed to dynamically challenge the trunk musculature to constantly changing flexion and lateral flexion external moments while minimizing trunk and pelvic motion (Butler et al., 2010; Hubley-Kozey et al., 2012). Participants were positioned with their body midline aligned with a table that was height-adjusted to the participant's standing elbow height. Subjects were

requested to transfer a 2.9 Kg load from a standard lift position (60° right of midline) (Figure 1a) to a replace position (60° left of midline) (figure 1c) while maintaining a lift height of 5 cm and within a standardized 5 second count (lift on 1, midline on 3, and replace on 5). Pressure sensors positioned on the bottom of the mass relayed time of lift and replace with midline time relayed through the breaking of an optoelectric light sensor (Figure 3.2b). These sequences defined three phases: right hand transfer (RHT), hand transition (HT), and left-hand transfer (LHT) or phase 1, phase 2, and phase 3; respectively (figure 3.2 a-c). Times to complete each phase as well as total time was recorded for each trial. Participants were instructed to maintain a maximum reach position with their elbows fully extended and were encouraged to minimize trunk and pelvic motion through contact feedback with a tactile sensor placed in the mid thoracic region. Trials were repeated in cases of motion detection as well as poor timing. The participants performed the task until 5 successful trials were recorded (Hubley-Kozey et al., 2014, Trudel, 2014).

(A) Right-hand transfer phase **(B) Hand transition phase** **(C) Left-hand transfer phase**

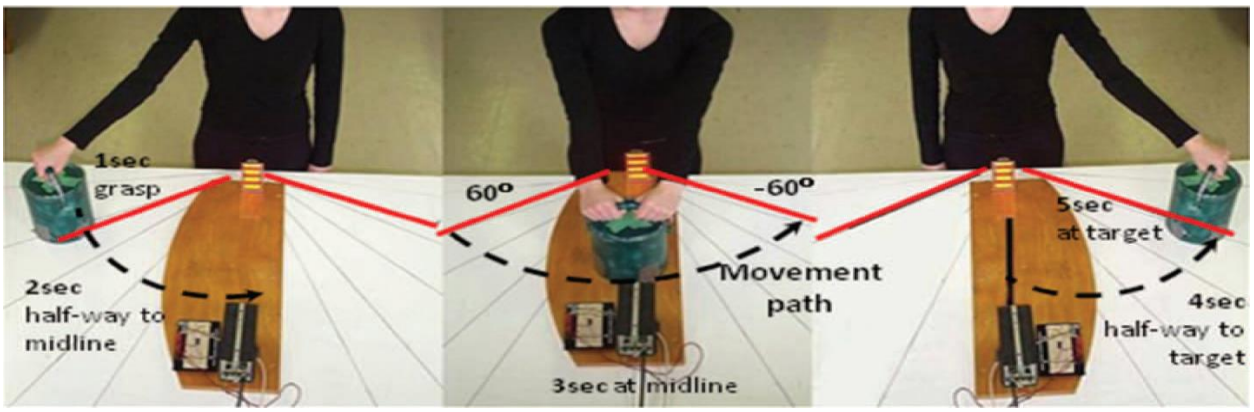


Figure 3.2: Experimental set-up, subject position and movement path for the transfer task.

3.6 DATA PROCESSING

All raw EMG signals were visually inspected for quality, noise levels or artefacts (eg. spikes, DC offsets). A custom program in Matlab® (MathWorks, Inc., Natick, MA. Version 7.3) was used to process the data. Signal contamination due to electrocardiograph artifact was minimized by applying a recursive fifth order Butterworth high pass filter at 30Hz (Butler et al., 2009a) and an inverse Fast Fourier Transform Filter was applied to remove any low-level noise. The raw EMG was corrected for the system and subject bias, adjusted for the true channel gain and full wave-rectified. Root mean square (RMS) amplitude was then calculated for each phase of the transfer task. Data were time normalized from lift off to replace using a linear interpolation algorithm, and then amplitude normalized to the maximum amplitude from the normalization exercises producing %MVIC values.

The normalized RMS (NRMS) data were then used to calculate 3 different variables using various combinations of averaged abdomen and back extensor amplitudes for each subject during the HT phase (phase 2) of the right-to-left transfer task. The purpose of using the variables for comparison was to generate simplistic discrete measures from the normalized EMG amplitude that were physiologically relevant and highly correlated with previously documented PC scores. The three variables that were investigated included:

1. VAR 1 = $\Sigma\, NRMS\ of\ Abdomens / \Sigma\, NRMS\ of\ Extensors$
2. VAR 2 = $\Sigma\, NRMS\ of\ Abdomens / \Sigma\, L13, L16, and\ L33\ all\ together$
3. VAR 3 = $\Sigma\, NRMS\ of\ Abdomens / \Sigma\, L13, L33, and\ L52\ all\ together$

Only the second variable has been previously investigated and has been shown to be highly correlated with PC scores calculated for subjects during a symmetrical bilateral lift and replace

task (Moslehi et al., 2014). Principal component analysis is a multivariate statistics technique used to describe the variability within a group of related variables, and has been shown to be an efficient data reduction technique for understanding intricate co-activation and temporal synchronies when analysing large quantities of data (Butler et al., 2009b; Hubley-Kozey & Vezina, 2002). Following calculation of all variable scores, correlations were performed to determine the variable that demonstrated the highest correlation to PC scores during phase 2 of the dynamic transfer task. This variable was used in statistical analysis to perform subsequent between-group comparisons. Previously documented PC scores calculated from normalized EMG amplitude data captured during the transfer task have been shown to represent neuromuscular responses to the changing flexion and lateral flexion moments resulting from the external load (container). Specifically, PC1 has been shown to represent the overall amplitude of activity during the transfer task and PC2 has been shown to represent the neuromuscular response to the changing lateral flexion moment. Most important to this secondary analysis, PC3 has been shown to capture the neuromuscular response to the change in flexion moment.

For the purpose of this secondary analysis, only phase 2 (HT) EMG amplitude data were analysed due to the similar effect of the moment generated during the HT phase of the transfer task and the PIT. Specifically, the effect of the external flexion moment during HT is an anterior shear force through the intervertebral segments of the lumbar spine. This force must be countered by appropriate levels of back extensor activity resulting in an active stiffening of the spine. Similarly, the posterior-anterior force generation (by the examiner) during the PIT results in an anterior shear force through the intervertebral segment that must be countered by appropriate levels of extensor activity during the active component of the PIT.

3.7 STATISTICAL ANALYSIS

Descriptive statistics were calculated for all variables. Differences between groups for age, height, weight, body mass index (BMI), VAS initial, VAS final, RMQ, PCS, and occupation level were compared using independent t-tests. A general linear model was used to test the variable scores. Specifically, univariate ANOVAs were conducted to test for differences in variable scores between recovered LBI groups during HT (Objective 1). Similarly, univariate ANOVAs were conducted to test the effect of PIT as well as re-injury status on variable scores during HT (sub-Objective 1 and 2). In addition, independent t-tests were used to compare phase 2 PC scores between groups (non-military vs. military) as well as between subgroups (PIT response and re-injury status). For each analysis, assumptions of normality and homogeneity of variance were examined and transformations were performed in cases of non-normal distribution. All tests were performed using SPSS (IBM Corporation, Somers, NY, version 22). A critical alpha level of 0.05 was used for all comparisons.

CHAPTER 4 RESULTS

4.1 Correlations between Variables and Principal Component Scores

All NRMS values for individual muscle sites used in the calculation of VAR 2 scores are illustrated in Appendix 1. Correlations between variables 1-3 and PC scores calculated for phase 2 (HT) are demonstrated in Table 4.1. All variables demonstrated high correlation with PC3 scores (response to changing flexion moment) and generally poor correlation with PC2 scores (response to changing lateral flexion moments). Specifically, the highest R^2 value (0.898) was demonstrated for variable 2 and PC3 (Figure 4.1). Therefore, variable 2 was used for all subsequent statistical analysis. As a confirmation of similar findings across phases, correlations were also conducted for variables and PC scores during phase 1 (RHT) and phase 3 (LHT). Tables containing R^2 values for all phase 1 and 3 correlations can be found in Appendix 2. Generally speaking, all variables remained highly correlated to PC3 scores for phase 1 and 3 with variable 1 demonstrating the highest correlation to PC3 for phase 1 (0.892) and phase 3 (0.906), respectively. It is also worth noting that all variables were highly correlated with each other for phase 2 (Table 4.1) as well as phase 1 and 3 (Appendix 2).

Table 4.1: Phase 2 Correlations

	PC3	PC2	VAR 1	VAR 2	VAR 3
VAR 1	0.886**	-0.168	1	0.948**	0.985**
VAR 2	**0.898****	-0.240	0.948**	1	0.913**
VAR 3	0.844**	-0.116	0.985**	.913**	1
PC2	-0.3	1	-0.168	-0.240	-0.116
PC3	1	-0.3	0.886**	0.898**	0.844**

VAR 1 = Abdomens/Extensors, VAR2 = Abdomens/L13, L16, L33, VAR 3 = Abdomens/L13, L33, L52, PC3 = Principal Component 3 score, PC2 = Principal Component 2 score. ** = significant correlation at the 0.01 level (2-tailed). Highest correlation between VAR and PC scores is bolded.

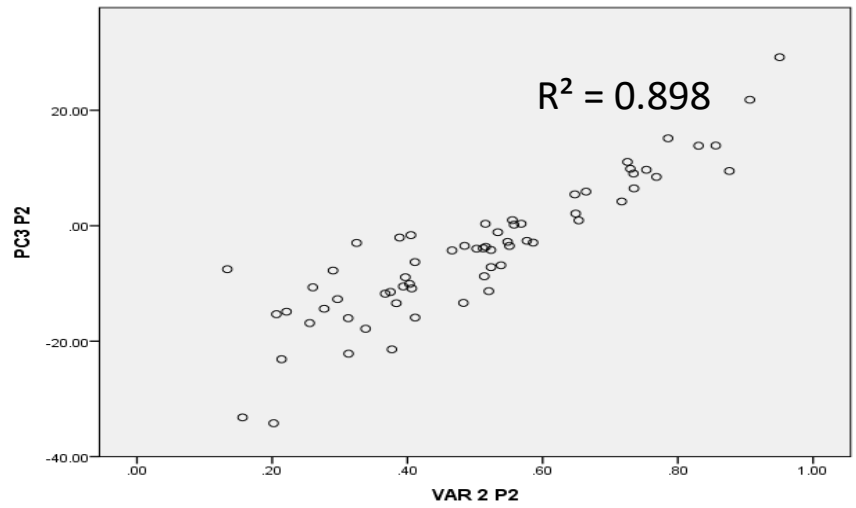


Figure 4.1: Phase 2 Correlations

VAR 2 = Abdomens/L13, L16, L33; PC3 = Principal Component 3. Correlation scatterplots between VAR 2 and PC3 phase 2 (P2) values.

4.2 Comparison of VAS Scores (Military vs. Non-Military and PIT Positive vs. PIT Negative)

In the preliminary design of this study the intent was to focus on participants who had an initial VAS score of 20 or less, to reduce the effect, if any of pain on the EMG activation levels in the analysis. A review of the available data showed that of the 32 participants in the military and the 32 participants in the non-military data sets, removing the participants with scores greater than 20 would reduce the sample size to 30 and 25 respectively. In addition, the number of final VAS scores exceeding 20mm were 10 in the military data set and 10 in the non-military data set. Rather than removing a substantial number of participants, an analysis was performed to compare the effects of the VAS scores on the samples and the EMG amplitudes.

As anticipated, the VAS scores (initial, final, and difference) demonstrated a non-normal distribution. Therefore, a non-parametric analysis using the Kruskall-Wallis test was performed

to compare VAS scores between military and non-military groups as well as between those who tested positive and negative during the PIT. Results of the Kruskall-Wallis tests can be found in Appendix 3. There were no significant differences in median VAS scores between the military and non-military groups (p=0.909) or between those who tested positive and negative during the PIT (p=0.598). Additionally, a correlational analysis was also conducted to see if there was a relationship between the EMG amplitude (variable 2 scores) and the VAS scores (initial, final and difference). The correlations (r values) ranged from .037 to 0.123; none of which were determined to be significant (Appendix 4).

In considering these findings, the assumption is maintained that differences detected in variable 2 scores based upon PIT status as demonstrated by this secondary analysis were not influenced by pain levels prior to, or during the experimental protocol.

4.3 Military vs. Non-Military Group Comparisons

4.3.1 Group Demographic Variables

There were 32 subjects in the military LBI group and 32 subjects in the non-military LBI group for a total of 64 participants. Descriptive data for both groups are found in Table 4.2. Significant higher mass (p=0.001), BMI (p=0.002), and occupation activity (p=0.001) were demonstrated in the military group compared to the non-military group. Further, the military group demonstrated a greater percentage of men (84%) compared to the non-military group (44%).

Table 4.2: Subject Demographics (Military vs. Non-Military): mean (SD)

Variable	Military(n=32)	Non-Military (n=32)	P-value
Age (yr)	37.8(10.0)	39.5(11.8)	0.547
Height (cm)	172.7(7.0)	170.1(8.8)	0.209
Mass (Kg)	93.7(14.6)	79.0(20.1)	**0.001**
BMI (m^2/Kg)	31.3(3.7)	27.2(6.3)	**0.002**
VAS initial	9.5(8.0)	12.8(14.8)	0.273
VAS final	13.8(13.3)	14.8(16.1)	0.771
RMQ	3.3(3.4)	4.4(4.4)	0.283
PCS	9.4(10.5)	11.8(9.3)	0.335
Occupational Activity Level	2.1(1.0)	0.8(1.1)	**0.001**
Number of Females	5	18	
Number of Males	27	14	
Number of Reinjured	15	14	
Number of Non-Reinjured	17	14	

BMI = Body Mass Index; VAS = Visual Analogue Scale; RMQ = Roland Morris Disability Questionnaire; PCS; Pain Catastrophizing Scale. Bolded values indicate significant differences between groups

4.3.2 Group EMG Analysis

A general linear model was used to compare variable 2 scores (dependant variable) from phase 2 between military and non-military recovered LBI groups. In addition to group (non-military vs. military), fixed factors age and sex were also included in the model based upon the previously documented effects of age (Quirk & Hubley-Kozey, 2013) and sex (Hubley-Kozey et al., 2012) on EMG activity during the transfer task. ANOVA results are illustrated in Table 4.3.

Table 4.3: ANOVA results comparing Group (Non-Military vs. Military) VAR 2 Scores for Phase 2

Variable	Mean Square	F	P-value
Group	0.013	0.327	0.572
Age	0.039	1.008	0.496
Sex	0.003	0.089	0.768

Observation of the linear model ANOVA results demonstrates that significant differences were not detected in variable 2 scores based upon group (non-military vs. military), age, or sex variables. Further, independent t-tests were conducted to compare PC scores between groups as demonstrated in Table 4.4. Similarly, no significant between-group differences were detected in phase 2 PC scores.

Table 4.4: t-test results comparing Phase 2 PC scores between groups

Variable	Military (n=32)	Non-Military (n=32)	P-value
PC1	71.08(25.5)	82.62(37.67)	0.156
PC2	-1.36(5.79)	-1.13(9.44)	0.906
PC3	-4.46(10.66)	-4.29(13.12)	0.955

4.4 PIT Positive vs. PIT Negative Comparisons

4.4.1 PIT Status Demographic Variables

Descriptive data for PIT positive and PIT negative individuals are illustrated in Table 4.5. In the combined data there were a total of 33 and 31 individuals that tested negative and positive for the PIT, respectively. Only occupation activity level was significantly different (p=0.006) between groups with those who tested positive during the PIT demonstrating higher occupational demand. A large majority of PIT positive individuals (n=23, 74%) were from the military population while the non-military contributed a large portion of the PIT negative individuals (n=24, 73%). Further, 71% of PIT positive individuals were also men. Qualitatively, the occurrence of re-injury appears to be similar between those who test positive and negative during the PIT.

Table 4.5: Subject Demographics (PIT Negative vs. PIT Positive): mean (SD)

Variable	PIT Negative (n=33)	PIT Positive (n=31)	P-value
Age (yr)	38.3(11.4)	39.0(10.9)	0.782
Height (cm)	180.0(8.0)	172.9(8.1)	0.669
Mass (Kg)	82.0(18.5)	90.9(18.9)	0.063
BMI (m^2/Kg)	28.0(5.0)	30.6(5.2)	0.057
VAS initial	11.2(12.8)	11.1(11.2)	0.997
VAS final	11.4(12.5)	17.4(16.3)	0.106
RMQ	3.8(4.6)	3.9(3.5)	0.979
PCS	9.9(8.7)	11.3(11.2)	0.584
Occupational Activity Level	1.0(1.1)	1.9(1.3)	**0.006**
Number Reinjured	14	14	
Number Non-Reinjured	14	16	
Number of Females	14	9	
Number of Males	19	22	
Number of Military	9	23	
Number of Non-Military	24	8	

BMI = Body Mass Index; VAS = Visual Analogue Scale; RMQ = Roland Morris Disability Questionnaire; PCS; Pain Catastrophizing Scale. Bolded values indicate significant differences between groups

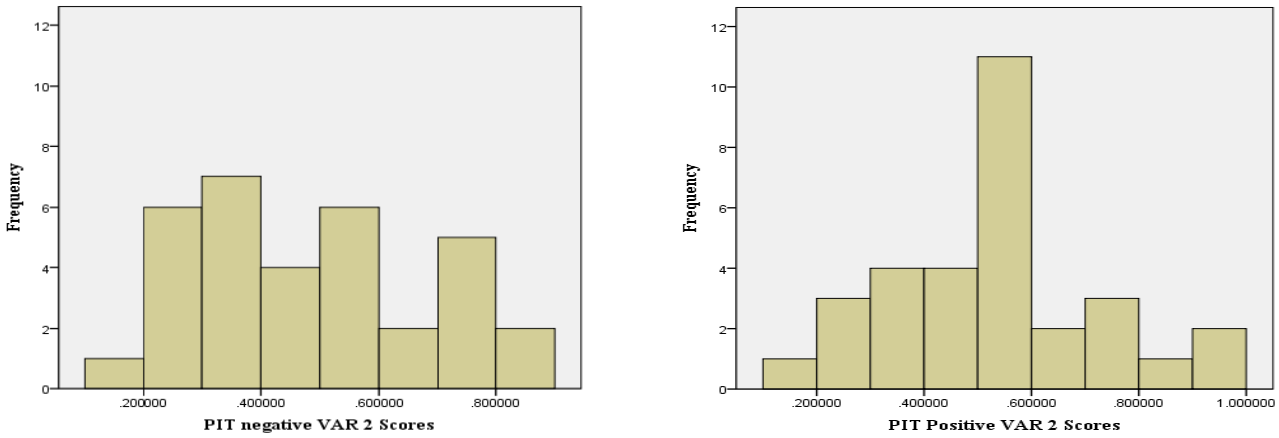
4.4.2 PIT Status EMG Analysis

Factors included in the general linear model for the PIT status analysis included group (military vs. non-military), age, sex, and PIT status with phase 2 variable 2 scores being the dependant variable. Results of the ANOVA are illustrated in Table 4.6. Out of the 4 variables, differences in variable 2 scores could only be detected based upon PIT status (p=0.038). Similar to the previous analysis, differences in variable 2 scores were not detected based upon group, age, or sex. Qualitatively, variable 2 scores demonstrated by PIT positive responders appear to be more clustered around a ratio score of 50% compared scores demonstrated by PIT negative responders as illustrated by figure 4.2. Variable 2 scores are provided for both PIT positive and negative individuals in Table 4.7. In addition, because participants from the military group as well as those

who tested positive during the PIT demonstrated higher occupational activity scores (p= 0.001 and 0.006, respectively) a post-hoc analysis was conducted with the inclusion of the factor PIT*occupational activity to determine if significant differences in VAR 2 scores detected based upon PIT status were related to differences in occupational activity level. The results of the ANOVA can be found in Appendix 5. Based upon the post hoc analysis, significant differences in VAR 2 scores were not related to occupation activity as differences in scores were detected based upon PIT status (p=0.05) and not based upon the factor PIT*occupation (p=0.22).

Table 4.6: ANOVA results comparing Phase 2 VAR 2 scores for PIT status. Bolded values indicate significant differences between groups

Variable	Mean Square	F	P-value
Group	0.099	2.904	0.100
Age	0.043	1.262	0.270
Sex	0.004	0.119	0.733
PIT	0.164	4.781	**0.038**



a. PIT negative Variable 2 scores **b. PIT positive Variable 2 Scores**

Figure 4.2: Histograms showing frequencies of Variable 2 Scores for PIT negative and positive individuals

In addition, PC scores were compared between those who tested positive and negative during the PIT and results are demonstrated in Table 4.8. Significant differences were not detected in phase 2 PC scores between those who test positive and negative during the PIT.

Table 4.7: Variable 2 Scores for PIT positive and negative groups

Frequency	PIT negative	PIT Positive	Frequency
1	0.1562	0.1336	1
6	0.2022	0.2063	3
	0.2555	0.2139	
	0.2601	0.2212	
	0.2769	0.3771	4
	0.2901	0.3837	
	0.2966	0.3938	
7	0.3124	0.3972	4
	0.3131	0.4031	
	0.3248	0.4051	
	0.3382	0.4064	
	0.3672	0.4661	
4	0.3749	0.5023	11
	0.3884	0.5137	
	0.4111	0.5158	
	0.4114	0.5203	
	0.4828	0.5236	
	0.4845	0.5385	
6	0.5122	0.5482	
	0.5154	0.5552	
	0.5237	0.5578	
	0.5337	0.5768	
	0.5509	0.5861	
2	0.5683	0.6489	2
	0.6476	0.6533	
5	0.6642	0.7255	3
	0.7171	0.7535	
	0.7299	0.7856	
	0.7345	0.8561	1
	0.7351	0.9065	2
	0.7682	0.9506	
2	0.8306		
	0.8762		

Table 4.8: t-test results for Phase 2 PC score comparisons between PIT negative and positive

Variable	PIT negative(n=33)	PIT positive (n=31)	P-value
PC1	79.00(37.47)	74.55(26.47)	0.156
PC2	-.36(7.73)	-2.17(7.76)	0.906
PC3	-5.93(11.87)	-2.71(11.81)	0.955

4.5 Reinjured vs. Non-reinjured Comparisons

4.5.1 Re-injury Status Demographic Variables

From the original 64 participants, there were 59 available for follow-up details on re-injury status from which 30 non-reinjured and 29 reinjured individuals were identified. Descriptive data for non-reinjured and reinjured individuals are presented in Table 4.9. Significant differences were detected between reinjured and non-reinjured individuals including a higher VAS initial (p=0.003), VAS final (p=0.017), and RMQ score (p=0.021) in the reinjured subjects. Qualitatively, it does not appear that group status or PIT status is related to re-injury status with the frequency of re-injury being similar between military (n=15) and non-military (n=14) individuals as well as between those who test positive (n=14) and negative (n=14) during the PIT.

4.5.2 Re-injury Status EMG Analysis

A general linear model was used to determine if differences in phase 2 variable 2 scores could be detected based upon those who did, or did not reinjure. Similar to the previous two analyses, other fixed variables included in the model included group status, age, sex, and PIT status. Based on the result of the AVOVA (Table 4.10), differences in phase 2 variable 2 scores were not significant based upon re-injury status (p=0.986). Similarly, differences could not be detected based upon group status, age, or sex. As expected from the previous analysis, differences could be detected in variable 2 scores based upon PIT status (p=0.021). Comparisons between phase 2 PC scores for re-injury status are illustrated in Table 4.11. Significant differences in phase 2 PC scores were not detected between those who re-injured and those who did not.

Table 4.9: Subject Demographics (non-reinjured vs. reinjured): mean (SD)

Variable	NonReinjured(n=30)	Reinjured (n=29)	P-value
Age (yr)	37.0(11.6)	41.1(10.3)	0.153
Height (cm)	172.3(6.0)	171.0(9.0)	0.522
Mass (Kg)	90.2(19.7)	85.3(18.0)	0.316
BMI (m^2/Kg)	30.3(6.0)	29.0(5.2)	0.397
VAS initial	6.9(8.2)	16.1(14.1)	**0.003**
VAS final	10.4(12.9)	19.6(15.7)	**0.017**
VAS difference	3.5(7.7)	3.5(8.7)	0.993
RMQ	2.8(3.5)	5.2(4.4)	**0.021**
PCS	10.2(10.0)	11.6(10.5)	0.614
Occupational Activity Level	1.5(1.2)	1.4(1.3)	0.669
Number of Females	10	11	
Number of Males	20	18	
Number of Military	17	15	
Number of Non-Military	13	14	
Number of PIT positive	16	14	
Number of PIT negative	14	14	

BMI = Body Mass Index; VAS = Visual Analogue Scale; RMQ = Roland Morris Disability Questionnaire; PCS; Pain Catastrophizing Scale. Bolded values indicate significant differences between groups

Table 4.10: ANOVA results comparing Phase 2 VAR 2 scores for Re-injury status. Bolded values indicate significant differences between groups.

Variable	Mean Square	F	P-value
Group	0.080	2.298	0.143
Age	0.039	1.112	0.401
Sex	0.000	0.004	0.953
PIT	0.213	6.104	**0.021**
Reinjury	1.087	0.000	0.986

Table 4.11: t-test results comparing Phase 2 PC scores for Re-injury Status.

Variable	Non-Reinjured(n=30)	Reinjured (n=29)	P-value
PC1	76.23(32.56)	79.76(32.49)	0.679
PC2	-2.29(8.70)	-.87(6.94)	0.493
PC3	-6.01(10.99)	-3.78(12.97)	0.481

CHAPTER 5 DISCUSSION

During this secondary analysis of data, comparisons of EMG data collected during the HT phase of the transfer task were made between a recovered LBI military and non-military population. Additionally, comparisons of EMG data were made based upon PIT and re-injury status in the same recovered LBI populations. Specifically, three variables were developed and correlations were conducted between these variables and previously generated PC scores (Hubley-Kozey et al., 2014, Trudel, 2014) to determine the most appropriate measure for statistical analysis. All three variables were ratio variations of averaged abdominal to extensor amplitude activity and represented discrete measures calculated from the normalized EMG amplitude data. Consistent with a previous investigation (Moslehi et al., 2014), variable 2 ($\Sigma\, NRMS\ of\ Abdomens / \Sigma\, L13, L16, and\ L33\ all\ together$) demonstrated the highest correlation with PC scores and was subsequently selected for comparison between groups. Speaking to the high correlation between variable 2 and PC3 during HT ($R^2 = 0.898$), previously calculated PC3 scores for both groups (military and non-military) during the dynamic transfer task were shown to represent a temporal response to the increased flexion moment generated during HT (Hubley-Kozey et al., 2014; Trudel, 2014). As expected, the positive PC3 scores indicating an increased relative response to the flexion moment generation during HT were highly correlated with all three variables which represented subtle variations of abdominal to extensor ratio activity that would also be expected to elicit responses to the increasing flexion moment during HT. Similarly, poor correlation between all variables and PC2 scores was expected as the PC2 values have been previously shown to represent a response to changing lateral flexion moment which is expected to contribute minimally to trunk muscle activity during HT (Hubley-Kozey et al., 2012, 2014; Trudel, 2014).

Regardless of significant differences in mass, BMI, and occupation activity level; differences in EMG activity could not be detected between recovered LBI individuals recruited from a military population and recovered LBI individuals recruited from a non-military population during the HT phase of the transfer task. The significant difference in BMI (p = 0.002) was assumed to related to the significant difference in mass (p = 0.001) as there was no significant difference in height between groups. Irrespective of the tissue characteristics resulting in significantly higher mass in the military group, proper normalization procedures as well as appropriate gain levels were applied to minimize the effects of differences in mass on the EMG signals (Winter et al., 1994, Butler, 2009a). As demonstrated in this analysis, it may be assumed that military workers are subjected to higher activity occupational demands compared to their non-military counterparts. However, the dynamic transfer task was originally designed to reflect a classification of low effort jobs that better represents the physical activity demands of modern-day workers (Butler et al., 2010). A minimal load of 2.9 Kg was chosen for the task based upon the need to produce a measurable neuromuscular response without increasing unwanted excessive spinal load (Butler et al., 2009b). In considering the modest physical demand of the dynamic transfer task, similar performances were expected from each group regardless of previous conditioning from occupational activity demands.

In addition to a significantly greater mass, a greater portion of the military group (84%) were male in comparison to the non-military group (44%). Previous investigations have demonstrated important muscle morphological, anthropometric, and biomechanical differences in men and women (Jorgensen, Marras, Granata, & Wiand, 2001; Mazis et al., 2009). In an EMG study comparing activation amplitudes and temporal synergies among trunk muscles in healthy men and women during the dynamic transfer task, Hubley-Kozey and colleagues demonstrated

more asynchronies as well as higher overall amplitudes in women compared to men (Hubley-Kozey et al., 2012). In the current analysis, differences in EMG activity were not detected based upon sex. However, the current analysis compared only EMG data collected during HT and did not consider the neuromuscular responses to lateral flexion moments generated during left and right-hand transfer phases where Hubley-Kozey et al. documented discrepancies between men and women (Hubley-Kozey et al., 2012). Further, even though the averaged ratios used in the current analysis have been shown to be highly correlated with PC scores, challenges arise with detecting intricate differences between specific muscle groups as demonstrated by the previous study (Hubley-Kozey et al., 2012). The inclusion of recovered LBI subjects compared to healthy controls may also speak to the lack of differences found between men and women in the current analysis as previous findings from recovered LBI populations are similar to those previously demonstrated for women including increased overall amplitude activity and greater activation variability (Hubley-Kozey et al., 2012; Butler et al., 2013; Hubley-Kozey et al., 2014).

Differences in EMG activity during HT were detected based upon PIT status ($p = 0.038$). Demographically, a large portion of PIT positive individuals were men (n = 22, 71%) and military workers (n = 23, 74%). However, because differences in EMG activity were not detected based upon group status ($p = 0.100$), it is highly unlikely that differences detected based upon PIT status were influenced by the high percentage of military workers in the PIT positive group. In addition, similar to the previous analysis on group status, differences in EMG activity were not detected during HT based upon sex ($p = 0.733$). Similarly, findings from the post hoc analysis does not support an effect of occupational activity level on differences detected in VAR 2 scores based upon PIT status. Inter-rater reliability may speak to the higher percentage of PIT positive individuals in the military group (or lower percentage of PIT negative individuals) compared to

the non-military group. The examiners that conducted the PIT on each group were independent from one another. However, both examiners were experienced physiotherapists and detailed instruction for all clinical examination procedures including the PIT were provided prior to initiating subject assessments. In addition, previous investigations have demonstrated moderate to good interrater reliability for the PIT with kappa values ranging from 0.54-0.87 (Hicks et al., 2003; Fritz et al., 2005a; Schnieder et al., 2008). Thus, between-group discrepancies in PIT ratios are unlikely due to inter-examiner differences.

Upon examination of the HT variable 2 scores for positive PIT responders, it appears that a great number of scores appear to be clustered around the 50 % level with respect the VAR 2 ratio calculations. The task was highly controlled so differences between groups cannot be explained by task-specific differences such as timing and motion during task completion (Butler et al., 2013). Specifically, this demonstration of score clustering in those who tested positive during the PIT may represent a 'common neuromuscular strategy' in response to the increased flexion moment generated during HT. Higher agonist-antagonist co-activation is a strategy that has been previously shown to increase spinal stability through the generation of appropriate levels of compensatory active spinal stiffness in the presence of deficient passive spinal stiffness (Tucker and Hodges, 2009). Theoretically, the PIT is designed to exploit compromised passive spinal stiffness (passive component) as well as confirm the compensatory ability of the active subsystem to generate appropriate active stiffness in order to restore optimal spinal function (stability) related to the active component of the stability model. This common strategy demonstrated by the PIT positive group may represent an overall bracing mechanism in response to the increased neuromuscular demand during HT. However, due to the general nature of this analysis, specific neuromuscular strategies, such as co-activation and temporal synergies become difficult to identify

and therefore, the determination of exact neuromuscular discrepancies between those who tested positive and negative during the PIT during HT is outside the scope of this analysis.

The results of this study contribute preliminary evidence supporting the generalizability of the PIT amongst different demographic populations. Results were drawn from two seemingly different populations of recovered LBI individuals and considering all factors included in the general linear model for this analysis including group status, age, sex, and PIT status; differences in EMG activity during the HT phase of the transfer task could only be detected based upon whether individuals tested positive or negative during the PIT. These findings support a previous investigation that was limited to the military subjects used in this secondary analysis (Trudel, 2014). Specifically, the investigation showed minor differences in the trunk extensors sites during the transfer task whereby greater variability in EMG activation patterns were demonstrated for those who tested positive during the PIT compared to those who tested negative (Trudel, 2014). Even though the averaged abdominal/extensor ratios used in the current analysis where shown to be highly correlated to PC3 scores providing a level of temporal consideration, this method was limited to averaged EMG activation and failed to investigate specific between-muscle interactions and temporal synergies that were demonstrated in the original PCA (Trudel, 2014). Additionally, the current analysis considered only HT and therefore, cannot speak to variations between groups during the lift and replace phases of the transfer task which were considered in the original analysis. Hand transition was specifically selected for analysis because the flexion moment and the resultant trunk EMG activity generated during HT more closely replicates the assumed sagittal moment and resultant trunk EMG activity generated during the PIT. Consideration of the theoretical premises supporting the PIT assumes that trunk neuromuscular responses generated during the active component of the PIT are minimally affected by frontal or lateral flexion

moments as demonstrated during the right and left-hand transition phases (also called phase 1 and 3) of the transfer task.

Based on the results of this investigation, it does not appear that the PIT is associated with risk of re-injury as a stand-alone factor. A total of 29 subjects experienced a re-injury during a 1-year follow-up period of which 50% (n =14) tested positive and 50% (n = 14) tested negative during the PIT (1 subject did not have PIT data). As previously defined and classified in this secondary analysis (Hubley-Kozey et al., 2014), LBI individuals were considered to have re-injured if they experienced a re-injury episode to the low back resulting in lost time from work and/or normal activities or requiring medical attention during the year. Factors that may render individuals vulnerable to re-injury may include incomplete recovery from an original LBI as well as underlying predisposing conditions (Butler et al., 2012; Cholewicki et al., 2005). At any rate, identifying predictors or clinical indicators of complete recovery as well as subsequent risk of re-injury may improve clinical decision-making with respect to appropriate discharge from care as well as generation of appropriate return to work/activity strategies. Realistically, such clinical decisions and subsequent recommendations/actions are not based upon any single factor or specific clinical test or measurement. More commonly, combinations of clinical tests and/or measurements are clustered together and used to guide appropriate evidence-based clinical decisions. During a recent EMG investigation which demonstrated modest differences in a group of recovered LBI individuals that reinjured compared to a group that did not re-injure, Hubley-Kozey and colleagues also classified a higher portion of the re-injured individuals as having control impairment (46%) (non-reinjured group = 17%) based upon a previously documented clinical criterion using a cluster of clinical tests and palpatory findings (Stuges et al., 2005). Therefore, the PIT should be further examined as one of several potential factors associated with risk of re-injury in conjunction with

other potential clinical predictors. This also indicates that more likely, no one single clinical test can completely classify individuals in a multi-factorial problem like LBI.

In contrast to Hubley-Kozey et al. and the hypothesis of this study, the current analysis failed to detect differences in EMG activity based upon re-injury status during the HT phase of the transfer task (Hubley-Kozey et al., 2014). In their analysis, Hubley-Kozey et al. demonstrated significant higher PC1 scores calculated from data captured during the entire transfer task for abdominal and extensor groups in a re-injured group compared to non-reinjured and healthy groups. In general, PC1 scores reflect the mean EMG activity in the active muscles. The ratio scores used in the current analysis were generated from EMG data captured during HT and were shown to correlate poorly with PC1 scores speaking to the potential discrepancy in findings. In part, this is reasonable given that the ratio method used in this study may mask the mean levels. For example, two individuals of similar ratio scores may have different mean absolute scores between muscle groups. Similar to the previous investigation (Hubley-Kozey et al., 2014), the re-injured group in this analysis had higher VAS scores before (p = 0.003) and after (p = 0.017) testing as well as higher RMQ scores (p = 0.021) compared to the non-reinjured group. However, these scores were considered to represent low pain and minimal disability (Jensen, Chen, & Brugger, 2003; Lee, Hobden, Stiell, & Wells, 2003; Stratford, Binkley, Riddle, & Guyatt, 1998). Further, when considering increased pain levels resulting directly from test procedures (VAS difference = VAS final - VAS initial), differences between groups were not detected (p = 0.993) indicating a minimal effect of testing on pain levels for both groups.

While this analysis demonstrated differences in EMG activity in a group of recovered LBI subjects who tested positive compared to those who tested negative during the HT phase of the transfer task, the question of re-injury predictability still remains. These study findings add

preliminary evidence supporting the theroretical framework of the PIT as well as preliminary evidence of test generalizability among different LBI populations. However, the next step would be to combine the PIT with other clinical measures that may be associated with re-injury as well as objective EMG evidence to begin to develop a more complete model of reinjury prediction.

CHAPTER 6 SUMMARY AND CONCLUSIONS

6.1 SUMMARY

The following is a review of the main and two sub-objectives of this secondary analysis as well as the specific investigative findings for each:

1. To determine if differences in trunk muscle EMG activity could be detected during a standardized functional task between two groups recruited from different recovered LBI populations.

- Demographic differences were detected between a group of military and non-military recovered LBI participants including mass, BMI, and occupational activity levels. However, differences in EMG activity during the HT phase of the transfer task could not be detected based upon group status.

2. To determine if differences in trunk muscle EMG activity could be detected between recovered LBI participants based upon a positive or negative response during the PIT.

- Differences in EMG activity during the HT phase of the transfer task were detected in recovered LBI participants based upon a positive or negative response during the PIT. Individuals who tested positive during the PIT demonstrated a qualitative clustering of VAR 2 ratio scores around the 50% level when compared to scores demonstrated by those who tested negative during the PIT. Further, the demographic variable occupation activity level was also different between those who tested positive and negative during the PIT. However, in consideration of the results of a post hoc analysis, differences in VAR 2 scores detected based upon PIT status were not influenced by occupational activity level.

3. Determine if differences in trunk muscle EMG activity could be detected between recovered LBI participants based upon the presence or absence of re-injury.

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- Demographic differences were detected between individuals based upon reinjury status including higher initial and final VAS scores as well as higher RMQ scores in those who reinjured during a 1-year follow-up period compared to those who did not reinjure. However, in contrast to the original hypothesis of this study, differences in EMG activity during the HT phase of the transfer task could not be detected between recovered LBI participants based upon reinjured status.

The intent of this secondary analysis was to use an objective and physiologically-relevant measurement to compare two groups of recovered LBI participants that were recruited from potentially different demographic populations. Electromyographic data captured during a portion (HT) of previously documented highly-controlled transfer task were used to make comparisons between a military and non-military group. In addition, EMG findings were combined with a clinical measurement typically used to identify lumbar spine instability in attempt to identify differences that may exist between those who test positive and negative during the PIT.

Differences in in variable 2 scores were not detected during HT were not detected based upon group status (military vs. non-military). Demographically, the groups exhibited some differences in characteristics such as sex, mass, BMI, and occupational activity levels. However, as previously discussed, because of the highly controlled nature of the transfer task as well as the modest physical demand imposed during the task; these demographic differences were assumed to contribute minimally to the EMG data. Further, methodologies used in this analysis considered only muscle group (abdominals and extensor) ratio activity and therefore may have missed specific between-

muscle discrepancies that could have contributed to differences between groups reported by a previous analysis (Hubley-Kozey et al., 2014).

Differences in variable 2 scores were detected based upon PIT status. A larger majority of PIT positive participants were male and from the military group. In addition to PIT status, other factors included in the general linear model included group status, age, and sex. Differences in variable 2 scores were not detected based upon group status or sex and therefore, the documented differences based upon PIT status were not influenced by the larger percentage of male and military subjects in the PIT positive group. Qualitatively, variable 2 ratio scores were clustered around 50% for PIT positive individuals during HT compared to PIT negative individuals. A detailed explanation for this finding is outside the scope of this secondary analysis due to the limitation of comparing group (abdominal and extensor) averaged EMG activity. However, due to the high correlation with PC3, the clustering demonstrated by PIT positive responders could represent a more common startegy to the increasing flexion moment during HT involving increased abdominal-extensor activity resulting in a reactionary or preparatory bracing strategy.

Electromyographic activity comparisons were also made based upon re-injury status. Differences were not detected between recovered LBI individuals who re-injured during a 1-year follow-up period compared to those who did not re-injure. As previously stated, limitations in the current analysis with respect to data analysis (amplitude ratios) as well as limiting the analysis to a single phase (HT) of a three-phase dynamic task may speak to the discrepancy between the current analysis and a previous report using PCA (Hubley-Kozey et al., 2014). Qualitatively, the frequency of positive PIT responders was similar in both groups. Validated clinical predictors of recovery as well as reinjury would add value to clinical decision-making with respect to appropriate therapeutic care and subsequent discharge from care. The PIT has been shown to be

reliable as well as predictive of successful exercise management of LBP (Hicks et al., 2003). However, as in most evidence-based guidelines and CPRs, clinical decisions are rarely based upon a single test or measurement but more commonly are based upon a battery of clinical subjective and objective assessments. Therefore, it may be more practical to investigate the PIT as being predictive of recovery and/or reinjury only in conjunction with other associated clinical tests and measurements.

6.2 LIMITATIONS

As with all EMG investigations, a limitation is the potential for contribution of crosstalk to the desired signal. However, published protocols were followed for electrode placement as well as validation and processing to ensure minimal signal contribution from unwanted sources (Butler et al., 2009a, Butler et al., 2010). Further, concerns have been generated with respect to obtaining a true representation of MVIC during normalization exercises. In considering minimal pain levels (VAS <20mm) as well as no reports of pain during the MVIC exercises, these concerns were minimized during normalization procedures.

Even though the variable 2 scores used in this secondary analysis were shown to be highly correlated with previously generated PC3 scores, detection of specific between-muscle and/or group-muscle interactions including important muscle synergies becomes challenging following data reduction by way of averaging group (abdominal and extensor) activity. However, only the HT portion of the transfer task was investigated during the current analysis and therefore, consideration of dynamic neuromuscular responses to the changing lateral flexion moment resulting from the hand transfer phases was outside the scope and intent of this analysis. Selection of HT for comparison was based upon the assumed generation of similar moment characteristics during HT and the PIT and therefore, the high correlation between variable 2 data used in this

analysis and previously generated PC3 scores satisfied the intent to consider responses to the flexion moment generated during HT. In addition, overall interpretation of the variable scores were limiting with respect to determining individual contributions from each muscle site to the overall score. For example, a higher variable score (increased overall abdominal activation compared to extensor activation) may indicate a co-activation strategy in response to the flexion moment during HT. However, this assumption may not be accurate do to the masking effect the ratio scores may have on the individual amplitude comtributions each muscle site.

6.3 IMPLICATIONS

This secondary analysis detected differences in variable 2 scores based upon PIT status during the HT phase of a controlled transfer task in a group of recovered LBI subjects. In addition, individuals from a military and non-military population contributed to this analysis supporting the generalizability of the PIT across different demographic populations. This study also adds preliminary objective evidence supporting the PIT theoretical framework as defined by the Panjabi three-subsystem model. Confirmation of a theoretically deficient passive subsystem during the passive component of the PIT as well as efficient active subsystem compensation during the active component of the PIT may speak to the objective EMG discrepancies demonstrated during HT in this analysis based upon PIT status.

6.4 FUTURE RESEARCH

This study failed to demonstrate differences in EMG activity during the HT phase of the dynamic transfer task between a group of military and non-military recovered LBI individuals. As previously mentioned, data reduction techniques used as well as limiting the analysis to HT in the current study prevented the detection of intricate between-muscle differences relating to

neuromuscular responses to changing lateral flexion and flexion moments. Therefore, a future PCA comparison between similar groups during entire transfer task may be warranted. The analytical methods used in this investigation were sufficient to detect differences in EMG activity during HT based upon PIT status. A logical progression from this analysis would be to directly compare positive and negative PIT responders during the actual PIT procedure using EMG analysis. Findings from a direct EMG comparison during the PIT could add preliminary evidence validating theoretical premises that support the PIT as an appropriate clinical measure of neuromuscular control.

Qualitatively, the PIT was not shown to be predictive of re-injury in the current analysis. A future investigation of the predictive capabilities of multiple clinical assessment tools including the PIT may be warranted to identify measures, or clusters of measures, that are most predictive of re-injury. Identification of such measures could aid in appropriate clinical decision-making with respect the to development and implementation of evidence-based therapy interventions as well as determining appropriate criteria for therapy discharge.

6.5 CONCLUSION

In conclusion, the main hypothesis of this investigation was supported in that differences in EMG activity could not be detected during the HT phase of the transfer task based upon group status. In addition, the results also support the hypothesis that differences in EMG activity during the HT phase of the transfer task could be detected based upon a positive or negative response during the PIT. Specifically, variable 2 ratio scores calculated during HT demonstrated a qualitative clustering around 50% in those who tested positive during the PIT compared to those

who tested negative. Conversely, the results did not support the hypothesis that differences in EMG activity would be detected based upon re-injury status.

This secondary analysis has contributed preliminary objective evidence supporting the theoretical framework PIT as well as it's potential in contributing, along with other documented measures, to the clinical assessment and diagnosis of lumbar spine instability. By detecting differences in EMG activity in a diverse group of combined military and non-military individuals that have been deemed recovered from a LBI, this analysis also contributes preliminary evidence supporting the generalizability of the PIT across differing LBI populations.

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APPENDIX 1

Normalized RMS values for all muscle sites used in the calculation of VAR 2 scores for all participants (non-military=red, military=blue). + indicates a positive response during the PIT.

Subject	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO	RL13	LL13	RL16	LL16	RL33	LL33
1	5.85	4.25	2.34	4.93	13.61	15.83	11.97	13.68	10.18	11.45	6.81	4.69	36.39	34.53	33.79	34.21	32.51	33.31
2+	5.22	4.21	4.11	2.43	3.18	1.98	4.71	3.38	4.48	4.77	8.68	8.76	13.78	15.58	11.62	14.94	16.38	12.92
3	29.45	31.76	13.35	15.14	15.05	13.32	26.02	27.41	12.77	13.35	33.06	36.36	43.32	44.68	41.19	46.56	56.30	57.21
4	3.17	1.95	2.69	2.90	3.69	5.55	2.39	6.41	2.81	5.85	8.50	7.21	9.51	15.32	8.69	13.18	13.49	17.54
5+	3.54	3.38	2.73	3.02	5.51	15.40	2.80	6.66	13.71	6.62	10.17	14.61	28.13	35.24	23.61	29.64	27.77	28.52
6	2.83	2.82	1.52	2.20	6.13	3.43	3.00	3.74	3.16	4.05	21.82	20.65	30.48	28.47	30.10	27.17	18.25	19.60
7	4.30	3.89	2.60	3.20	4.57	6.89	18.55	16.99	6.13	5.52	15.21	14.85	14.25	15.24	12.51	13.87	24.27	14.35
8	1.32	1.81	3.10	1.71	4.66	5.31	3.53	2.99	2.28	3.79	6.44	12.35	12.33	18.09	9.60	11.22	22.43	21.98
9	3.34	5.53	7.52	5.50	11.20	8.96	10.84	5.23	6.22	3.14	9.29	15.50	21.72	19.11	18.21	19.38	13.87	11.57
10	4.92	5.41	4.96	7.40	3.98	6.61	5.07	10.54	6.79	4.35	6.69	8.08	35.94	30.49	27.05	21.54	31.07	25.55
11+	18.13	17.18	23.02	25.67	22.71	23.40	24.74	32.40	9.58	22.58	27.34	18.35	19.90	26.02	17.56	28.16	23.65	33.24
12	35.69	12.77	10.19	11.70	30.88	30.86	20.54	11.57	13.44	6.64	32.76	43.99	55.67	21.68	50.71	17.52	54.90	40.05
13	7.56	7.40	6.93	6.77	16.27	12.49	21.12	21.32	10.74	3.48	13.63	7.64	9.25	24.34	9.64	24.83	14.18	17.72
14+	3.62	2.56	1.81	1.41	3.85	4.40	3.06	7.15	5.10	3.47	10.21	14.05	30.53	27.00	33.60	23.09	42.12	29.10
15	1.04	1.05	1.58	0.94	1.60	1.37	0.63	0.74	2.29	1.04	3.58	7.04	6.11	5.49	8.04	7.64	3.40	4.26
16	1.50	1.51	2.09	4.13	2.31	3.66	2.53	2.57	3.88	2.80	9.24	5.92	19.70	13.23	20.49	16.87	30.13	16.58
17	1.73	2.01	5.20	6.30	5.11	3.56	4.28	6.95	5.46	7.46	14.19	8.25	31.91	24.66	36.06	31.07	20.55	33.78
18+	12.57	13.35	6.15	9.90	17.31	16.39	26.93	35.11	17.31	7.77	15.79	15.40	37.18	28.49	39.32	31.02	24.68	21.81
19	8.22	7.46	9.39	6.69	8.50	9.86	8.66	8.76	8.43	10.46	12.80	14.87	34.70	35.84	16.77	22.11	22.38	22.35
20	1.36	1.31	1.79	1.67	7.56	4.72	1.78	1.54	2.53	2.09	3.86	7.13	18.68	16.56	17.47	17.87	18.67	13.35
21+	7.12	6.04	7.50	9.77	13.55	12.94	12.11	14.42	7.39	15.00	9.12	5.66	20.28	39.96	21.65	27.47	33.96	41.50
22	1.00	0.83	0.54	0.72	2.54	2.56	2.42	1.96	4.42	3.66	9.57	14.88	20.46	12.07	30.80	10.14	25.88	19.82
23+	4.54	4.06	3.99	4.25	15.50	10.94	8.87	6.49	4.77	2.24	17.02	19.96	14.16	11.99	7.90	10.47	12.23	14.74
24+	4.22	3.50	4.36	4.73	10.68	8.30	8.12	8.44	10.55	12.59	9.44	7.14	27.90	34.37	33.59	34.33	24.89	26.92
25	4.95	3.23	3.34	3.00	10.22	5.16	8.60	4.89	5.41	4.77	9.67	13.87	27.88	18.50	38.80	17.51	48.64	36.23
26	12.08	13.36	4.12	4.23	16.37	15.73	14.46	16.19	8.21	10.72	7.66	8.07	48.72	39.04	39.84	30.42	30.22	27.61
27	4.32	3.66	8.95	8.80	5.30	5.17	2.15	3.45	4.69	3.23	13.24	13.17	31.12	26.12	23.46	22.66	33.08	24.14
28	1.80	1.86	2.23	2.20	3.29	5.29	2.26	2.72	8.67	6.98	8.47	7.47	23.02	13.75	41.55	11.83	45.62	31.37
29	1.32	2.06	4.06	4.14	3.23	2.55	5.98	3.74	4.78	2.94	11.77	6.35	10.74	9.10	5.88	8.82	7.20	9.01
30	0.77	0.65	0.50	0.84	1.94	1.15	0.82	1.40	0.65	0.81	3.27	3.46	7.81	8.20	6.33	5.24	5.77	5.09
31	7.97	10.45	8.88	10.81	5.28	8.29	9.64	11.19	8.75	9.13	18.59	16.74	15.16	16.05	14.36	13.21	13.53	11.28
32+	7.71	3.71	9.46	13.33	6.14	9.74	6.38	14.23	3.40	9.00	5.74	8.01	41.71	31.12	36.01	24.76	40.05	29.65
33+	4.18	4.60	3.25	3.79	4.75	5.24	8.64	11.78	8.16	9.09	5.49	8.40	21.03	16.06	19.31	15.82	19.30	20.46
34	5.78	10.34	3.61	4.05	5.51	4.97	7.73	9.77	3.61	5.37	12.00	12.30	22.11	20.22	17.42	14.70	26.74	18.42
35+	2.68	2.69	0.97	1.23	3.86	2.36	5.12	5.45	4.15	3.78	8.93	9.61	12.97	11.65	11.49	11.59	12.08	10.57
36	19.39	12.07	6.26	5.91	15.57	11.87	14.32	14.81	12.95	14.97	14.16	17.22	18.34	29.75	24.06	22.92	22.60	30.87
37+	10.52	12.61	4.39	1.69	10.89	14.65	16.36	12.36	3.65	15.11	21.12	2.17	36.22	29.33	27.77	26.93	19.13	23.41
38+	4.44	4.12	1.75	2.22	4.45	2.99	3.06	6.36	5.50	5.79	5.89	8.80	22.08	18.51	20.90	15.48	17.94	16.04
39+	3.02	3.33	1.69	1.43	3.82	5.10	5.46	8.81	4.17	8.08	14.95	14.39	18.67	17.58	15.06	13.43	26.13	25.92
40+	2.09	3.47	3.54	2.81	3.42	1.60	4.16	2.49	3.74	4.31	3.96	8.00	9.43	9.51	8.32	7.44	19.89	11.15
41+	2.78	4.60	2.91	2.64	2.86	2.57	5.57	8.77	8.31	14.57	3.44	8.45	22.80	25.19	22.97	23.66	18.70	22.95
42+	3.37	2.66	2.03	2.60	6.92	4.50	8.05	5.72	3.00	5.41	5.14	3.47	12.77	14.82	13.14	10.56	14.35	13.54
43+	7.73	7.02	3.44	3.10	8.33	9.05	2.45	7.32	4.04	5.04	5.57	5.51	29.54	24.13	27.27	19.04	23.06	18.79
44	2.55	1.37	1.84	3.23	4.95	5.81	6.80	4.05	4.51	4.98	3.65	2.85	20.07	20.73	17.49	11.36	20.78	23.02
45+	0.69	0.64	0.91	0.67	1.35	1.74	1.44	1.32	1.38	1.36	1.06	1.45	12.21	7.59	8.71	6.29	9.12	7.55
46+	5.81	5.63	3.87	3.68	7.31	6.67	7.97	6.05	8.82	14.76	29.81	13.22	30.73	28.54	23.96	25.61	27.06	24.83
47	3.23	2.90	5.37	1.90	3.49	2.42	4.97	4.00	2.38	3.61	6.02	4.94	10.57	11.29	8.08	9.22	14.95	15.38
48+	2.37	2.10	1.46	1.12	2.57	2.87	4.01	8.76	3.77	5.07	6.04	7.14	34.25	33.05	19.72	20.72	20.83	18.46
49+	2.82	2.70	2.03	4.37	7.45	3.62	8.61	3.21	3.30	4.35	4.77	3.20	16.03	17.87	12.06	15.02	11.74	13.53
50+	3.06	3.79	2.63	4.86	4.52	4.06	8.56	8.30	4.37	7.77	12.65	9.35	18.48	23.71	12.95	17.62	13.69	16.44
51+	9.05	6.30	8.12	8.78	21.98	11.26	12.12	18.52	11.19	9.36	23.13	16.70	13.77	15.39	17.24	20.64	14.42	15.60
52+	1.31	1.82	5.00	5.29	4.44	4.40	3.53	1.81	3.66	2.11	10.10	9.28	12.32	10.36	11.12	8.27	8.58	8.55
53	2.75	2.55	5.72	3.37	15.35	7.20	5.37	4.92	6.13	6.92	9.02	11.19	28.17	38.42	21.46	34.52	27.01	23.19
54+	5.85	6.19	5.53	8.07	7.60	6.34	6.77	9.89	12.97	4.37	16.42	18.52	17.68	17.78	22.84	16.23	12.11	9.07
55+	1.91	1.70	1.57	1.28	2.79	3.61	1.92	1.50	2.10	2.02	7.24	4.00	3.86	20.66	12.48	10.24	6.99	7.97
56	4.30	4.07	5.32	8.05	5.60	6.94	9.91	10.11	8.51	4.88	11.52	14.00	14.90	14.62	14.08	10.97	10.89	13.99
57+	5.65	6.42	5.54	4.75	6.19	6.57	14.03	13.25	14.87	10.48	14.40	20.60	29.59	24.27	19.38	13.60	27.42	22.10
58+	2.17	1.81	1.24	2.07	3.41	4.06	2.02	5.11	6.02	6.15	2.42	3.11	18.64	19.85	13.11	18.10	34.79	14.47
59	4.64	3.08	4.94	4.89	3.20	3.02	4.86	3.87	5.16	5.05	3.60	4.25	18.86	22.14	9.35	11.40	10.04	11.06
60	2.49	3.77	1.30	1.27	4.56	2.62	5.82	7.67	6.86	7.02	8.30	8.33	42.93	27.52	38.35	18.67	44.43	37.55
61	9.40	11.36	11.14	11.81	7.72	13.36	14.84	19.85	10.11	14.90	12.22	12.72	54.30	37.95	33.31	26.88	27.22	44.07
62+	10.55	16.00	5.11	4.10	11.24	10.39	15.30	21.12	10.61	15.37	21.87	22.70	38.01	37.72	39.80	28.55	44.06	29.65
63+	10.31	10.84	5.17	7.07	10.55	11.43	5.98	7.13	9.14	11.37	20.20	19.88	16.89	19.65	17.78	23.31	11.63	16.47
64	1.82	3.42	0.93	1.00	1.42	1.68	1.85	1.61	1.47	1.60	2.63	6.14	16.20	14.56	5.78	6.34	11.67	10.99

R &LLRA = right and left lower rectus abdominis, R & LURA = right and left upper rectus abdominis, R & LEO1 = right and left anterior external oblique, R & LEO2 = right and left lateral external oblique, R &L EO3 = right and left posterior external oblique, R & LIO = right and left internal oblique, R & L13 = right and left longissimus at L1 level, R & LL16 = right and left iliocostalis at the L1 level, R & LL33 = right and left longissimus at the L3 level.

APPENDIX 2

Phase 1 and 3 Correlations (averaged Left and Right sides):

Phase 1 Correlations (both sides together)

		ABS/EXT P1	ABS/L13, L16, L33 P1	ABS/L13,L33, L52 P1	PC2 P1	PC3 P1
ABS/EXT P1	Pearson Correlation	1	.909**	.986**	.184	.892**
	Sig. (2-tailed)		.000	.000	.145	.000
	N	64	64	64	64	64
ABS/L13, L16,L33 P1	Pearson Correlation	.909**	1	.881**	.039	.840**
	Sig. (2-tailed)	.000		.000	.759	.000
	N	64	64	64	64	64
ABS/L13,L33,L52 P1	Pearson Correlation	.986**	.881**	1	.164	.867**
	Sig. (2-tailed)	.000	.000		.196	.000
	N	64	64	64	64	64
PC2 P1	Pearson Correlation	.184	.039	.164	1	.108
	Sig. (2-tailed)	.145	.759	.196		.396
	N	64	64	64	64	64
PC3 P1	Pearson Correlation	.892**	.840**	.867**	.108	1
	Sig. (2-tailed)	.000	.000	.000	.396	
	N	64	64	64	64	64

**. Correlation is significant at the 0.01 level (2-tailed).

Phase 3 Correlations (both sides together)

		ABS/EXT P3	ABS/L13, L16, L33 P3	ABS/L13,L33, L52 P3	PC2 P3	PC3 P3
ABS/EXT P3	Pearson Correlation	1	.907**	.984**	-.090	.906**
	Sig. (2-tailed)		.000	.000	.480	.000
	N	64	64	64	64	64
ABS/L13, L16,L33 P3	Pearson Correlation	.907**	1	.867**	-.280*	.870**
	Sig. (2-tailed)	.000		.000	.025	.000
	N	64	64	64	64	64
ABS/L13,L33,L52 P3	Pearson Correlation	.984**	.867**	1	-.021	.872**
	Sig. (2-tailed)	.000	.000		.870	.000
	N	64	64	64	64	64
PC2 P3	Pearson Correlation	-.090	-.280*	-.021	1	-.263*
	Sig. (2-tailed)	.480	.025	.870		.036
	N	64	64	64	64	64
PC3 P3	Pearson Correlation	.906**	.870**	.872**	-.263*	1
	Sig. (2-tailed)	.000	.000	.000	.036	
	N	64	64	64	64	64

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Phase 1 and 3 Correlations (averaged right vs averaged left sides):

Phase 1 Correlations (right and left sides)

		R ABS/EXT P1	R ABS/L13, L16,L33 P1	R ABS/L13, L33,L52 P1	L ABS/EXT P1	L ABS/L13, L16,L33 P1	L ABS/L13, L33,L52 P1	PC3 P1	PC2 P1
R ABS/EXT P1	Pearson Correlation	1	.949**	.984**	.791**	.708**	.802**	.878**	.055
	Sig. (2-tailed)		.000	.000	.000	.000	.000	.000	.665
	N	64	64	64	64	64	64	64	64
R ABS/L13, L16,L33 P1	Pearson Correlation	.949**	1	.916**	.736**	.728**	.729**	.871**	.002
	Sig. (2-tailed)	.000		.000	.000	.000	.000	.000	.990
	N	64	64	64	64	64	64	64	64
R ABS/L13,L33,L52 P1	Pearson Correlation	.984**	.916**	1	.789**	.695**	.815**	.853**	.072
	Sig. (2-tailed)	.000	.000		.000	.000	.000	.000	.574
	N	64	64	64	64	64	64	64	64
L ABS/EXT P1	Pearson Correlation	.791**	.736**	.789**	1	.936**	.981**	.813**	.270*
	Sig. (2-tailed)	.000	.000	.000		.000	.000	.000	.031
	N	64	64	64	64	64	64	64	64
L ABS/L13, L16,L33 P1	Pearson Correlation	.708**	.728**	.695**	.936**	1	.903**	.801**	.270*
	Sig. (2-tailed)	.000	.000	.000	.000		.000	.000	.031
	N	64	64	64	64	64	64	64	64
L ABS/L13,L33,L52 P1	Pearson Correlation	.802**	.729**	.815**	.981**	.903**	1	.801**	.226
	Sig. (2-tailed)	.000	.000	.000	.000	.000		.000	.072
	N	64	64	64	64	64	64	64	64
PC3 P1	Pearson Correlation	.878**	.871**	.853**	.813**	.801**	.801**	1	.108
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.000		.396
	N	64	64	64	64	64	64	64	64
PC2 P1	Pearson Correlation	.055	.002	.072	.270*	.270*	.226	.108	1
	Sig. (2-tailed)	.665	.990	.574	.031	.031	.072	.396	
	N	64	64	64	64	64	64	64	64

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Phase 3 Correlations (right and left sides)

		R ABS/EXT P3	R ABS/L13, L16,L33 P3	R ABS/L13, L33,L52 P3	L ABS/EXT P3	L ABS/L13, L16,L33 P3	L ABS/L13, L33,L52 P3	PC2 P3	PC3 P3
R ABS/EXT P3	Pearson Correlation	1	.961**	.984**	.765**	.727**	.768**	-.202	.900**
	Sig. (2-tailed)		.000	.000	.000	.000	.000	.110	.000
	N	64	64	64	64	64	64	64	64
R ABS/L13, L16,L33 P3	Pearson Correlation	.961**	1	.928**	.690**	.737**	.674**	-.306*	.917**
	Sig. (2-tailed)	.000		.000	.000	.000	.000	.014	.000
	N	64	64	64	64	64	64	64	64
R ABS/L13,L33,L52 P3	Pearson Correlation	.984**	.928**	1	.776**	.713**	.794**	-.119	.870**
	Sig. (2-tailed)	.000	.000		.000	.000	.000	.348	.000
	N	64	64	64	64	64	64	64	64
L ABS/EXT P3	Pearson Correlation	.765**	.690**	.776**	1	.891**	.978**	.174	.778**
	Sig. (2-tailed)	.000	.000	.000		.000	.000	.169	.000
	N	64	64	64	64	64	64	64	64
L ABS/L13, L16,L33 P3	Pearson Correlation	.727**	.737**	.713**	.891**	1	.857**	.173	.772**
	Sig. (2-tailed)	.000	.000	.000	.000		.000	.172	.000
	N	64	64	64	64	64	64	64	64
L ABS/L13,L33,L52 P3	Pearson Correlation	.768**	.674**	.794**	.978**	.857**	1	.166	.762**
	Sig. (2-tailed)	.000	.000	.000	.000	.000		.191	.000
	N	64	64	64	64	64	64	64	64
PC2 P3	Pearson Correlation	-.202	-.306*	-.119	.174	.173	.166	1	-.263*
	Sig. (2-tailed)	.110	.014	.348	.169	.172	.191		.036
	N	64	64	64	64	64	64	64	64
PC3 P3	Pearson Correlation	.900**	.917**	.870**	.778**	.772**	.762**	-.263*	1
	Sig. (2-tailed)	.000	.000	.000	.000	.000	.000	.036	
	N	64	64	64	64	64	64	64	64

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

APPENDIX 3

Kruskal-Wallis Test results: VAS comparisons

1. Military (DAN) vs. Non-Military (SARAH)

Descriptive Statistics

	N	Mean	Std. Deviation	Minimum	Maximum
VAS Initial	64	11.14903846	11.94934579	.000000	55.384615
VAS Final	64	14.28966346	14.67103820	.000000	57.692308
VAS Difference	64	3.14062500	11.35138994	-30.000000	32.000000
Sarah vs. Dan	64	.50	.504	0	1

Ranks

	Sarah vs. Dan	N	Mean Rank
VAS Initial	Sarah	32	32.77
	Dan	32	32.23
	Total	64	
VAS Final	Sarah	32	33.16
	Dan	32	31.84
	Total	64	
VAS Difference	Sarah	32	32.92
	Dan	32	32.08
	Total	64	

Test Statistics[a,b]

	VAS Initial	VAS Final	VAS Difference
Chi-Square	.013	.081	.034
df	1	1	1
Asymp. Sig.	.909	.776	.855

a. Kruskal Wallis Test

b. Grouping Variable: Sarah vs. Dan

2. PIT positive vs. PIT negative

Descriptive Statistics

	N	Mean	Std. Deviation	Minimum	Maximum
VAS Initial	64	11.14903846	11.94934579	.000000	55.384615
VAS Final	64	14.28966346	14.67103820	.000000	57.692308
VAS Difference	64	3.14062500	11.35138994	-30.000000	32.000000
PIT+ive vs. PIT-ive	64	.48	.504	0	1

Ranks

	PIT+ive vs. PIT-ive	N	Mean Rank
VAS Initial	Negative	33	31.32
	Positive	31	33.76
	Total	64	
VAS Final	Negative	33	29.52
	Positive	31	35.68
	Total	64	
VAS Difference	Negative	33	28.39
	Positive	31	36.87
	Total	64	

Test Statistics[a,b]

	VAS Initial	VAS Final	VAS Difference
Chi-Square	.278	1.779	3.389
df	1	1	1
Asymp. Sig.	.598	.182	.066

a. Kruskal Wallis Test

b. Grouping Variable: PIT+ive vs. PIT-ive

APPENDIX 4

Post Hoc Correlations for VAS (initial, final, and difference) and Phase 2 VAR 2 (ABS/L13, L16, L33) scores.

Correlations

		VAS Initial	VAS Final	VAS Difference	ABS/L13, L16, L33 P2
VAS Initial	Pearson Correlation	1	.654**	-.208	-.071
	Sig. (2-tailed)		.000	.099	.579
	N	64	64	64	64
VAS Final	Pearson Correlation	.654**	1	.604**	.037
	Sig. (2-tailed)	.000		.000	.770
	N	64	64	64	64
VAS Difference	Pearson Correlation	-.208	.604**	1	.123
	Sig. (2-tailed)	.099	.000		.335
	N	64	64	64	64
ABS/L13, L16,L33 P2	Pearson Correlation	-.071	.037	.123	1
	Sig. (2-tailed)	.579	.770	.335	
	N	64	64	64	64

**. Correlation is significant at the 0.01 level (2-tailed).

APPENDIX 5

Post Hoc ANOVA including *PIT*Occupational Activity*

Tests of Between-Subjects Effects

Dependent Variable: ABS/L13, L16,L33 P2

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	1.602[a]	41	.039	1.112	.425
Intercept	3.926	1	3.926	111.720	.000
DATAsource	.056	1	.056	1.585	.226
Age	1.336	30	.045	1.267	.314
Sex	.000	1	.000	.012	.915
PIT	.363	1	.363	10.315	.005
Reinjury	.036	1	.036	1.023	.327
Occupation	.218	4	.055	1.553	.235
PIT * Occupation	.172	3	.057	1.628	.222
Error	.562	16	.035		
Total	16.320	58			
Corrected Total	2.164	57			

a. R Squared = .740 (Adjusted R Squared = .074)