# COMPARING TRUNK NEUROMUSCULAR MEASURES TO A CLINICAL BATTERY OF TESTS IN A RECOVERED LOW BACK INJURED POPULATION

by

Raymond Daniel Trudel

Submitted in partial fulfillment of the requirements

for the degree of Master of Science

at

Dalhousie University

Halifax, Nova Scotia

August 2014

# TABLE OF CONTENTS

LIST OF TABLES	v
LIST OF FIGURES	vi
ABSTRACT	viii
LIST OF ABBREVIATIONS USED	ix
GLOSSARY OF TERMS	X
ACKNOWLEDGEMENTS	xii
CHAPTER 1 INTRODUCTION	1
1.1 PURPOSE	11
1.2 OBJECTIVES	11
1.3 HYPOTHESIS	12
1.4 ASSUMPTIONS	13
CHAPTER 2 BACKGROUND LITERATURE	14
2.1 recurrence and recovery	14
2.1.1 Recurrence	15
2.1.2 Recovery	16
2.2 SPINAL STABILITY	18
2.2.1 Theory	18
2.2.2 Assessment	25
2.3 STRENGTH	27
2.4 ELECTROMYOGRAPHY	29
2.4.1 Theory	29
2.4.2 Principal Component Analysis	31
CHAPTER 3 METHODOLOGY	35
3.1 RESEARCH DESIGN	35
3.2 SUBJECTS	35
3.3 PROCEDURE	40
3.4 NORMALIZATION PROCEDURE	43
3.5 MOTION MEASUREMENT	44

3.6 EXPERIMENTAL TRIALS	45
3.7 STRENGTH	47
3.8 EMG PROCESSING AND ANALYSIS	48
3.9 FOB PROCESSING AND ANALYSIS	49
3.10 STRENGTH PROCESSING AND ANA	LYSIS 49
3.11 STATISTICAL ANALYSIS	50
CHAPTER 4: COMPARING TRUNK MUSCLE EI	LECTROMYOGRAPHIC
PATTERNS AND STRENGTH BETWEEN THOSE	E RECOVERED FROM A LBI
WITH CLINICAL INSTABILITY AND THOSE W	ITHOUT51
4.1 INTRODUCTION	51
4.2 METHODOLOGY	55
4.2.1 Participants	55
4.2.2 Test procedure	57
4.2.3 Normalization procedure	60
4.2.4 Surface EMG data collection and p	rocessing 60
4.2.5 Motion capture and processing	63
4.2.6 Strength	64
4.2.7 Statistical analysis	64
4.3 RESULTS	65
4.3.1 Demographic, Motion and timing v	variables 65
4.3.2 EMG waveform analysis	67
4.3.3 Strength	77
4.4 DISCUSSION	
4.6 CONCLUSION	82
CHAPTER 5: COMPARING TRUNK MUSCLE EI	LECTROMYOGRAPHIC
PATTERNS IN A RECOVERED LBI POPULATIO	ON BETWEEN THOSE WHO
TESTED POSITIVE OR NEGATIVE ON THE PRO	ONE INSTABILITY TEST 83
5.1 INTRODUCTION	83
5.2 METHODOLOGY	85
5.2.1 Participants	85
5.2.2 Test procedure	87

5.3 RESULTS	87
5.3.1 Demographic and Motion data	87
5.3.2 EMG waveform analysis	89
5.3.3 Strength	96
5.4 DISCUSSION	96
5.5 CONCLUSION	98
CHAPTER 6 SUMMARY AND CONCLUSIONS	100
6.1 SUMMARY	100
6.2 LIMITATIONS	103
6.3 IMPLICATIONS	105
6.4 FUTURE RESEARCH	105
6.5 CONCLUSION	106
REFERENCES	108
APPENDIX 1 Comparison of articles using LBP/LBI	119
APPENDIX 2	122

# **LIST OF TABLES**

Table 4.1: Definition of clinical stability tests	59
Table 4.2: Subject demographics split by clinical instability grouping	66
Table 4.3: Trunk and pelvic motion data	67
Table 4.4: Mean PC scores for the abdominal muscles, by group and muscle	71
Table 4.5: Mean PC scores for the back muscles, by group and muscle	71
Table 4.6: Results for the group by muscle ANOVAs	72
Table 4.7: Post hoc results for muscle main effects	74
Table 4.8: Post hoc results of the group by muscle interactions	76
Table 4.9: Strength variables	77
Table 5.1: Subject demographics data split by results of prone instability test	87
Table 5.2: Trunk and pelvic motion data	88
Table 5.3: Mean PC scores for PIT postive vs. PIT negative	92
Table 5.4: Mean PC2 scores for the back extensors separated by group and muscle	92
Table 5.5: Results for the group by muscle ANOVAs	92
Table 5.6: Post hoc results for muscle main effects	95
Table 5.7: Post hoc results of the group by muscle interactions	96
Table 5.8: Strength variables	96

# **LIST OF FIGURES**

Figure 2.1: Visual representation of the spinal stability model	21
Figure 3.1: Detailed breakdown of all referrals	37
Figure 3.2: Recruitment flow chart for LBI participants	38
Figure 3.3: Flow chart of the experimental procedure.	41
Figure 3.4: Electrode placement	43
Figure 3.5: Experimental set up and participant position for the transfer task	46
Figure 3.6: Patient positioning for strength measure of lumbar extension	47
Figure 3.7: Schematic representation of the PCA procedure.	49
Figure 4.1: Classification of groups and breakdown of participants	56
Figure 4.2: Schematic representation of the PCA procedure	63
Figure 4.3: EMG waveforms for abdominal muscles during the transfer task	68
Figure 4.4: EMG waveforms for back extensor muscles during the transfer task	69
Figure 4.5:	
(a) Principal components for PC1	
(b) High low waveforms for PC1	
(c) PC1 scores for abdominal muscle interactions.	
(d) Principal components for PC2	
(e) High low waveforms for PC2	
(f) PC2 scores for back muscle interactions	73
Figure 4.6: Correlation scatterplots between PC1 and EMG RMS values for:	
(a) The abdominals	
(b) The back extensors	75

Figure 5.1: Classification of groups and breakdown of participants
<b>Figure 5.2:</b> EMG waveforms for abdominal muscles during the transfer task90
<b>Figure 5.3:</b> EMG waveforms for back extensor muscles during the transfer task91
Figure 5.4:
(a) Principal components for PC1
(b) High low waveforms for PC1
(c) PC2 scores for back muscle interactions.
(d) Principal components for PC2
(e) High low waveforms for PC294

#### ABSTRACT

#### **Introduction:**

The purpose of this study was to determine differences in trunk muscle activation patterns in recovered LBI groups classified as unstable or not. EMG from comprehensive sets of trunk muscles were compared during a controlled task between groups separated using a Modified Hicks's protocol.

#### **Methods:**

32 LBI participants who were deemed recovered were recruited from CFB Halifax within 12 weeks post-injury. EMG from trunk muscles was recorded during a standardized lift-and-replace task.

#### **Results:**

Significant group\*muscle interaction effects were found for amplitude and temporal EMG patterns. Post hoc findings showed fewer between-muscle differences in the unstable group.

#### **Conclusions:**

The unstable group utilized strategies for increased active stiffness through synergistic co-activation and altered temporal responses and indicates a "bracing" strategy. These findings are consistent with the hypothesis that passive stiffness decreases are compensated by the neuromuscular system. Furthermore they provide initial evidence linking clinical tests for instability to objective physiologically-based measures.

## LIST OF ABBREVIATIONS USED

EMG Electromyography

CNS Central nervous system

MUAP Motor unit action potential

LBI Low back injury

LBP Low back pain

VAS Visual analogue scale

RMQ Roland Morris questionnaire

PCS Pain catastrophizing scale

CFB Canadian Forces base

PCA Principal Component analysis

PC Principal Component

CPR Clinical prediction rule

PSLR Passive straight leg raise

MVIC Maximum voluntary isometric contraction

URA Upper rectus abdominus

LRU Lower rectus abdominus

EO1 External oblique (anterior fibers)

EO2 External oblique (lateral fibers)

EO3 External oblique (posterior fibers)

IO Internal oblique

ANOVA Analysis of variance

#### **GLOSSARY OF TERMS**

**Stiffness:** The ratio of the change in force to the change length of a structure is its stiffness and the reciprocal is called compliance. A muscle that has greater stiffness (or less compliance) will resist a perturbation more effectively.

Active stiffness: This is stiffness provided by the active components (muscles) of the system and is a function of muscle activation, reflex behaviors and the mechanical characteristics of the muscles. Depending on the level of muscle activation, different components of the muscle will provide greater resistance to stretch. Therefore, active stiffness is not just a function of muscle strength.

Passive stiffness: This is stiffness provided by the passive components of the system. Passive stiffness has a contribution from the muscles as well as the non-contractile components of a joint (i.e. ligaments, joint capsules, and bony structures). The passive components of the muscle are from an elastic component in series with the contractile component (tendons and intramuscular components such as attached cross bridges) and a parallel elastic component (intramuscular connective tissue).

**Spinal stability:** The spines ability under normal physiologic loads to limit patterns of displacement in order to not damage or irritate the spinal cord and nerve roots and to prevent incapacitating deformity or pain caused by structural changes (White, Johnson, Panjabi, & Southwick, 1975). Also defined as the capacity of the vertebrae to remain aligned and to preserve the normal displacements in all physiological body movements (Kirkaldy-Willis, 1985).

**Spinal instability:** The loss of the ability of the spine under normal physiologic loads to maintain its patterns of displacement so there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain (White & Panjabi, 1978). The loss of stiffness leading to abnormal and increased movement in the motion segments (Pope &

Panjabi, 1985). With instability, movement can be abnormal in quality (abnormal coupling patterns) and/or quantity (increased motion).

**Low back pain (LBP):** Defined as pain located between the lower ribs and the gluteal folds.

Low back injury (LBI): Any event or occurrence that leads to pain between the lower ribs and the gluteal folds, and/or results in a decrease in function or an increase in disability such that the person cannot continue with their usual activities.

**Non-specific LBP:** Defined as pain not attributed to a recognizable pathology. A diagnosis of non-specific LBP is dependent on the clinician being satisfied that there is not a specific cause for their patients' pain (all specific causes of LBP have been ruled out or excluded) (Manusov, 2012; National Collaborating Centre for Primary Care (UK), 2009).

#### **ACKNOWLEDGEMENTS**

To begin, I would like to acknowledge that the Department of National Defence and the Surgeon General's Health Research Program funded this study.

For the individuals involved with this project I would like, first and foremost, to express my sincere gratitude to my supervisor, Dr. Cheryl Kozey for her patience, knowledge, motivation and guidance during this process. She always kept me on point and her attention to detail hopefully made me a better writer and researcher, she made this an amazing learning experience.

I would also like to thank the other members of my supervisory committee; Dr. Janice Moreside for her thoughts, suggestions and support, and Dr. Anne Fenety for her support, guidance and for convincing me that Dalhousie was the right choice.

This project could not have been completed without the help of everyone in the Neuromuscular Functions lab therefore, thank you to Amir, Warren and Colin for helping with data collections and everything else that was going on. A special thanks is required for Adam, whose computer expertise and expansive knowledge in the lab made things run smoothly and allowed us to trouble shoot any glitches that arose.

A thank you is also required for everyone in the DOHM lab for giving me different perspectives with all our Cabals; it was nice to think about different topics from time to time.

Not many clinicians get the chance to take time away from work to complete a Masters degree. Therefore, for this opportunity, I would like to thank LCol Peter Rowe for supporting my application for the PGTP and being my sponsor, your insight and help with the process was much appreciated.

This would not be complete without thanking my family. Mom and Dad for helping out with anything and everything, you have always been there for me and I can't express how thankful I am for that. To my brother for always lending an ear during my times of frustration. Especially to Amanda for not only putting up with me over these past two years, but giving me the best two gifts that anyone could ever ask for, words are not capable of expressing how much your support and friendship has meant to me, I love you. To Sophie, you always managed to bring a smile to my face no matter how busy I was and finally to my baby boy (on the way), even though you are not here yet you and your sister have brought joy to my life.

Without everyone's support and encouragement I could never have completed this thesis, thank you.

#### CHAPTER 1 INTRODUCTION

Why is the majority of low back pain (LBP) research focused on chronic LBP? It could be because one third of all chronic pain in Canada is attributed to the low back (Schopflocher, Taenzer, & Jovey, 2011) or because chronic musculoskeletal disorders are the second costliest category of illness in the country that also results in a significant amount of disability (Mirolla, 2004). While these numbers are disturbing, by utilizing our resources to study the end state, chronic LBP, we are being reactive to the problem by providing tertiary prevention (Mirolla, 2004) i.e. controlling a condition that has already developed. Presumably it would be more efficient and cost effective to be proactive and deal with low back injuries (LBI) before they become chronic, thereby providing either primary prevention (preventing LBI from occurring) or secondary prevention (early detection and interruption of progression) (Mirolla, 2004). It has been shown that recurrent LBI is a major factor for the development of a chronic state (Wasiak et al., 2009), therefore identifying factors that can predict recurrence of LBI could be used for earlier detection and potentially prevent repeat injuries from progressing to chronic disability.

Recurrent LBI, although understudied, is still a draw on health care resources and causes significant disability with four out of five people experiencing at least one episode that results in LBP, defined as pain between the lower ribs and the gluteal folds, at some point in their life (Dagenais, Caro, & Haldeman, 2008; Stanton, Latimer, Maher, & Hancock, 2009). While it is often claimed that 90% of LBP episodes resolve spontaneously within one month, a wide range (24% to 84%) of re-injury incidence within one year have been reported (Delitto et al., 2012; Hestback, Leboeuf-Yde, &

Manniche, 2003; Pengel, Herbert, Maher, & Refshauge, 2003; Stanton et al., 2008; Stanton et al., 2009). The concept of recurrence is difficult as it is not clear whether a predisposing condition exists or whether incomplete recovery leaves individuals vulnerable to re-injury (Butler, Hubley-Kozey, & Kozey, 2012; Cholewicki et al., 2005) highlighting a difficulty with the terminology currently found in the literature. Thus the large reported variation in recurrence could be due to differing ideas of what constitutes recurrence, where common convention believes true recurrence requires that the patient has firstly recovered from the original episode and then experiences a new episode of LBP (Stanton et al., 2009). Another issue identified with terminology is the use of LBP versus LBI, with the former focusing on the symptom, pain and not on the actual injury/damage to structures, which is implied with the term LBI. When examining the literature, no clear rationale was found in articles for using the terms LBP or LBI; in fact many articles used the terms interchangeably. Additionally, many articles did not provide a definition of what LBP or LBI was instead using it as a blanket term and trying to further subdivide it into categories such as acute or chronic (for example, defining chronic LBP as LBP lasting greater than 12 weeks (Cedraschi, Nordin, Nachemson, & Vischer, 1998)). Appendix 1 outlines examples of articles using LBP, LBI or both and provides the included definitions. This inconsistent use of terms could also lead to the variation in reported recurrence rates, as pain is only a symptom that is not necessarily always present during an injury. Additionally, someone could experience pain in the early phases of an injury that resolves while other deficiencies associated with the injury remain. An example of this is seen when using ICF body-structure codes associated with LBP. Code s7601 is for Muscles of the Trunk and while someone may have inappropriate

muscle responses while lifting, the underlying problem is still there whether the person experiences pain or not. Therefore, although pain may be associated with an injury, its presence or absence does not represent all aspects of said injury and caution should be exercised when pain is the sole metric used to identify a LBI. This study will use both terms, with LBP describing the symptom of pain and LBI referring to an event or injury that results in a deficiency in one of the many structures contained in the back, understanding that this term is used interchangeably in the literature.

The issues identified above with defining recurrence are mirrored when trying to define and measure recovery. Multiple tools have been developed to measure different aspects of LBI, but what construct is actually being measuring and whether this can predict recovery is different for each tool. Some tools, such as functional capacity evaluations (FCEs), consider the ability to complete certain functional tasks and ability to return to work as a favorable outcome. It is postulated that these outcomes predict successful return to, and sustained activity at work (Gross & Battie, 2004; Gross, Battie, & Cassidy, 2004). Unfortunately, multiple studies and reviews of FCEs have determined that these instruments only add modestly to the prediction of initial return to work beyond that provided by other prognostic factors including floor-to-waist lift, self-rated pain and disability measures (Gross & Battie, 2004; Gross et al., 2004; Innes & Straker, 1999b; Matheson, Isernhagen, & Hart, 2002). Additionally, it was shown that FCEs have no ability to predict sustained recovery with the reported results actually contrary to FCE's theoretical basis (Gross & Battie, 2004). It was found that those with more failed tasks on the FCE had a lower risk of recurrence while those with less failed tasks had a higher risk, exactly opposite to what the test predicts, calling into question the validity. Other

measures that are frequently used in clinics are self-report measures of disability or pain such as the Roland Morris Questionnaire (RMQ) and the Visual Analogue Scale (VAS). They assume that a successful outcome of treatment, seen in a decreased score on the measure, correlates with recovery and consequently a low recurrence rate (Resnik & Dobrzykowski, 2003; Roland & Morris, 1983). This assumption is unsubstantiated as no longitudinal studies have been completed that attempt to predict the recurrence of injuries with these outcome measures. Therefore, even though there are multiple measures that are frequently employed in the clinical setting in an attempt to measure recovery, there is no conclusive evidence that they can accurately determine who has recovered from a LBI or who will have a recurrence.

The deficiencies noted in these clinical measures could be a consequence of the mostly subjective nature of the measures or their reliance on outcomes. They do not attempt to address potential physiological or mechanical alterations that could contribute to an underlying pathology. These alterations can be assessed through objective physiological based measures. One such measure that has been investigated in low back research is strength where not only has sufficient muscle strength and endurance been shown to be required to maintain proper function, but specific deficiencies have been shown in different low back disorders (Davarian, Maroufi, Ebrahimi, Farahmand, & Parnianpour, 2012; Descarreaux, Blouin, & Teasdale, 2004; Gruther et al., 2009; Mannion, Taimela, Muntener, & Dvorak, 2001; McGill, Childs, & Liebenson, 1999). Specifically, muscle strength was diminished in both a chronic LBP population and one with clinical instability (Alston, Carlson, Feldman, Grimm, & Gerontinos, 1966; Davarian et al., 2012; Descarreaux et al., 2004; Gruther et al., 2009; Mannion et al.,

2001). Another way that strength has been used in the literature was to examine the strength ratio between the trunk flexors and extensors. Although strength ratios have been explored in LBP research, no consensus has been reached when reporting differences between a LBP and a control population (Alston et al., 1966; Gruther et al., 2009; Holmstrom, Moritz, & Andersson, 1992a; Shirado, Kaneda, & Ito, 1992).

Other objective physiological based measures have shown potential for predictive ability including electromyography (EMG). Heydari et al. have shown that objective neuromuscular measures using EMG are capable of predicting who will have a first incidence of LBI regardless of history of LBI (Heydari, Nargol, Jones, Humphrey, & Greenough, 2010). Additionally, two independent research groups (Butler et al. and MacDonald et al.) have shown that EMG measures can also differentiate between those individuals that had a LBI but are deemed recovered and those that never had a LBI (Butler et al., 2012; MacDonald, Moseley, & Hodges, 2009; MacDonald, Moseley, & Hodges, 2010; Macdonald, Dawson, & Hodges, 2011). In follow-up work by Hubley-Kozey et al. that built on these results, not only could they show who had a LBI but also they provided modest evidence that differences exist for those who have a recurrent LBI at one-year follow-up (Hubley-Kozey, Moreside, & Quirk, 2013.). In summary, objective neuromuscular measures using EMG have shown the ability to not only differentiate between LBI and no LBI but also predict who will have a recurrence of LBI.

As more research is completed in the domain of LBP it is becoming evident that the term LBP constitutes a large heterogeneous group, and as such it is believed that there are distinct subgroups (Delitto et al., 2012). While groups, such as discogenic back pain or stenotic LBP have specific identifiable causes and are relatively easy to identify others

such as non-specific LBP due to spinal instability are less evident, possibly due to the multiple different structures that are identified as contributing to achieving and maintaining spinal stability (Panjabi, 1992a). These structures and the interplay among them are similar across multiple different spinal stability models that all describe three different interdependent components that are necessary to maintain a stable spine (Bergmark, 1989; Cholewicki & McGill, 1996; McGill & Cholewicki, 2001; Panjabi, 1992a). The different components identified in all models can be grouped into three subsystems, an active (muscular), a passive (osteoligamentous) and a nervous (control) subsystem. Currently there are difficulties in diagnosing spinal instability, but there are multiple working models that all agree on the components that are necessary to maintain spinal stability.

One of the above-mentioned models, by Panjabi, utilizes terminology that is more clinically relevant. This is useful when dealing with injuries as it allows stability to be viewed on a continuum instead of an absolute as in the mechanical models, which are either stable or unstable (Bergmark, 1989). Panjabi describes a passive subsystem consisting of the ligaments, bones and connective tissues of the spine that provides increasing stiffness and hence stability to the spine, the closer it is to its maximum range of motion (Brown, Vera-Garcia, & McGill, 2006; Panjabi, 1992b; Panjabi, 2003). The active subsystem, made up of the contractile tissues, provides stability to the spine during all ranges by contracting in response to different postures or loads applied to the spine (Bergmark, 1989; Brown et al., 2006; Granata & Wilson, 2001; Hodges et al., 2003; Panjabi, 1992a; Panjabi, 2003). To effectively respond to these loads and postures, the neural subsystem receives input from the other two subsystems and formulates an

appropriate response (Panjabi, 1992a). The resulting patterns of muscle contractions are the response to the interplay among all three subsystems to the specific demands put on the spine demonstrating that no one subsystem could achieve adequate stability in isolation (Hodges et al., 2003; Hubley-Kozey et al., 2013.; Panjabi, 1992a).

This close interaction of all subsystems is most evident when one is deficient. As the contribution of one subsystem to spinal stability is impaired, it is proposed that the others develop compensatory mechanisms in an attempt to maintain stability (Marras & Granata, 1997; Panjabi, 1992a), and research has shown that there are indeed differences in the subsystems in individuals that have a LBI (Brown et al., 2006; Butler, Hubley-Kozey, & Kozey, 2009; Butler et al., 2012; Hubley-Kozey et al., 2013.; MacDonald et al., 2010). Although it may appear that stability is maintained, different methods have been developed to determine if deficiencies exist in the different subsystems. For the passive subsystem clinical tests, radiographs or movement assessments have been employed with varying success (Hicks, Fritz, Delitto, & McGill, 2005; Kotilainen & Valtonen, 1993; Panjabi, 2003). A 2012 review of the literature states that a diagnosis of clinical instability can reliably be made with a reasonable level of certainty when employing the Hicks prediction rule where the patient presents with at least three of the following clinical findings(Delitto et al., 2012; du Bois et al., 2011; Fritz, Brennan, Clifford, Hunter, & Thackeray, 2006; Hicks et al., 2005; Reme, Hagen, & Eriksen, 2009):

- i) Average SLR ROM >91°
- ii) Positive prone instability test
- iii) Positive aberrant movement during lumbar flexion, and
- iv) Age <40 years.

Since this rule has only been used with a symptomatic population, whether it would be as effective in a recovered population is unknown. Subsequently, when examining the specific tests, the only one that is expected to yield the same result in both a symptomatic and a recovered population is the prone instability test (Biely, Silfies, Smith, & Hicks, 2014; Magee, 1997; Rabin, Shashua, Pizem, Dickstein, & Dar, 2014). For the active subsystem, different measures of muscle function (strength and strength ratios) have been tested but have not been able to demonstrate when a problem exists (Mannion et al., 2001; McGill et al., 1999), and are unable to come to a consensus on what deficiency exists (Bayramoglu et al., 2001; da Silva, Arsenault, Gravel, Lariviere, & de Oliveira, 2005; Descarreaux et al., 2004; Iwai, Nakazato, Irie, Fujimoto, & Nakajima, 2004; Sjolie & Ljunggren, 2001). Even though consensus is lacking when looking exclusively at the active subsystem, when looking at the combination of the active and neural subsystems, EMG has demonstrated the capability to determine if someone has a LBI and more recently, modest evidence shows if they will have a recurrence (Butler et al., 2009; Cholewicki et al., 2005; Heydari et al., 2010; Hodges & Richardson, 1996; Hubley-Kozey et al., 2013.; MacDonald et al., 2009; MacDonald et al., 2010; Macdonald et al., 2011). Although methods have been developed to determine if there are deficiencies in the individual subsystems, there is not one method that can test all three. Therefore, a combination of methods may produce the most effective way to determine if a spinal instability exists.

Even though clinical tests can determine if someone has a deficient passive subsystem, they have not been able to determine if a person recovered from a LBI is at risk of recurrence. There is some evidence that objective EMG recordings have the ability

to accurately determine who had a LBI as well as determine who will have a recurrence of a LBI (Butler et al., 2012; Heydari et al., 2010; Hodges & Richardson, 1996; Hubley-Kozey et al., 2013.; MacDonald et al., 2009; MacDonald et al., 2010; Macdonald et al., 2011). To achieve this, different groups have employed different methods, from examining onset times and amplitudes of one muscle in response to a disturbance (Cholewicki et al., 2005; MacDonald et al., 2009; MacDonald et al., 2010; Macdonald et al., 2011), to examining muscle activation patterns of a comprehensive group of muscles during a functional task(Hubley-Kozey, Moreside, & Quirk, 2014a). Aside from the fact that modeling evidence suggests that all trunk muscles are important (Cholewicki & VanVliet, 2002) analysis of a comprehensive group of muscles is a more objective method and mitigates some of the subjectivity of choosing which specific muscles to measure, while providing a more complete picture.

Although analysis from a comprehensive group on muscles is the preferred objective method for EMG analysis of the lumbar spine, it presents a logistical issue with the amount of data it creates. To effectively manage this data, reduction techniques can be employed (Jackson, 2003; Landry, McKean, Hubley-Kozey, Stanish, & Deluzio, 2007). One approach based on pattern recognition techniques, Principal Component Analysis (PCA) has been shown to reduce the number of variables while still maintaining important features of the original data (Butler et al., 2009; Hubley-Kozey & Vezina, 2002; Landry et al., 2007). Just as using a comprehensive group of muscles increases objectivity, so does using PCA as it analyzes the entire waveform and assigns scores based on the variation in the data. This allows the most relevant features of the data to be uncovered(Jackson, 2003). This method is contrasted by the use of discrete parameters

such as co-contraction indices (Brown et al., 2006) or onset times (Cholewicki et al., 2005; MacDonald et al., 2009) in an EMG waveform that have been selected a priori as representing an important metric and are therefore more subjective (Brandon et al., 2013; Landry et al., 2007). Furthermore it allows for the examination of both amplitude and temporal patterns from waveforms simultaneously, thus allowing for the quantification of co-activation and muscle synergies/coordination among many muscle sites. In summary, by utilizing both a comprehensive group of trunk muscles and applying PCA to the EMG data we are employing an objective method of analyzing the neuromuscular components of a task allowing us to accurately identify the potential source of the problem.

It has already been stated that LBP is a heterogeneous group and work is being done to effectively identify more homogeneous subgroups, which would aid in more successful assessment and treatment. Part of the difficulty that the clinical community has had with this is in the clinical tests that are used to assess and classify LBP. Traditionally, clinical tests have been more symptom or outcome based, which may be sufficient for determining the immediate problem while the person is symptomatic. The problem that arises is that those tests have not been examined for their ability to detect underlying issues that could predict recurrence or recovery. Objective physiologically based measures, on the other hand, focus more on specific deficiencies and research has been completed on their ability to detect differences between groups and also for recurrence and re-injury. Ideally, tests used clinically would have the best of both of these; the ability to identify symptoms as well as underlying problems that could lead to re-injury or define objectively when someone is recovered. To achieve this, comparative analysis could be done between both types of measures to obtain the optimal combination;

whether it consists solely of the objective tests or a mix with the clinical ones also.

Therefore, the overall goal of this thesis was to better understand the relationship between clinical tests of spinal instability and an objective physiological-based test of neuromuscular function including trunk muscle activation patterns and abdominal and back extensor strength variables in those deemed recovered from a low back injury.

# 1.1 PURPOSE

This study aims to build on previous knowledge by comparing objective physiological and biomechanical based measures to clinical tests for spinal instability. This was achieved by capturing surface electromyograms from a comprehensive set of trunk muscles sites during a controlled dynamic task and comparing the resulting patterns to a standardized clinical assessment that includes multiple tests for spinal instability.

#### 1.2 OBJECTIVES

The main specific objective was:

- 1. To determine whether there are differences in trunk muscle activation patterns during a standardized functional task in those deemed recovered from a LBI when they are classified as having a clinical instability versus those that do not. The three sub objectives are:
  - 1.1. To determine if there are differences in the isometric torque of trunk flexion and extension between those with clinical instability and those without.
  - 1.2. To determine if there is a difference in the ratio of abdominal to back isometric torque between those with clinical instability and those without.

1.3. To determine if there is a difference in EMG patterns between those deemed recovered from a LBI with a positive prone instability test (PIT) and those with a negative PIT.

#### 1.3 HYPOTHESIS

The main hypothesis of this research is that those with clinical instability will have different trunk neuromuscular patterns compared to those with no instability while performing a highly controlled function task that has dynamic external moments. More specifically, it is hypothesized that those with instability have higher antagonist/agonist co-activation during the transfer task compared to those without instability and they have more sustained activity i.e. less response to changing external moments (flexion and lateral flexion). Additionally, it is hypothesized that those with clinical instability have lower back muscle strength and consequently a higher abdominal to back strength ratio. Since the PIT is one component of the protocol used to define clinical instability and it tests the ability of the active subsystem to compensate for a deficient passive subsystem, it is hypothesized that those with a positive PIT will have different neuromuscular patterns compared to those that are negative. Specifically, it mirrors what is hypothesized for clinical instability in that it is hypothesized that those that are PIT positive have higher antagonist/agonist co-activation during the transfer task compared to those that are negative and they have more sustained activity i.e. less response to changing external moments (flexion and lateral flexion).

#### 1.4 ASSUMPTIONS

The assumptions of this study are:

- That minimal movement will occur between the electrodes and the skin during the EMG recordings.
- The same electrode placement will be used on each participant and therefore the same muscles will be sampled.
- That fatigue will not be present during the testing.
- Participants with LBI are able to exert their maximal voluntary isometric contractions during the normalization exercises.
- No significant learning effect will occur between trials (this is controlled for by using familiarization sessions, practice sessions prior to testing and practice trials).

This thesis is comprised of 6 chapters. Chapter 1 provides the introduction and outlines the purpose, objectives and hypothesizes. Chapter 2 examines and synthesizes the relevant background literature. Chapter 3 provides the detailed general methodology for the entire study. Chapter 4 is a self-contained paper written in journal format that specifically addresses the main objective comparing the muscular activation patterns and strength between a group testing positive for clinical instability and another that is negative. Chapter 5 addresses the sub-objective examining the neuromuscular patterns between a group that had a positive PIT and one that was negative. While Chapters 4 and 5 include conclusions for their specific objectives, Chapter 6 provides a general summary and conclusions for the entire study.

#### **CHAPTER 2 BACKGROUND LITERATURE**

The body of literature examining issues related to LBI/LBP is extensive, therefore this proposal will endeavor to review those aspects that are relevant to the objectives and hypothesis for this proposal. A key aspect in this study is that all participants are part of a sub-acute cohort of LBI that are deemed recovered before testing. This background literature review will examine current issues encountered with defining recovery and recurrence and how it was defined for this project. Another concept that requires defining and is a common thread in all objectives is spinal stability, what it is and how it is measured. Since this study is using the idea of clinical spinal instability to differentiate the participant groups, how it is defined will be examined. Another common thread in all the objectives is that of EMG, therefore what it is and it's relevance to the LBI literature will also be examined.

#### 2.1 RECURRENCE AND RECOVERY

The frequency of LBP in modern society has been referred to as an epidemic and reports in the literature consistently support this view (Delitto et al., 2012). Not only is the one year incidence of any episode of LBP as high as 36% (Delitto et al., 2012), current research suggests that LBP is typically recurrent with up to 62% of individuals who have an episode of LBP suffering a recurrence within one year (Maetzel & Li, 2002). During the past decade, scientific literature has recognized the difficulty in diagnosing and treating lower back disorders. This partly stems from treating people with LBP as a homogeneous group when it is a heterogeneous mix of many disorders (Fritz & George, 2000; Fritz et al., 2006; Hicks, Fritz, Delitto, & Mishock, 2003). Recently, studies have focused on creating pathways or criteria that would separate this large group

into homogeneous subgroups for proper diagnosis and treatment (Delitto et al., 2012; Fritz & George, 2000; Fritz et al., 2006; Hicks et al., 2003). One large subgroup of recurrent low back injury (LBI) is those classified with spinal instability and it is this group that is the focus of this paper.

#### 2.1.1 Recurrence

While the incidence and prevalence of LBP in general is relatively easy to find, the incidence of spinal instability is more difficult to determine partially due to the lack of an accepted definition. Estimates of the percentage of patients with LBP arising as a consequence of spinal instabilities range from 15% to 30% of all patients with LBP (O'Sullivan, 2000; Pitkanen, Manninen, Lindgrer, Turunen, & Airaksinen, 1997; Twomey & Taylor, 2000). This is a relatively wide range and how well it truly represents the number of individuals is difficult to determine due to the multiple definitions of spinal instability. This will be further discussed in the background literature section on spinal stability. The concept of recurrence of LBI faces similar difficulties as spinal instability in that it lacks a universally agreed upon definition or gold standard. A true definition of recurrence needs to be differentiated from both persistence and/or a flare up of the original episode (Stanton et al., 2009). To achieve this, it has been suggested that recurrence requires that the individual has both fully recovered from the original episode and subsequently experiences a new and separate LBI. Stanton et al. suggests that a definition of recurrence needs to include operational definitions for both recovery and commencement of a new episode (Stanton et al., 2009).

## 2.1.2 Recovery

Recovery, while seemingly a simple concept is actually fairly complex. It has been proposed that an individual have a minimum recovery period of 30 days where they are pain free (de Vet et al., 2002). While this definition could be useful, it puts undue emphasis of recovery on a singular metric in pain that may not adequately define recovery. It has therefore been suggested that other measures of disability or function be used to have a more robust definition of what recovery is (Stanton et al., 2009). An examination of different tools that claim to measure disability or function and if they can predict recovery shows that the evidence is lacking. Tools that are frequently used in clinics are self-report measures, with two of the most popular being the RMQ and VAS. These questionnaires have been shown to be reliable and valid when looking at reduction in the symptoms of LBI and/or an increase in function (Jensen, Strom, Turner, & Romano, 1992; Resnik & Dobrzykowski, 2003; Roland & Fairbank, 2000), but they have not been able to predict recovery or recurrence. These measures assume that a decrease in score and disability correlates with recovery (Resnik & Dobrzykowski, 2003; Roland & Morris, 1983). Even though this is an assumption, and many clinicians use this measure in discharge planning, it is unsubstantiated as no studies have been performed that attempt to tie these measures to actual recovery.

Another tool that is frequently used to make determinations for return to work and recovery are Functional Capacity Evaluations (FCEs). These standard assessments of function consider the ability to complete certain work simulated tasks as indicative of recovery and therefore predictive of return to, and sustained activity at, work (Gross & Battie, 2004; Gross et al., 2004). Unfortunately there is limited research looking at the

reliability of the subsections of these tools with no studies examining the reliability of the tool as a whole (Innes & Straker, 1999a). While there are some studies that examined subsections of FCEs, many used samples that included only non-injured subjects bringing into question the generalizability of these measures, making inferences to an injured population problematic. Due to these shortcomings it was concluded that FCEs have not demonstrated levels of reliability sufficient for clinical (and legal) purposes (Innes & Straker, 1999a). The difficulties seen with reliability of FCEs are mirrored with validity where it was not formally established and the developers of different FCEs felt that it was adequate to leave the validity as theoretical (Innes & Straker, 1999b; King, Tuckwell, & Barrett, 1998). Due to these issues with the psychometric properties of FCEs it was found that they only add modestly to the prediction of initial return to work and they do not have the ability to predict sustained recovery; the reported results were actually contrary to the FCEs theoretical basis (Gross et al., 2004; Gross & Battie, 2004; Innes & Straker, 1999b; Matheson et al., 2002).

Due to the lack of a gold standard for describing or predicting recovery, it is suggested (de Vet et al., 2002; Stanton et al., 2009) to use other measures of disability in conjunction with being pain free for 30 days to define when a participant is recovered. Specifically, recovery was defined by three rules i) when pain level is less than 20mm on the VAS, ii) a score of less than 8 on the RMQ and iii) resuming usual activities. This method ensures that the participants' symptoms are not artificially low due to a lack of activity, if they are able to complete usual activities while maintaining minimal symptoms they will be considered recovered. The cutoff points of 20mm on the VAS and 8 on the RMQ are consistent with previous studies on minimal detectable changes for

pain levels and minimal disability scores for the RMQ (Jensen, Chen, & Brugger, 2003; Lee, Hobden, Stiell, & Wells, 2003; Stratford, Binkley, Riddle, & Guyatt, 1998). This method allows for a more objective definition of recovery and consequently recurrence as de Vet et al. suggest that recurrence is the pain free period followed by a minimum of 24 hours of pain (de Vet et al., 2002; Hubley-Kozey et al., 2013.).

# 2.2 SPINAL STABILITY

## 2.2.1 Theory

From a mechanical engineering point of view, stability is the ability of a loaded structure to maintain static equilibrium even at small fluctuations around a position (Bergmark, 1989). If stability does not prevail, an arbitrarily small change of position is sufficient to cause collapse (Bergmark, 1989). In reference to the spine, it would be the ability of a loaded spine to maintain a static posture even with small movements around its resting posture. Bergmark acknowledges that this definition has limitations in a clinical setting. His model looks only at static environments and even though he describes how different postures require different needs to achieve stabilization, it cannot explain the requirements as a person transitions from one posture to another (Bergmark, 1989). Biomechanists on the other hand, have described spinal stability using potential energy, stating that a system is in stable equilibrium when its potential energy is at a minimum (Cholewicki & McGill, 1996; McGill & Cholewicki, 2001). This theory utilizes the analogy of a ball in a bowl, where if a perturbation were applied to the ball, it would roll up the side of the bowl and then come to rest again at the bottom, which represents the position of least potential energy (McGill & Cholewicki, 2001). The system is more stable the deeper the bowl is, representing the need for greater energy to upset the system,

and conversely less stable the more shallow the bowl is. The steepness of the sides of the bowl is represented by anatomical structures such as muscles that act as guy wires to provide compressive loads that are also augmented by increased intra abdominal pressure (Brown et al., 2006; Cholewicki & McGill, 1996). A change in any of these structures, whether due to degeneration of osseous structures or stiffness of the muscular structures can affect the overall stability of the system making it more or less stable.

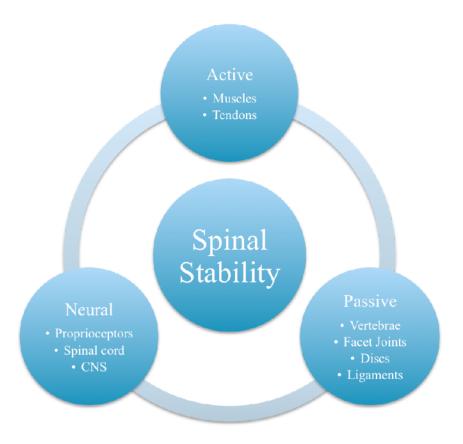
A third description of stability utilizes the same concepts as the previous two but with terminology that gives it more clinical relevance and therefore is possibly more relevant to the spine. This clinical relevance is important as acknowledged by Bergmark when he stated that, in mechanical terms, clinical stability is associated with the magnitude of the deformations when the spine is loaded. Thus the spine can be more or less clinically stable and therefore can be regarded as a continuously variable phenomenon (Bergmark, 1989). This is in contrast to mechanical stability, which is an absolute variable; the system is either stable or unstable (Bergmark, 1989). This third description, by Panjabi, defines spinal stability 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). Where he defines the neutral zone as a region of intervertebral motion around the neutral posture where little resistance is offered by the passive spinal column (Panjabi, 1992b). Additionally, he stated that any disruption of the passive spinal components (ligaments, discs or facet joints) holding the spine together would decrease the clinical stability of the spine (Bergmark, 1989; Panjabi, 1992a). Panjabi recognized that the strain curve for the spine is non linear (Brown et al., 2006; Panjabi, 1992b). This

nonlinearity represents high flexibility around the neutral position of the spine and a stiffening effect towards the end of the range of motion (Panjabi, 1992b). This is necessary for efficient functioning of the spinal system. It allows spinal movements near the neutral posture with minimal energy expenditure while still protecting the spine at more extreme range of motions by increasing stiffness (Panjabi, 1992b).

While these definitions may differ semantically, they all contain similar components or subsystems that make these models interchangeable. All these models refer to three components;

- 1.) The passive subsystem that includes vertebrae, facet joints, intervertebral discs, spinal ligaments, and joint capsules, as well as the passive mechanical properties of the muscles(Bergmark, 1989; McGill & Cholewicki, 2001; Panjabi, 1992a).
- 2.) The muscles and tendons that surround the spinal column form the active subsystem, the second common component mentioned in all models (Bergmark, 1989; Cholewicki & McGill, 1996; McGill & Cholewicki, 2001; Panjabi, 1992a).
- 3.) The third and final component is the control mechanism that receives input to coordinate an appropriate response from the two other subsystems (feedback) and the central nervous system (feed forward) (Bergmark, 1989; McGill & Cholewicki, 2001; Panjabi, 1992a). This is the neural subsystem, and consists of the various force and motion proprioceptors located in the ligaments, tendons, muscles, the spinal cord and the neural control centers in the brain (Panjabi, 1992a).

These three subsystems, although described separately, are functionally interdependent and do not work in isolation (Figure 2.1) (Panjabi, 1992a). This paper will utilize the terminology of Panjabi's model to describe spinal joint mobility and function.



**Figure 2.1:** Visual representation of the three subsystems comprising the spinal stability model.

An explanation of the interplay between subsystems comes from examining each one separately and then exploring how they work in a dysfunctional setting. The passive subsystem does not provide any significant stability to the spine in the vicinity of the neutral position (Brown et al., 2006; Panjabi, 1992a; Panjabi, 2003). It is towards the extremes of the spines' range of motion that the ligaments develop reactive forces that resist spinal motion (Panjabi, 1992a) and the osseous structures prevent motion. When considering the stabilizing effect of the passive subsystem in isolation, it is seen to be inadequate around the neutral position. This is evidenced by a calculated critical load of 90 N that is required to buckle the spinal column when placed atop it (Bergmark, 1989;

Cholewicki & McGill, 1996; Panjabi, 2003). This force is much less than the typical loads experienced or measured in daily life (Bergmark, 1989).

The differences between the critical load of 90 N required to buckle a passive spine and normal loads experienced can only be explained on the basis that muscles act to stiffen the spine and thus increase the load it can carry and its' stability (Bergmark, 1989; Panjabi, 2003). The muscles and tendons of the active subsystem are the force generators of the spine and provide the required mechanical stability and stiffness that the passive subsystem cannot (Bergmark, 1989; McGill & Cholewicki, 2001; Panjabi, 2003). In trying to model the active subsystem Bergmark detailed how the muscles introduce additional variables and create an increasingly complex system. He explained how a simple force couple model is inadequate in that it can only provide compressive information but cannot get at the intricacies of all the different muscles and their unique direction of pull with dynamic motion (Bergmark, 1989). While Panjabi's model does not try to resolve these issues by calculating loads and moments, it does provide a platform to explain and describe more accurately how stability is achieved and maintained with different movements and postures (Panjabi, 1992a; Panjabi, 1992b).

As indicated above, the ability of the active subsystem to adapt to differing challenges is controlled by the neural subsystem (Panjabi, 1992a). The magnitude of the force generated in each muscle is measured by force proprioceptors found in the muscles and tendons (Panjabi, 1992a), which is similar to the proprioceptors in the ligaments that give information about the position of the spine (Panjabi, 1992a). These proprioceptors feed into the neural subsystem, which then determines the specific requirements to maintain spinal stability and causes the active subsystem to meet those requirements

through specific patterns of muscle contractions (Panjabi, 1992a). These specific muscle patterns present a significant challenge when modeling or attempting to understand how this system works. Bergmark discussed multiple models that attempt to resolve this issue but no one model adequately explains how the neural subsystem maintains stability (Bergmark, 1989). Again, as with the active subsystem, Panjabi does not try to model the neural subsystem but rather acknowledges the complexity of the system and attempts to explain the many different challenges that it must meet to maintain stability and what happens when this control is not met (Panjabi, 1992a).

One unique aspect of Panjabi's model compared to the others discussed is his inclusion of how the body responds to dysfunction. He explains that the body has an amazing ability to compensate for deficiencies in the spinal stability system. These deficiencies may occur as ligamentous injuries or joint space narrowing in the passive subsystem, muscle tears or fatigability in the active subsystem or in the neural subsystem we could see delays in muscle activation or an abnormal response to load. All these sequelae could act to decrease the stability contribution of the different subsystems, which would result in an attempt by the other subsystems to compensate (Panjabi, 1992a). Although stability of the spine may be reestablished, the necessary compensation may prove to be detrimental to the individual components of the spine, which may compromise the ability of the body to maintain compensation and therefore stability (Panjabi, 1992a).

Specifically, with the passive subsystem it has been shown that functional alterations can occur due to working conditions and repetitive or sustained loads. These alterations can be the result of multiple different factors, the first of which is creep.

Creep results when a ligament is loaded with a constant load, and subsequently elongates over time up to a finite maximum and requires up to 24 hours to return to resting length (Solomonow, 2009). The second factor is the tension-relaxation phenomena where the tension in a ligament subjected to a constant stretch will decrease exponentially to a finite minimum and as with creep up to 24 hours can be required for tension to return to resting levels (Solomonow, 2009). These two factors also impact a third factor, hysteresis, where with repetitive loading the same tension is not developed in the ligament. The ligament length increases with each cycle reflecting the hysteresis associated with the development of creep. Conversely, when cycles of peak stretch are applied, the peak tension decreases in sequential cycles reflecting the ongoing development of tension-relaxation (Solomonow, 2009).

The impact of all these factors, therefore, is manifested by gradually decreasing tension in the ligaments, development of joint laxity, reduced joint stability and an increased risk of injury (Solomonow, 2009). Just as ligaments exhibit hypertrophy in response to loads, they can also lengthen in response to prolonged stretch and when coupled with load can result in micro trauma. This trauma and cumulative creep for lack of rest and successive work cycles could result in instability and potentially long-term injury. A similar phenomenon has also been examined for the spinal disks and the term cumulative loading as an injury mechanism has been well described by Callaghan (Callaghan, Salewytsch, & Andrews, 2001). The loads themselves are not outside physiological ranges but the cumulative effects result in altered passive stiffness and tissue damage. The type of work or physical activity associated with these changes can range from loaded repetitive tasks to prolonged sitting (Kumar, 1990).

# 2.2.2 Assessment

As already discussed, there exists multiple definitions of stability, as could be expected, there are also multiple ways to test for stability depending on the tester. Surgeons have employed simple rules such as looking for an instability catch, a painful catch or apprehension to movement (Kotilainen & Valtonen, 1993) or more complex criteria such as lateral radiographs to diagnose spinal instability (Panjabi, 2003). Some Physiotherapists use clinical tests in isolation or follow a clinical prediction rule developed by Hicks et al. and later tested and refined by others (Delitto et al., 2012; du Bois et al., 2011; Fritz et al., 2006; Hicks et al., 2005; Rabin et al., 2014; Reme et al., 2009) where a set of clinical tests are used. A 2012 review of the literature states that a diagnosis of clinical instability can reliably be made with a reasonable level of certainty when employing the Hicks prediction rule where the patient presents with at least three of the following clinical findings: i) Average SLR ROM >91°, ii) Positive prone instability test, iii) Positive aberrant movement during lumbar flexion, and iv) Age <40 years (Delitto et al., 2012; du Bois et al., 2011; Fritz et al., 2006; Hicks et al., 2005; Reme et al., 2009). The theory behind the Hicks clinical prediction rule (CPR) and its use in diagnosing clinical instability is based on the current trend of treatment-based diagnosis. This follows the premise of classifying patients into groups based on clinical characteristics and matching these subgroups to management strategies likely to benefit them (Delitto et al., 2012). The Hicks CPR was developed to predict a participant's success in a stabilization program (Hicks et al., 2005). They found that those that were positive for the rule were four times more likely to succeed in an 8 – week stabilization program. Following a treatment based diagnosis approach; those that were successful in a

stabilization program must have had a clinical instability, therefore the Hicks classification is used to diagnosis clinical instability. Even though this inference has been made it has not been validated as a gold standard is lacking in the diagnosis of spinal instability.

Hicks et al. discovered that while each individual item in the CPR has some predictive ability on its own, it is greatly increased by combining multiple items together. When examining the theoretical backing behind the individual tests, two of the tests (the PIT and aberrant motion) both examine the ability of the active subsystem to compensate for a passive instability (Biely et al., 2014; Magee, 1997). The PIT achieves this by noting a reduction in symptoms with testing and can be found in symptomatic and asymptomatic individuals (Magee, 1997), while a positive aberrant motion tests occurs when symptoms increase and usually is associated in individuals who are symptomatic (Biely et al., 2014). This calls into question the utility of aberrant motion testing or a CPR with this test in it on a recovered asymptomatic population, and in that instance would the PIT be just as selective as the entire CPR?

A recent RCT by Rabin et al. in 2014 failed to validate the Hicks CPR and suggested that a modification may be necessary to increase its predictive power. One suggestion Rabin had was to remove age from the rule and even Hicks et al. acknowledged that although they found age to have a high positive likelihood ratio, it was mostly due to the length of the stabilization program and older individuals could be just as successful with a longer program (Hicks et al., 2005). While the positive likelihood ratio is 4.0 when there are three positive tests out of four, Hicks et al. also determined that with two positive tests out of four the positive likelihood ratio is 1.9.

Therefore, while there exists multiple methods to test for spinal instability, the recommendations from Rabin et al. and Hicks et al. that employed a modification of the original Hicks CPR where the condition of age was removed and a positive finding was two out of the remaining three parameters was shown to have validity.

## 2.3 STRENGTH

While these clinical instability-testing strategies rely primarily on assessing the passive subsystem additional tests are required that focus on the active and neural subsystems. Concerning the active subsystem, the literature has shown that isometric strength is diminished in a chronic LBP group (Mannion et al., 2001) and that even though sufficient strength is important to function, so is muscle endurance (McGill et al., 1999). Furthermore, it was shown that muscle activity was decreased in the multifidus of individuals with recurrent LBP compared to controls during different loading tasks (MacDonald et al., 2010). Finally, it was found that even though spinal stability requires trunk muscle co-activation (Brown & Potvin, 2005; Cholewicki & McGill, 1996; Vera-Garcia, Brown, Gray, & McGill, 2006) a naturally selected activation pattern was superior to conscious adjustments when attempting to maintain spinal stability (Brown et al., 2006). This body of evidence points to the important contribution of the active subsystem to spinal stability and any model should include some aspect or measurement of this subsystem.

Most of the studies examining strength in LBP have been on individuals with chronic LBP and while some have shown that there is a difference in back muscle strength between a group with LBP and a control group (Alston et al., 1966; Descarreaux et al., 2004; Gruther et al., 2009; Mannion et al., 2001), others reported no difference

(Bayramoglu et al., 2001; da Silva et al., 2005; Iwai et al., 2004; Sjolie & Ljunggren, 2001). Only one study was found reporting decreased strength in a group with chronic LBP and clinical instability compared to no instability and a control group (Davarian et al., 2012). Additionally, of interest is the ratio of strength between the abdominal and back muscles. It has been shown that those with LBP have greater co-contraction during lifting activities than those without LBP (Butler et al., 2012). This difference in cocontraction could be reflected in the abdominal to back muscle strength ratio and although this ratio has been examined previously, no consensus was reached concerning a difference between those with LBP and a control group, with some reporting a difference (Gruther et al., 2009; Holmstrom, Moritz, & Andersson, 1992b) while other stated that there is no difference (Alston et al., 1966; Shirado et al., 1992). To my knowledge no research has examined if a difference exists in strength ratios between a group with clinical instability and a group without. Measures of strength could help explain EMG findings and any differences found between the groups. If, for example, an amplitude difference was found between the two groups, it could be verified if that is due to a difference in strength of specific muscles or a difference in activation levels, or both. In summary there is minimal literature on the topic of strength in subjects with recurrent injuries.

Furthermore, muscle strength only gives an indication of the total voluntary force production that the muscle can generate at that moment and while that is related to the neural subsystem, it only provides a piece of the picture. Thus, to truly examine the neuromuscular subsystem (active and neural subsystems), the muscle activation patterns need to be examined as well.

#### 2.4 ELECTROMYOGRAPHY

# 2.4.1 Theory

EMG provides us with an assessment of the neural input to the muscle and the muscle response by measuring the number of motor units that are active in the pickup region of an electrode. Works by Cholewicki et al. and Heydari et al. demonstrated that EMG measures can predict who will develop LBP (Cholewicki et al., 2005; Heydari et al., 2010), and a number of studies have shown differences in EMG measures for those with chronic low back pain compared to a group without low back pain (Cholewicki & McGill, 1996; Geisser et al., 2005; Radebold, Cholewicki, Polzhofer, & Greene, 2001). Recently, MacDonald et al. showed that not only do differences exist in the muscle firing times from deep and superficial fibers of one back extensor site, but that there were also differences in the thickness of that same muscle between a group that were in remission of a LBI and a control group (MacDonald et al., 2009; MacDonald et al., 2010; Macdonald et al., 2011).

Collectively, the above EMG studies and the majority of the literature are limited to discrete measures and often few muscle sites, thus do not provide a comprehensive picture of the trunk musculature or coordination and synergies during dynamic tasks. Previous work by Hubley-Kozey et al. has addressed this issue by examining coordination of muscle activation patterns during controlled dynamic tasks using a comprehensive EMG protocol for the trunk muscles and pattern recognition techniques that can measure dynamic responses and the relative amplitude differences among muscle sites during highly controlled tasks (lift and replace and leg-loading exercises) (Butler et al., 2009; Butler et al., 2012; Hubley-Kozey & Vezina, 2002; Hubley-Kozey, Butler, &

Kozey, 2012; Hubley-Kozey et al., 2013.). Hubley-Kozey et al. showed that this EMG technique has the ability to detect differences in those with chronic low back pain (Hubley-Kozey & Vezina, 2002) but, more important to the present study, it differentiated those who are deemed recovered from a LBI versus controls, thus indicative of level of recovery (Hubley-Kozey et al., 2012; Moreside, Quirk, & Hubley-Kozey, 2014). These preliminary findings suggest that this comprehensive EMG approach has potential predictive capabilities for those who are at risk for a recurrent LBI within one year after they are deemed recovered and can serve as an adequate test of the neuromuscular system (Hubley-Kozey, Moreside, & Quirk, 2014b). This emphasizes that there is indeed a deficiency in the active and/or neural subsystems that contribute to the recurrence of LBI and can be measured using EMG. EMG therefore, gives us an objective method to measure neuromuscular activity as well as coordination.

To understand how EMG can measure the active and neural subsystems we need to understand what EMG is and what it is measuring. EMG is the study of muscle electrical activity and can provide information about the control and execution of movements (Winter, 2009). Although there are many technical features necessary to interpret the EMG signal, the signal itself has a physiological origin and it is this origin that details the EMG signal's involvement with the active and neural subsystems (Winter, 2009). An EMG signal is the sum of all motor unit action potentials (MUAP) recorded by a pair of electrodes on the surface over or inserted into a muscle(Winter, 2009). The MUAP is the result of a stimulus at the central nervous system (CNS) that is propagated along the alpha motor neuron across the neuromuscular junction causing a change in ion concentration across the muscle cell (fiber) membrane resulting in an action potential

being generated that propagates along the muscle fiber (Robertson, 2004; Vander, Sherman, & Luciano, 1994; Winter, 2009).

Skeletal muscle plasma membrane is an excitable membrane capable of generating and propagating action potentials, which precede force development. As the MUAP spreads across the plasma membrane and into invaginations of the membrane the depolarization causes increased calcium release which serves as the instigator for cross-bridge formation and subsequent contraction of the muscle fiber (Vander et al., 1994) and force production. This sequence of events from the MUAP to cross-bridge formation is termed excitation-contraction coupling. The EMG signal that is recorded from a pair of electrodes is a summation of all the MUAPs that occur in the pick-up region of those electrodes and represents the overall strength and timing of a contraction (Robertson, 2004; Winter, 2009) and represents the neural (excitation) and muscle response and in part the active (contraction) subsystems as described by Panjabi (Panjabi, 1992a).

Although the force or output of the muscle is not captured there are many factors including activation responsible for the force production (Alkner, Tesch, & Berg, 2000; Lawrence & Luca, 1983; Woods & Bigland-Ritchie, 1983).

# 2.4.2 Principal Component Analysis

As stated in the section on spinal stability, the overall mechanical stability of the spine is an interaction of all the subsystems (i.e. provided by the spinal column and the precise neural coordination of the surrounding muscles) (Panjabi, 2003). Coordination of the motor system is an incredibly complex task that includes, not exclusively, motor programs and synergies to effectively meet its goals (Schmidt, 1982). The concept of coordination of the motor system speaks to the larger concept of motor control, which is

the process by which humans organize and execute their actions. Fundamentally, it is the integration of sensory information to determine the appropriate sequence of muscle forces and joint actions to achieve the desired movement or action. This process requires cooperative interaction between the central nervous system and the musculoskeletal system and is therefore a problem of information processing, coordination and mechanics (Rosenbaum, 1991). Panjabi stated that the synergies and coordination required for the stabilizing role of the spinal muscles cannot easily be studied by EMG measurements of the muscle alone (Panjabi, 2003). This concept has recently been challenged by Wakeling who demonstrated that with EMG and Principal Component Analysis (PCA), recruitment and temporal patterns of muscles can be recorded that speak to the synergies created by, and the coordination of, the muscles (Lee, Miara, Arnold, Biewener, & Wakeling, 2011; Wakeling, 2009).

PCA is a multivariate statistical technique used to describe the variability within a group of related variables (Jackson, 2003), and has been shown to be an effective data reduction technique that is useful when analysing large quantities of data to understand co-activation and temporal synchronies among muscles (Butler et al., 2009; Hubley-Kozey & Vezina, 2002). PCA has been shown to reduce the number of variables while still maintaining important features of the original data (Butler et al., 2009; Hubley-Kozey & Vezina, 2002; Landry et al., 2007). This is achieved by transforming the original variables into new, uncorrelated variables (Jackson, 2003). It is effective in analysing the overall magnitude, shape and temporal patterns of kinematics, kinetics and EMG waveforms as well as other movement tasks (Hubley-Kozey & Vezina, 2002; Landry et al., 2007).

PCA uses inter-subject variation throughout entire waveforms to identify features that can be used to describe differences between subject groups and/or tasks (Landry et al., 2007). PCA lends more objectivity to the analysis of data as it analyzes the entire waveform and assigns scores based on the variation in the data itself (Landry et al., 2007). This is in contrast to the subjective use of discrete parameters that have been deemed a priori to be important such as peak values in EMG waveforms or the timing of certain events. While these parameters may be important, the subjectivity comes from the arbitrary choice of these values, whereas PCA finds the most relevant features based on the data (Jackson, 2003; Landry et al., 2007).

In summary, although there is no consensus on an operational definition for recovery, there is evidence to support using measures of disability in conjunction with being pain free for 30 days to define when a participant is recovered. Spinal stability as defined by Panjabi relies on the interaction of three subsystems, and while they are described separately it is the interaction between the different subsystems that dictates if there is a problem or not. Hicks et al. developed a prediction rule to clinically assess aspects of the passive subsystem while EMG and strength have been shown to measure different aspects of the active and neural subsystems. The Hick's classification approach, which identifies a subcategory of those with nonspecific low back pain as clinically unstable has been shown to have construct and face validity. Evidence is clear that trunk muscle activation patterns as measured by EMG are altered in the presence of chronic low back pain but recent work illustrates that differences also exist in those deemed recovered for a low back injury. Furthermore preliminary evidence suggests that these EMG patterns might be predictive of re-injury risk. What has not been examined is

whether EMG patterns are different in those who are classified as clinically unstable. Finally, when analyzing EMG, discrete measures have most often been used, however PCA provides an objective method of identifying important features that include both amplitude and temporal features that can assess co-activation and temporal synergies, as both are described in the literature as being important activation patterns needed to maintain spinal stability.

#### CHAPTER 3 METHODOLOGY

#### 3.1 RESEARCH DESIGN

This cross-sectional comparative study measured activation patterns from a comprehensive set of trunk muscles and maximum trunk flexor and extensor muscle strength from a group of participants with a LBI who were deemed recovered from their injury based on return to work status and self-reports of pain and function. For the main objective the group was divided into those classified with a clinical instability (Clinical Instability Group or CIG) and those with no clinical instability (No Clinical Instability Group or NCIG) as defined by the modified Hicks CPR. For the sub-objective the groups were divided by the PIT (Hicks et al., 2005; Rabin et al., 2014). Specifically, the study group included those people who were in the sub-acute phase (between 4 and 12 weeks from injury) of LBI who were deemed recovered. Recovery was consistent with previous work and was based on self-report remission of symptoms and minimal functional disability (VAS less than 20 mm and disability RMQ scores less than 8) and resumption of normal activities or if they were within one week of returning to these activities (Butler et al., 2012). Approval for this study was obtained from the Health Sciences Research Ethics Board, Dalhousie University and the Canadian Armed Forces through the Surgeons Generals Health Research Program.

#### 3.2 SUBJECTS

The study population included 32 participants with a LBI who were recruited from the military hospital at CFB Halifax from 12 December 2013 to 5 June 2014. For a detailed breakdown of all referrals please refer to Figure 3.1. As this is the primary site

for all military members to access health care, it was inclusive of all members stationed at CFB Halifax. When a military member accessed the base hospital for any issue related to their lower back they were asked if they consented to have their contact information given to the research coordinator who contacted them to determine if they were interested in participating in this study. If they agreed to participate, the research coordinator then proceeded with screening measures. This occurred according to the recruitment flow chart located at Figure 3.2.

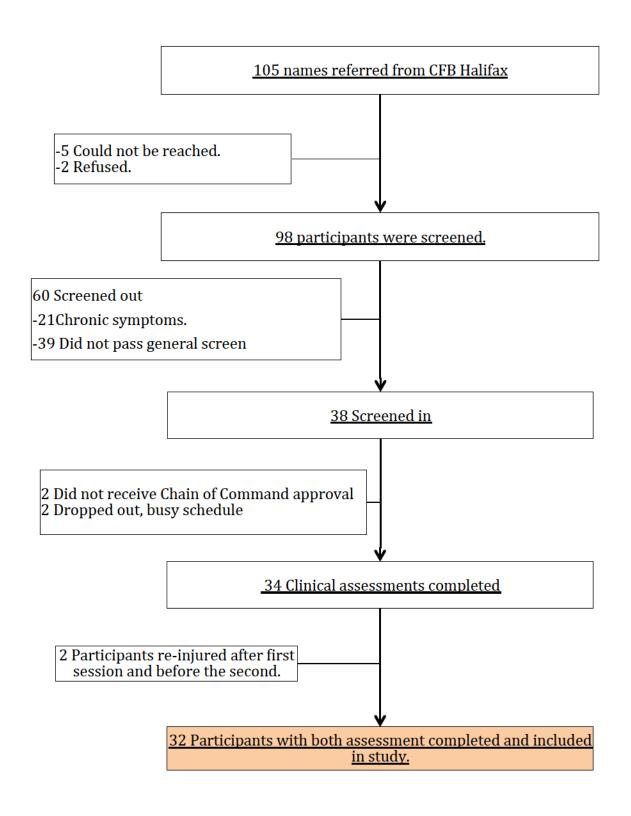


Figure 3.1: Detailed breakdown of all referrals.

# **Recruitment Flow Chart**

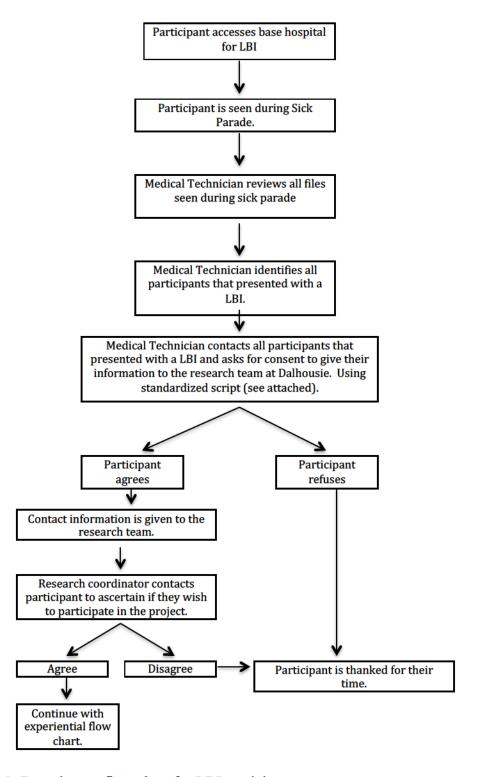


Figure 3.2: Recruitment flow chart for LBI participants.

Inclusion and exclusion criteria were first addressed through an initial telephone health-screening questionnaire and were confirmed during the initial testing session where a standard postural, neurological and Physiotherapy assessment was conducted (Appendix 2). The inclusion and exclusion criteria were as follows:

#### Inclusion

- Men and women in the Canadian Armed Forces between the ages of 18 and 55 years.
- Defined as having pain between the lower ribs and the gluteal folds as a result of a specific acute event.
- Within the sub-acute phase defined as greater than 4 weeks and less than 12 weeks post injury.
- VAS less than 20mm, RMQ less than 9 and deemed recovered based on resuming usual work and leisure activities with no restrictions or be within one week of their return to activities.

#### Exclusion:

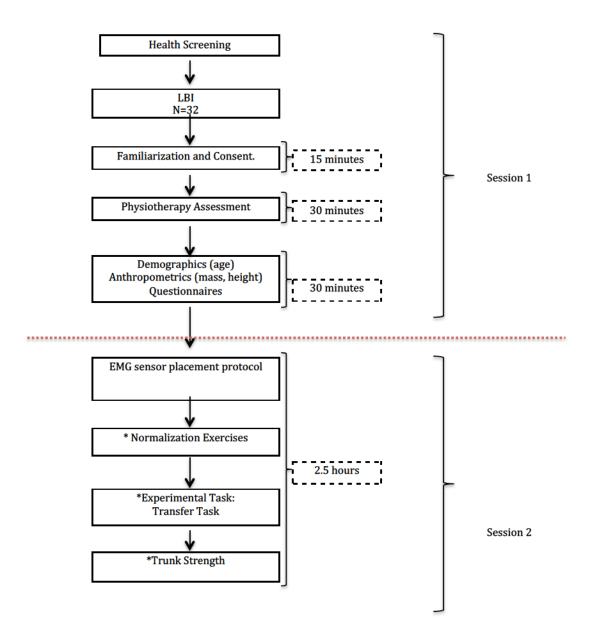
- Cardiovascular, neurological or other musculoskeletal problems that would place them at risk or prevent them from completing the study tasks correctly.
- LBP that has lasted for greater than 3 months.
- Nerve root pain.
- Neurological signs and symptoms.
- Complications such as tumor or infection.
- Previous spinal surgery.
- Spinal fracture.
- Any structural deformity such as scoliosis or spondylolisthesis.

• 12 weeks post injury and unable to resume their usual work or leisure activities.

#### 3.3 PROCEDURE

All participants were seen on two different occasions. An outline of the experimental procedures and their associated durations are shown in Figure 3.3. During the first session the participant read and signed the informed consent. Descriptive variables were collected including: age, sex, height, mass, occupational activity level (Matheson, 1982) and how they perceive and cope with their pain (as measured by the pain catastrophizing scale (PCS) (Sullivan, Bishop, & Pivik, 1995)). A registered physiotherapist conducted a postural (including scoliosis and kyphosis), and neurological assessment including reflexes (patellar, Achilles tendon), myotomes and dermatomes, which were used in conjunction with the VAS and RMQ to inform decisions on inclusion or exclusion of the participant. Spinal instability was screened for utilizing specific tests detailed by Hicks et al (Hicks et al., 2005). These tests included: aberrant movement during lumbar flexion/extension, passive straight leg range of motion, and the prone instability test (Hicks et al., 2005). Detailed descriptions of each of these tests can be found in Appendix 2. Participants were classified in the CIG if they had at least two of the following: i) passive straight leg raise greater than 90°, ii) a positive prone instability test or iii) presence of aberrant movement during sagittal lumbar movement, as is consistent with a modification of the Hicks CRP (Hicks et al., 2005; Rabin et al., 2014). The NCIG was made up of all participants not fitting the above criteria.

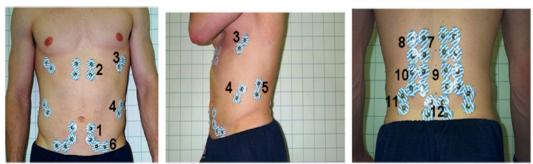
# **Experimental Procedures Flow Chart**



**Figure 3.3:** Flow chart of the experimental procedure and an overview of research variables that were measured with the estimated time cost of each. Total participation time is approximately 4 hours.

\*All tasks denoted with an \* include the collection of surface EMG, trunk/pelvis motion and pre/post VAS.

During session 2, participants were prepared for data collection using a standardized protocol based on previous work to capture EMG and motion data of the trunk (Butler et al., 2012). For EMG recordings, Ag/AgCl single use disposable surface electrodes (10mm diameter, Red Dot, 3M, London, Ontario, Canada) were placed by a trained researcher based on standardized locations in line with the muscle fibers in a bipolar configuration (interelectrode distance of 25 mm) over 12-bilateral trunk muscle sites as seen in Figure 3.4 (Butler et al., 2009). Minor adjustments were made to accommodate participant anatomical differences as confirmed by palpation and submaximal validation exercises for each specific muscle site (Butler et al., 2009). Abdominal sites included: lower (LRA – midpoint between the pubis symphysis and umbilicus) and upper rectus abdominis (URA – midpoint between the umbilicus and the sternum); anterior (EO1 – over the eighth rib), lateral (EO2 – approximately 15 cm lateral to the umbilicus at a 45° angle) and posterior fibers (EO3 – halfway between the iliac crest and lower portion of the ribcage) of external oblique and internal oblique (IO – centered in the triangle formed by the inguinal ligament, lateral border of rectus sheath and the line between the two anterior superior iliac spines). Six bilateral back extensor sites were also used and included: lumbar erector spinae at L1 and L3 at 3 and 6 cm from the midline to represent the longissimus and iliocostalis muscle sites, respectively (L13, L16, L33, L36); quadratus lumborum at L4 at approximately 8 cm from the midline (L48); and multifidus at L5 at 1–2 cm from the midline (L52). Once electrode placement was determined, the skin was prepared for surface electrodes using an alcohol/water solution to improve signal conduction.



**Figure 3.4:** Electrode muscle sites on right and left sides of the body. 1 = LRA; 2 = URA; 3 = EO1; 4 = EO2; 5 = EO3; 6 = IO; 7 = L13; 8 = L16; 9 = L33; 10 = L36; 11 = L48; 12 = L52. Taken from (Butler, Hubley-Kozey, & Kozey, 2010) with authors' permission.

EMG signals were pre-amplified (200x) and further amplified using three AMT-8 EMG systems (band pass 10-1000 Hz; CMRR = 115db, input impedance 10G $\Omega$ ; Bortec INC., Calgary Ablerta). Raw EMG signals and event markers (a step voltage change) were digitized at 2000 Hz using a 16-bit resolution analog-to-digital conversion board (National Instruments, CA-1000) and Labview<sup>TM</sup> software (version 7).

#### 3.4 NORMALIZATION PROCEDURE

Following electrode placement the participants were asked to perform a series of standardized exercises that require maximum voluntary isometric contractions (MVIC) for normalization purposes (Butler et al., 2010). The purpose of these MVICs was to obtain maximum EMG activation amplitudes to which all data was normalized so that muscle activation can be expressed as a percent of MVIC. This procedure is utilized to allow for more valid between subject and between muscle comparisons (Burden, 2010; Geisser et al., 2005; Ng, Parnianpour, Kippers, & Richardson, 2003; Vera-Garcia, Moreside, & McGill, 2010). During these normalization exercises, participants were provided with standard verbal encouragement, which has been shown to improve the reliability of the MVIC procedure (Ng et al., 2003). Additionally, to ensure participants'

safety and that no movement will occur during these contractions, participants were secured using non-elastic straps (Butler et al., 2009). The following eight different MVIC exercises were performed: a supine sit-up was performed to recruit the rectus abdominis sites (Vezina & Hubley-Kozey, 2000). Seated axial rotation (right and left) and side-lying lateral flexion (right and left coupled with ipsilateral hip hike) were utilized to maximally activate the oblique muscle sites (Butler et al., 2009). Back extension and back extension coupled with axial rotation (right and left) performed in a prone position are to maximally recruit the back musculature. All normalization exercises were held for three seconds and repeated twice with a 2-minute rest between trials resulting in a total of 16 trials. After the normalization trials, a three second trial of EMG data was collected for baseline muscle activity (subject bias) while the subject was lying supine and relaxed. System bias was recorded for one second at the end of the session. These two biases were used during processing to correct the EMG data.

## 3.5 MOTION MEASUREMENT

Following MVIC exercises, participants were prepared for motion analysis data collection. The purpose of measuring motion during the task was to confirm that minimal motion occurred during the task and to determine that similar motion occurred between groups. By controlling the motion occurring at the trunk and confirming that there was no difference in motion between groups we assumed that the EMG recorded was responding to the two main moments (flexion and lateral flexion) created by moving the load during the transfer task. Three-dimensional motion (kinematics) was collected from an electromagnetic Flock of Birds<sup>TM</sup> (FOB) Motion Capture system (Ascension Technology Inc., Burlington, Vermont). The FOB recorded the angular motion of the

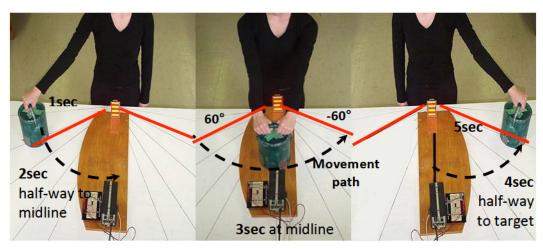
pelvis, lumbar spine and thoracic spine throughout the transfer task in 3D with respect to the global coordinate system (x = frontal, y = sagittal, z = transverse). One sensor was placed superior to the left anterior superior iliac crest, the second over the L4 spinous process and the third over the T8 spinous process. Maximum angular displacements for each phase of movement was calculated and compared between groups, providing a quantitative measure of how much motion occurred. Minimal motion was desired and thus, during the collection, any trials identified with excessive motion were redone.

#### 3.6 EXPERIMENTAL TRIALS

This study used portions of the same experimental protocol as previously described in work from this laboratory(Butler et al., 2009; Butler et al., 2012; Davidson & Hubley-Kozey, 2005; Hubley-Kozey et al., 2012; Hubley-Kozey et al., 2013.). The participants were asked to perform a standardized lift and replace task. This task was designed to provide a dynamic challenge the trunk musculature to respond to constantly changing flexion and lateral flexion moments while participants were instructed to minimize trunk and pelvis motion (Butler et al., 2010; Hubley-Kozey et al., 2012). The following is a brief description of the Transfer Task and a more details description is provided in Figure 4. The task involved transferring a 2.9Kg load from the right side of their body (at 60° from their body midline) to the left side of their body (60° from the midline of their body). During this task participants were instructed to perform the movement to a 5 second external pace, where the load must be at a unique position every second (Butler et al., 2010). This task was performed in one direction (right-to-left) and was performed using maximum (elbows fully extended) reach (Figure 3.5). A pressure sensor was fixed to the bottom of the load to indicate when the task started and finished.

A photoelectric sensor was positioned at the midline to detect when the participant transferred the load. This set-up allowed detection of the start, middle and end of the movement so that movement times could be compared between groups.

## (a) Right-hand transfer phase (b) Hand transition phase (c) Left-hand transfer phase



**Figure 3.5**: Experimental set up, participant position and right-to-left movement path in maximum reach for the transfer task. The simulated movement requires the participants to first lift the load positioned at 60° to their body midline with their right hand, then move the load toward their midline, transfer the load to the left hand at their midline and then move the load away from the midline and replace it on the other side of their body midline at -60°. During this movement the participants will be required to follow the path in a slow and controlled manner while maintain the load approximately 4-5cm above the table surface. Using a standard five-second count and an event marker system, the lifting movement will be divided into three phases: (a) right hand transfer, (b) hand transition and (c) left hand transfer phases. Taken from (Butler et al., 2010) with authors' permission.

During this task participants were positioned at a table adjusted to elbow height. To ensure minimal movement participants were provided tactile feedback applied to their thoracic spinous process using a vertical jig as well as using the above-described motion capture system(Waters, Putz-Anderson, & Garg, 1994). Participants were given as many practice trials as needed to feel comfortable with the task. Once comfortable, the participant performed the task until 5 successful trials were recorded. Trials were

included if they adhered to the 5 second count and exhibited minimal trunk and pelvic motion.

## 3.7 STRENGTH

Strength variables collected were from trunk flexor and extensor muscle groups and included torques measured from two MVIC exercises performed at the end of the data collection. The participants were positioned in the prone and crook lying positions for lumbar extension and flexion respectively (Figure 3.6). Standardized placement was used for the arm of the HUMAC where it was located just below the clavicles for trunk flexion and in line with the spines of the scapula for trunk extension. Each exercise was performed twice and held for a 3-second count with a two-minute rest between repetitions using a Humac Norm Dynamometer (Computer Sports Medicine Inc, Stoughton, MA, USA). The participants were provided with the same verbal encouragement as during the normalization trials.



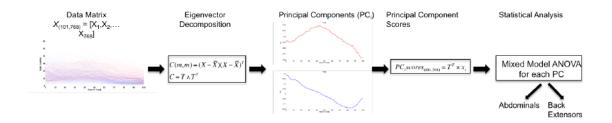
**Figure 3.6**: Patient positioning for strength measure of lumbar extension.

# 3.8 EMG PROCESSING AND ANALYSIS

A 30 Hz high pass filter was applied to the EMG signals to minimize noise due to electrocardiographic artifact (Butler, Newell, Hubley-Kozey, & Kozey, 2009). The power spectrum was calculated for each EMG signal and if any abnormal signals were detected, such as a 60 Hz noise from the power system or specific noise from the electromagnetic field generated by the FOB, it was removed with an inverse Fast Fourier Transform filter. All EMG data were corrected for bias and gain, full wave rectified and low pass filtered at 6 Hz using a second order recursive Butterworth filter to yield a linear envelope profile. Data were time normalized from lift off to replace using a linear interpolation algorithm, and then amplitude normalized to the 500 ms peak amplitude from the normalization exercises (Hubley-Kozey & Vezina, 2002). All five trials were visually inspected for consistency then an ensemble-average waveform was calculated for each muscle and subject.

The time and amplitude normalized ensemble-averaged EMG waveforms were the input to a principal component analysis model. A schematic of the procedure can be found in Figure 3.7. To extract important amplitude and temporal patterns from the EMG waveforms an eigenvector decomposition was performed on the covariance matrix of the ensemble-average profile of the trunk muscles ([32 subjects × 24 muscles] = 768 × 101). Principal components (PCs) were extracted based on how much variance they represented, and those PCs that accounted for greater than 95% of the variation were included if they explained more than 1% of the variation in the data. Next, each measured waveform was scored (PC score) based on how similar the waveform corresponds to each PC (feature) (Hubley-Kozey et al., 2012; Hubley-Kozey et al., 2014a). PC scores were used in statistical analysis. PC1 scores reflect the overall amplitude of the EMG

waveform with a zero score being equivalent to the amplitude of the mean pattern (Hubley-Kozey, Hanada, Gordon, Kozey, & McKeon, 2009; Hubley-Kozey et al., 2012). A positive score represents an amplitude value greater than the mean and a negative score less than the mean. For PC2 scores, a zero indicates that the pattern does not contribute to the overall measured pattern, a positive score adds to the mean pattern and a negative score subtracts from it (Hubley-Kozey et al., 2012).



**Figure 3.7:** Schematic representation of the PCA procedure. PCs were identified as containing the most relevant features. Modified from(Butler et al., 2009)

#### 3.9 FOB PROCESSING AND ANALYSIS

FOB motion data were filtered at 1Hz with a recursive second order Butterworth low pass filter and the maximum angular displacements in all three planes for each sensor were calculated using custom software during the motion relative to the global coordinate system.

#### 3.10 STRENGTH PROCESSING AND ANALYSIS

A 500 msec moving average was used on the torque output for each trial to identify the peak. The maximum torque across the two trials for each condition was used as the maximum strength values for further analysis. Torque was normalized to body mass in Kg to compensate for any anthropometric differences between individuals.

Additionally a flexion to extension torque ratio was calculated and compared between groups.

#### 3.11 STATISTICAL ANALYSIS

Means and standard deviations for all continuous variables were calculated and frequency counts for categorical data. Differences between groups for age, height, weight, motion data, BMI, VAS initial, VAS final, RMQ, PCS, normalized and nonnormalized flexion and extension torques as well as the flexion to extension ratio were tested using Student T-tests ( $\alpha=0.05$ ). ANOVAs were used to test the different PC scores. Due to the small sample size, the exploratory nature of this study and the need for an alpha adjusted for multiple comparisons, an  $\alpha=0.10$  was used for interaction while  $\alpha=0.05$  was maintained for main effects. Homogeneity of variance and normality of the distributions were checked for demographic data, motion variables and muscle activation PC scores. If data was found to not be normally distributed, Johnson transformations were applied. Fisher's exact test was used to test for differences in occupational activity level, which is a standardized self report measure on job demands ranging from sedentary to very heavy (please see Appendix 2 for an explanation). All tests were performed using Minitab (Minitab Inc, State Collage, PA, version 17)

Specifically two-factor (group and muscle) ANOVA models tested for differences in PC scores for the back extensor and abdominal muscles separately. Post hoc Tukey comparisons were applied to the significant findings. This resulted in 4 ANOVA's (two for the abdominals and two for the low back musculature).

# CHAPTER 4: COMPARING TRUNK MUSCLE ELECTROMYOGRAPHIC PATTERNS AND STRENGTH BETWEEN THOSE RECOVERED FROM A LBI WITH CLINICAL INSTABILITY AND THOSE WITHOUT.

# 4.1 INTRODUCTION

Low back disorders constitutes a large problem, with 80% of people experiencing at least one episode of low back pain (LBP), defined as pain between the lower ribs and the gluteal folds, at some point in their life (Dagenais et al., 2008; Stanton et al., 2009). While it is claimed that 90% of LBP will resolve itself, up to 62% will experience a reinjury within one year (Hestback et al., 2003). Furthermore, recurrence in LBP has been shown to be a major determinant for it transitioning into a chronic state (Wasiak et al., 2009). Presumably it would be more efficient and cost effective to be proactive and deal with LBP before it becomes chronic, thereby providing either primary prevention (preventing LBI from occurring) or secondary prevention (early detection of injury and interruption of progression into chronic) (Mirolla, 2004).

There is a lack of clarity in terminology, which can cause confusion, for example the term low back injury (LBI) and LBP are used synonymously in the literature (Briner & Benjamin, 1999; Kraus, Schaffer, McArthur, & Peek-Asa, 1997; Lusted, 1993). The latter while not well defined does relate more to the cause and an event that results in pain whereas the former focuses mainly on symptoms i.e. pain (McGill, 1997; Ozguler, Leclerc, Landre, Pietri-Taleb, & Niedhammer, 2000; Spitzer, LeBlanc, & Dupuis, 1987). Furthermore it is becoming evident that the term LBP/LBI is actually a large heterogeneous group consisting of multiple distinct subgroups (Delitto et al., 2012). This

large heterogeneous group that has no specific cause is categorized as nonspecific low LBP. The present study will use both terms, with LPB describing the symptom and LBI referring to an event or occurrence that results in a decrease in function or a deficiency in one of the many structures contained in the back, understanding that this terms is used interchangeably in the literature.

In an effort to adequately divide and identify these subgroups, an emphasis has been placed on groupings of clinical tests in place of a single test. These clinical prediction rules (CPRs) use clinical findings from the history and physical exams to make a diagnosis or predict outcomes. They quantify the relative importance of particular findings when evaluating a patient (Laupacis, Sekar, & Stiell, 1997; Wasson, Sox, Neff, & Goldman, 1985). One subgroup of individuals with LBP that has been defined is nonspecific LBP due to spinal instability and a CPR was developed by Hicks et al. in 2005 to identify individuals in this group (Hicks et al., 2005). A 2012 review of the literature states that a diagnosis of clinical instability can reliably be made with a reasonable level of certainty when employing the Hicks prediction rule where the patient presents with at least three of the following clinical findings: i) Average SLR ROM >91°, ii) Positive prone instability test, iii) Positive aberrant movement during lumbar flexion, and iv) Age <40 years (Delitto et al., 2012). When developing the CPR, Hicks et al. also identified that reducing the number of positive tests to 2 still retains some diagnostic power of the CPR (Hicks et al., 2005). More recently, modifications have been suggested to better define this group specifically related to decreasing the number of tests in the CPR to include those more related to how the active and neural subsystems (muscles and control mechanisms) compensate for a deficient passive subsystem (ligaments, joints, and

bones). This, plus a more recent study also identified age as a factor that could be removed from the CRP while remaining valid as this factor was more related to the ability to succeed in a stability program than as a risk factor for clinical instability (Hicks et al., 2005; Rabin et al., 2014).

While clinical measures represent a feasible tool to define groups, evidence is lacking in demonstrating their ability to identify specific physiological alterations that define these groups. More objective measures such as EMG and strength measure the neuromuscular system and the active subsystem, and have shown some ability to identify those physiological alterations, which may make them more applicable tests to help define subgroups of LBI. Not only have EMG measures been used to differentiate individuals recovered from a LBI and those who never suffered a LBI, (Butler et al., 2012; MacDonald et al., 2009; MacDonald et al., 2010; Macdonald et al., 2011) but also modest evidence has been produced using EMG to predict recurrence (Hubley-Kozey et al., 2014a). While trunk extensor strength has shown to be diminished in both a chronic LBP population (Alston et al., 1966; Descarreaux et al., 2004; Gruther et al., 2009; Mannion et al., 2001) and one with clinical instability (Davarian et al., 2012) no work has been found that examined strength in a recovered population. Aside from just basic strength, ratios are examined and can provide some insight into individual muscle imbalances. Although no consensus has been reached in the literature when reporting differences between a LBI and a control population, due to reported decreases in extensor strength it is hypothesized that the flexion to extension ratio would be greater in a group with clinical instability with one without (Davarian et al., 2012). Ideally, clinical tests could identify some of these underlying physiological alterations, as done with the

objective measures that could lead to re-injury. To achieve this, comparative analysis could be done between both clinical and objective measures to obtain the optimal combination; whether it consists solely of the objective tests or a mix with the clinical ones also.

Therefore, the purpose of this study was to determine whether there are differences in objective measures of trunk muscle function in those deemed recovered from LBI classified as having a clinical instability versus those that do not. The objective measures of muscle function include a comprehensive examination of trunk muscle amplitude and temporal activation patterns during a standardized functional task, and maximal voluntary isometric trunk flexor and extensor strength (both as a ratio and as an independent measure). The hypotheses examined are that those with clinical instability will have different trunk neuromuscular patterns compared to those with no instability while performing a highly controlled function task that has dynamic external moments. More specifically, it is hypothesized that those with instability have increased antagonist/agonist co-activation during the transfer task compared to those without instability and they have more sustained activity i.e. less response to changing external moments (flexion and lateral flexion). Additionally, it is hypothesized that those with clinical instability have decreased back muscle strength and consequently an increased abdominal to back strength ratio.

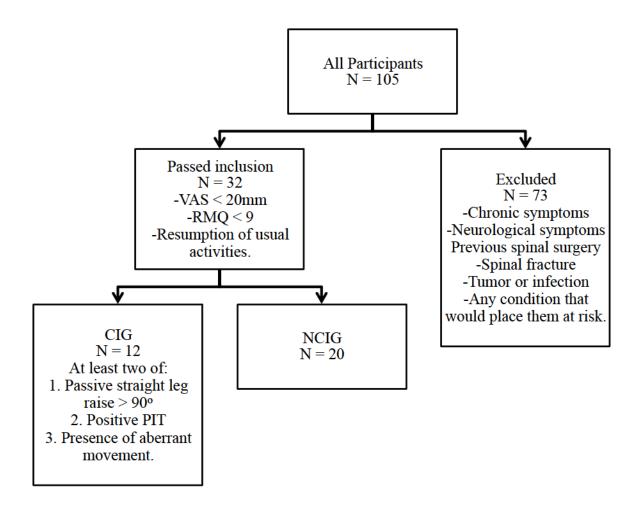
#### 4.2 METHODOLOGY

# 4.2.1 Participants

The study population included 32 participants with a LBI recruited from the military hospital at CFB Halifax representing an inclusive sample of all military members stationed at CFB Halifax. Inclusion criteria were: being in the sub-acute phase (between 4 and 12 weeks post injury) of a LBI (pain between the lower ribs and the gluteal folds as a result of a specific event). Additionally the participants were deemed recovered as indicated by experiencing minimal pain (defined as less than 20mm on the visual analogue scale (VAS) where 0mm = no pain, 100mm = worst imaginable pain), minimal disability (defined as a Roland Morris (RMQ) score less than 8 out of a possible 24 where 0 is no disability and 24 is maximum disability) and either already resuming usual activities or being within a week of resumption of usual activities (de Vet et al., 2002; Jensen et al., 2003; Lee et al., 2003; Stanton et al., 2009; Stratford et al., 1998). Excluded were those whose pain was related to a specific cause such as a fracture, tumour or infection, those that had a previous spinal surgery or were experiencing chronic LBP (pain lasting greater than 12 weeks). Participants were also excluded if they had neurological symptoms or other unrelated issues that would preclude them from completing the experimental task. Figure 4.1 outlines the breakdown of participants with inclusion and exclusion criteria as well as group definitions.

Participants were separated into two groups based on a classification of clinical instability as defined by a modified Hicks clinical prediction rule (Hicks et al., 2005; Rabin et al., 2014). Participants were in the clinical instability group (CIG) if they tested positive on at least two of the following clinical tests, otherwise they were in the no

clinical instability group (NCIG) (Figure 4.1): 1. Aberrant motion, 2. Passive straight leg raise greater than 90°, 3. Prone instability test. An orthopaedic physiotherapist with 12 years of experience performed all clinical testing. Prior to testing all participants signed an informed consent that was approved by the Health Sciences Research Ethics Board, Dalhousie University and the Canadian Armed Forces through the Surgeon Generals Health Research Program.



**Figure 4.1:** Classification of groups and breakdown of participants.VAS = Visual analogue scale, RMQ = Roland Morris questionnaire, CIG = Clinical instability group, NCIG = no clinical instability group.

# 4.2.2 Test procedure

Health screening for inclusion/exclusion was done initially over the telephone based on self-report and then confirmed during testing. Participants attended two separate sessions, the first consisted of a postural (including scoliosis and kyphosis), and neurological assessment including reflexes (patellar, Achilles tendon), myotomes and dermatomes conducted by a registered Physiotherapist. Descriptive variables were collected including age, sex, height, weight, occupational activity level (Matheson, 1982), and how they perceive and cope with their pain (as measured by the Pain Catastrophizing Scale (PCS)) (Sullivan et al., 1995). Additional measures were collected in the first session to ensure recovery such as the Visual Analogue Scale (VAS) to measure pain at the beginning and end of the session as well as the Roland Morris Questionnaire (RMQ) to measure disability. Spinal instability tests were also conducted as part of this assessment (Hicks et al., 2005) and were used to define the CIG and NCIG as stated above (Table 4.1). During session 2, participants performed a highly controlled right-toleft transfer task (Figure 3.5) while a comprehensive set of EMG recordings and trunk and pelvis motion data were simultaneously collected. This task was designed to provide a dynamic challenge with constantly changing flexion and lateral flexion moments that the trunk musculature had to respond to (Butler et al., 2010; Hubley-Kozey et al., 2012). The following is a brief description of the transfer task and a more details description is provided in Figure 3.5. The task involved transferring a 2.9Kg load from the right side of their body (at 60° from their body midline) to the left side of their body (60° from the midline of their body). During this task participants were instructed to perform the movement to a 5 second external pace, where the load must be at a unique position every

second (Butler et al., 2010) and were required to minimize pelvis and trunk motion with the use of both motion sensors and tactile feedback (Butler et al., 2010). This task was performed using maximum (elbows fully extended) reach (Figure 3.5). A pressure sensor was fixed to the bottom of the load to indicate when the task started and finished. A photoelectric sensor was positioned at the midline to detect when the participant transferred the load. This set-up allowed detection of the start, middle and end of the movement so that movement times could be compared between groups.

Table 4.1: Definition of clinical stability tests. (Hicks et al., 2005)

Clinical Test	Procedure	Positive
Aberrant motion	The patient is asked to reach down	Any aberrant motions
during lumbar range	as far as possible towards the toes	believed to be associated
of motion.	while keeping the knees straight.	with lumbar spine
		instability occurring
		during the performance of lumbar range of
		motion were noted,
		including an instability
		catch, painful arc of
		motion, "thigh climbing"
		(Gower's sign), or a
		reversal of lumbopelvic
-		rhythm.
Straight leg raise	The patient is supine. The	If greater than 90°
	goniometer is positioned over the	
	center of rotation of the hip joint.	
	The leg is raised passively by the examiner, whose other hand	
	maintains the knee in extension. The	
	leg is raised slowly to the maximum	
	tolerated straight leg raise (not the	
	onset of pain).	
Prone instability test	The patient lies prone with the body	If pain is present in the
	on the examining table and legs over	resting position but
	the edge and feet resting on the	subsides in the second
	floor. While the patient rests in this	position, the test is
	position, the examiner applies	positive.
	posterior to anterior pressure to the	
	lumbar spine. Any provocation of	
	pain is reported. Then the patient lifts the legs off the floor (the patient	
	may hold table to maintain position)	
	•	
	and posterior compression is applied again to the lumbar spine.	

During this task participants were positioned standing at a table adjusted to elbow height. To ensure minimal movement participants were provided tactile feedback applied to their thoracic spinous process using a vertical jig as well as using the above-described motion capture system (Waters et al., 1994). Practice trials were performed until the

participant was comfortable, upon which recording continued until 5 successful trials were recorded. Trials were included if they adhered to the 5 second count and exhibited minimal trunk and pelvic motion.

# 4.2.3 Normalization procedure

Prior to the test trials the participants were asked to perform 8 standardized exercises that require maximum voluntary isometric contractions (MVIC) for normalization purposes (Butler et al., 2010). The purpose of these MVICs was to obtain maximum EMG activation amplitudes to which all data was amplitude normalized so that muscle activation can be expressed as a % MVIC. These included restrained sit-up, resisted lateral bend (left and right), resisted trunk extension, resisted trunk extension with left or right rotation, and resisted seated rotation (left and right). All normalization exercises were held for three seconds and repeated twice with a 2-minute rest between trials resulting in a total of 16 trials.

# 4.2.4 Surface EMG data collection and processing

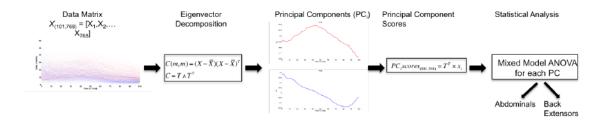
Participants were prepared for data collection using a standardized protocol based on previous work to capture EMG and motion data of the trunk (Butler et al., 2012). For EMG recordings, Ag/AgCl single use disposable surface electrodes (10mm diameter, Red Dot, 3M, London, Ontario, Canada) were placed by a trained researcher based on standardized locations in line with the muscle fibers in a bipolar configuration (interelectrode distance of 25 mm) over 12-bilateral trunk muscle sites as seen in Figure 3.4 (Butler et al., 2009). Abdominal muscle sites included placement over the lower rectus abdominis (LRA), upper rectus abdominis (URA), the anterior, lateral and posterior fibres of the external obliques (EO1, EO2, EO3 respectively), internal obliques

(IO). Posterior sites includederector spinae at the L1 and L3 levels, both 3 cm and 6 cm from the midline representing longissimus and iliocostalis sites respectively (L13, L16, L33, L36); as well as over quadratus lumborum (L48) and multifidus (L52). Specific anatomical landmarks used for these electrode sites have been previously described in detail (Butler et al., 2010) and in Chapter 3.3. Minor adjustments were made to accommodate participant anatomical differences as confirmed by palpation and submaximal validation exercises for each specific muscle site (Butler et al., 2009). Once electrode placement was determined, the skin was prepared for surface electrodes using an alcohol/water solution to improve signal conduction.

EMG signals were pre-amplified (200x) and further amplified using three AMT-8 EMG systems (band pass 10 – 1000 Hz; CMRR = 115db, input impedance 10GΩ; Bortec INC., Calgary Ablerta). Raw EMG signals and event markers (a step voltage change) were digitized at 2000 Hz using a 16 bit resolution analog-to-digital conversion board (National Instruments, CA-1000) and Labview<sup>TM</sup> software (version 7). A 30 Hz high pass filter was used on the EMG signals to minimize noise due to electrocardiographic artifact (Butler et al., 2009). The power spectrum was calculated for each EMG signal and if any abnormal signals were detected, such as a 60 Hz noise from the power system or specific noise from the electromagnetic field generated by the FOB, it was removed with an inverse Fast Fourier Transform filter. All EMG data were corrected for bias and gain, full wave rectified and low pass filtered at 6 Hz using a second order recursive Butterworth filter to yield a linear envelope profile. Data were time normalized from lift off to replace using a linear interpolation algorithm, and then amplitude normalized to the 500 ms peak amplitude from the normalization exercises (Hubley-Kozey & Vezina, 2002). All five

trials were visually inspected for consistency then an ensemble-average waveform was calculated for each muscle and subject.

The time and amplitude normalized ensemble-averaged EMG waveforms were the input to a principal component analysis model. A schematic of the procedure can be found in Figure 4.2. To extract important amplitude and temporal patterns from the EMG waveforms an eigenvector decomposition was performed on the covariance matrix of the ensemble-average profile of the trunk muscles ([32 subjects  $\times$  24 muscles] = 768  $\times$  101). Principal components (PCs) were extracted based on how much variance they represented, and those PCs that together accounted for greater than 95% of the variation were included if they individually explained more than 1% of the variation in the data. Next, each measured waveform was scored (PC score) based on how similar the waveform corresponds to each PC (feature) (Hubley-Kozey et al., 2012; Hubley-Kozey et al., 2014a). PC scores were used in statistical analysis. PC1 scores reflect the overall amplitude of the EMG waveform with a zero score being equivalent to the amplitude of the mean pattern (Hubley-Kozey et al., 2009; Hubley-Kozey et al., 2012). A positive score represents an amplitude value greater than the mean and a negative score less than the mean. For PC2 scores, a zero indicates that the pattern does not contribute to the overall measured pattern, a positive score adds to the mean pattern and a negative score subtracts from it (Hubley-Kozey et al., 2012).



**Figure 4.2:** Schematic representation of the PCA procedure. Two PCs were identified as containing the most relevant features. Modified from(Butler et al., 2009)

# 4.2.5 Motion capture and processing

Following MVIC exercises, participants were prepared for motion analysis data collection. The purpose of measuring motion during the task was to confirm that minimal motion occurred during the task and to determine that similar motion occurred between groups. By controlling the motion occurring at the trunk and confirming that there was no difference in motion between groups we assumed that the EMG recorded was responding to the two main moments (flexion and lateral flexion) created by moving the load during the transfer task. Three-dimensional motion (kinematics) was collected from an electromagnetic Flock of Birds™ (FOB) Motion Capture system (Ascension Technology Inc., Burlington, Vermont). The FOB recorded the angular motion of the pelvis, lumbar spine and thoracic spine throughout the transfer task in 3D with respect to the global coordinate system (x = frontal, y = sagittal, z = transverse). One sensor was placed superior to the left anterior superior iliac crest, the second over the L4 spinous process and the third over the T8 spinous process. Maximum angular displacements for each phase of movement was calculated and compared between groups, providing a quantitative measure of how much motion occurred. Minimal motion was desired and thus, during the collection, any trials identified with excessive motion were redone.

FOB motion data were filtered at 1Hz with a recursive second order Butterworth low pass filter and the maximum angular displacements in all three planes for each sensor were calculated using custom software during the motion relative to the global coordinate system.

# 4.2.6 Strength

Strength variables collected were from trunk flexor and extensor muscle groups and included torques measured from two MVIC exercises performed at the end of the data collection. The participants were positioned in the prone and crook lying positions for lumbar extension and flexion respectively. Each exercise was performed twice and held for a 3-second count with a two-minute rest between repetitions using a Humac Norm Dynamometer (Computer Sports Medicine Inc, Stoughton, MA, USA). The participants were provided with the same verbal encouragement as during the normalization trials.

A 500 msec moving average was used on the torque output for each trial to identify the peak. The maximum torque across the two trials for each condition was used as the maximum strength values for further analysis. Torque was normalized to body mass in Kg to compensate for any anthropometric differences between individuals. Additionally a flexion to extension torque ratio was calculated and compared between groups.

# 4.2.7 Statistical analysis

Means and standard deviations for all continuous variables were calculated and frequency counts for categorical data. Differences between groups for age, height, weight, motion data, BMI, VAS initial, VAS final, RMQ, PCS, normalized and non-

normalized flexion and extension torques as well as the flexion to extension ratio were tested using Student T-tests ( $\alpha$  = 0.05). ANOVAs were used to test the different PC scores. Due to the small sample size, the exploratory nature of this study and the need for an alpha adjusted for multiple comparisons, an  $\alpha$  = 0.10 was used for interaction while  $\alpha$  = 0.05 was maintained for main effects. Homogeneity of variance and normality of the distributions were checked for demographic data, motion variables and muscle activation PC scores. If data was found to not be normally distributed, Johnson transformations were applied. Fisher's exact test was used to test for differences in occupational activity level, which is a standardized self report measure on job demands ranging from sedentary to very heavy (please see Appendix 2 for an explanation). All tests were performed using Minitab (Minitab Inc, State Collage, PA, version 17)

Specifically two-factor (group and muscle) ANOVA models tested for differences in PC scores for the back extensor and abdominal muscles separately. Post hoc Tukey comparisons were applied to the significant findings. This resulted in 4 ANOVA's (two for the abdominals and two for the low back musculature).

### 4.3 RESULTS

# 4.3.1 Demographic, Motion and timing variables

There were 32 participants classified into two groups: CIG (n=12) and NCIG (n=20). Descriptive data for both groups are found in Table 4.2. The only significant differences (p<0.05) between groups for demographic variables were higher occupational activity level found in the CIG. As the average time from injury to the first session was greater than 40 days, the likelihood that any instability found was due solely to transient issues (creep and/or tension-relaxation) is low.

**Table 4.2:** Subject demographics, mean (SD). Bolded values are significantly different between groups.

Variable	CIG $(n = 12)$	NCIG $(n = 20)$	P-value
Age (yr)	36.2(11.1)	38.8 (9.4)	0.501
Height (cm)	171.6 (7.7)	173.3 (6.7)	0.523
Weight (kg)	91.0(15.6)	95.3 (14.1)	0.445
$BMI (m^2/kg)$	30.8 (4.0)	31.6 (3.7)	0.550
VAS initial	10.8 (8.3)	8.7 (7.9)	0.481
VAS Final	16.0 (14.4)	12.4 (12.8)	0.484
RMQ	4.0 (4.3)	2.9 (3.3)	0.457
PCS	12.2 (12.0)	7.7 (7.7)	0.324
Time from injury to first session (days)	42.4 (21.4)	54.2 (22.3)	0.160
Time between sessions (days)	8.5 (6.1)	7.7 (4.9)	0.668
Occupation activity level	2.3	1.9	0.018
Number of Female	2	3	

Data from the FOB motion sensors indicated that motion from all three sensors was less than  $7^{\circ}$  in all three directions (see Table 4.3). The greatest motion was seen in the transverse plane (axial rotation) for all three sensors, which would have minimal effect on the flexor moments. There was no significant differences between groups in any of the directions ( $\alpha = 0.05$ ), thus confirming that similar trunk and pelvic motion occurred during the testing between groups and that neither trunk nor pelvic motion contributed to any group differences observed. The total time to complete the task was  $4.6 \pm 0.2$  s for the CIG and  $4.4 \pm 0.4$  s for the NCIG with no significant difference found (p > 0.05) between groups for the time to complete the task.

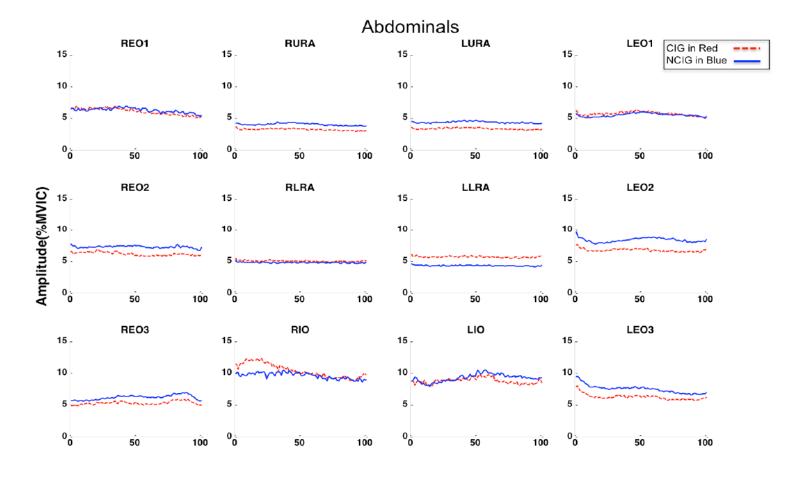
**Table 4.3:** Trunk and pelvic motion data in degrees mean (SD). No significant differences between groups (p > 0.05).

	T8 sensor		
	Frontal	Sagittal	Transverse
CIG	2.6 (1.2)	3.8 (2.2)	6.7 (3.0)
NCIG	2.2 (1.3)	3.6 (2.2)	6.6 (2.8)
		L4 senso	<u>r</u>
	Frontal	Sagittal	Transverse
CIG	1.1 (0.4)	1.3 (1.0)	3.6 (1.3)
NCIG	1.2 (0.5)	1.3 (1.2)	3.1 (1.3)
	Il	iac crest se	nsor
	Frontal	Sagittal	Transverse
CIG	2.7 (1.0)	2.1 (1.5)	4.4 (2.0)
NCIG	2.5 (1.4)	1.3 (0.6)	4.2 (1.8)

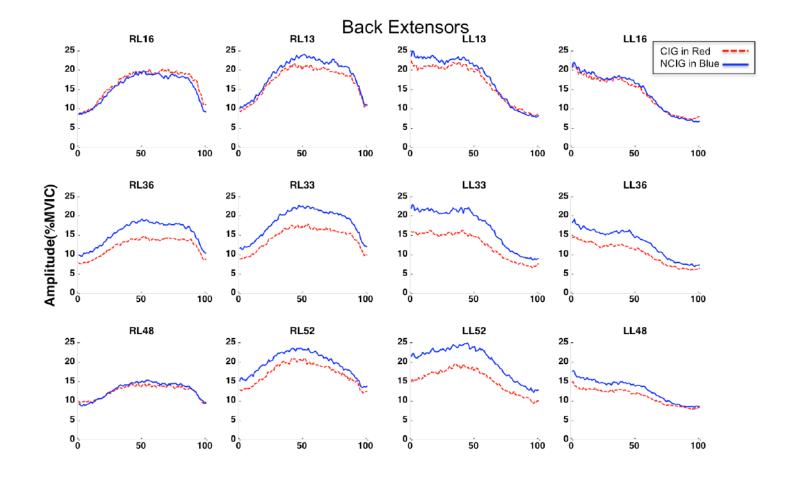
# 4.3.2 EMG waveform analysis

Average EMG waveforms for all abdominal muscle sites during the transfer task with maximal reach are depicted in Figure 4.3. Qualitatively there is no systematic amplitude difference between groups although the posterior and lateral external oblique fibers and upper rectus abdominus (LEO3, LEO2, REO3, REO2, LURA, RURA) have lower activation amplitudes in the CI group. Qualitatively only the internal obliques (RIO and LIO) have deviations in temporal patterns between groups.

Average EMG waveforms for all back muscle sites during the transfer task with maximal reach are depicted in Figure 4.4. Qualitatively the CIG has lower amplitudes for the majority of the muscle sites with the exception of RL16 and RL48, but no pattern or temporal differences were evident. While the waveforms for the abdominals were relatively constant, the ones for the back show more variability as would be expected as the muscles respond to changing flexion and lateral flexion moments. This is demonstrated as an increase in activation of the right-sided back muscles and a decrease in the left-sided back muscles as the mass was transferred from the right side of the body to the left.



**Figure 4.3:** Ensemble average waveforms for each of the 12 abdominal muscle sites for each group. Averaged waveforms for the CIG group are shown in dashed red and NCIG in blue. X-axis is percentage of the task, y-axis is amplitude (%MVIC). EO1 = external obliques (anterior fibers); EO2 = external obliques (lateral fibres); EO3 = external obliques (posterior fibres); URA = upper rectus abdominus; LRA = lower rectus abdominus; IO = internal obliques.



**Figure 4.4:** Ensemble average waveforms for each of the 12 back muscle sites. Averaged waveforms for the CI group are shown in dashed red and no CI group in blue. L13, L16, L33, L36 = Lumbar erector spinae at L1 and L3 at 3 and 6 cm from the midline to represent the longissimus and iliocostalis muscle sites, respectively; L48 = quadratus lumborum at L4 at approximately 8 cm from the midline; L52 = multifidus at L5 at 1–2 cm from the midline.

Two principal patterns extracted from the PCA for the combined back and abdominal muscle sites explained 97.2% of the variance (PC1 explained 90.3%, PC2 6.9%). Means and standard deviations of PC scores for all abdominal and back muscles for both groups are found in Tables 4.4 and 4.5 respectively. ANOVA results are in Table 4.6.

Table 4.4: Mean (SD) PC scores for the abdominal muscles, by group and muscle.

Muscle	PC1Pooled	PC2 CIG	PC2 NCIG
RLRA	-50.5 (40.0)	-1.2 (2.2)	-1.4 (1.7)
LLRA	-49.6 (40.0)	-1.6 (2.1)	-1.2 (1.2)
RURA	-63.3 (21.5)	-0.2 (0.7)	-0.7 (0.8)
LURA	-61.1 (27.6)	-0.6 (0.7)	-1.3 (1.1)
REO1	-37.8 (42.3)	1.9 (6.3)	-0.3 (5.0)
LEO1	-43.5 (36.3)	-1.7 (3.3)	-3.1 (3.4)
REO2	-32.6 (40.7)	-0.5 (3.2)	-2.5 (2.8)
LEO2	-24.2 (49.7)	-1.2 (2.3)	-3.5 (2.5)
REO3	-42.4 (32.8)	-3.4 (2.8)	-5.3 (5.6)
LEO3	-31.0 (39.5)	0.7 (2.6)	2.4 (4.0)
RIO	0.3 (70.7)	5.6 (9.0)	-0.6 (4.1)
LIO	-9.6 (55.6)	-3.3 (7.2)	-7.5 (7.3)
Mean (SD)	-37.1 (46.2)	-0.4 (4.8)	-2.1 (4.4)

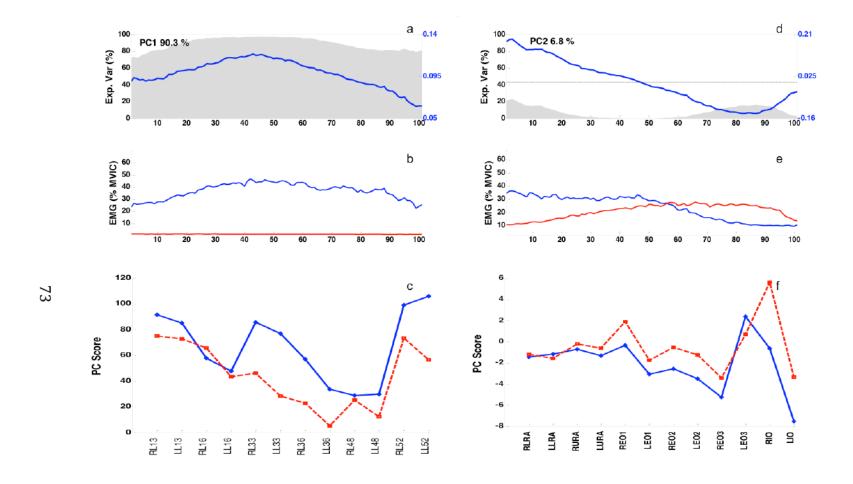
Table 4.5: Mean (SD) PC scores for the back muscles, by group and muscle.

Muscle	PC1 CIG	PC1 NCIG	PC2 Pooled
RL13	75.2 (97.7)	91.6 (91.9)	-31.9 (16.6)
LL13	72.9 (78.6)	85.1 (67.6)	38.1 (15.4)
RL16	65.8 (81.4)	57.9 (71.2)	-33.4 (23.7)
LL16	43.5 (66.9)	47.9 (54.6)	37.2 (23.6)
RL33	46.1 (87.3)	85.6 (88.1)	-25.5 (15.1)
LL33	28.4 (60.7)	77.0 (79.0)	29.2 (13.8)
RL36	22.7 (69.3)	57.0 (72.3)	-24.3 (16.7)
LL36	5.4 (54.5)	33.6 (57.8)	24.5 (14.5)
RL48	25.4 (56.0)	28.9 (53.8)	-17.0 (10.7)
LL48	12.4 (55.7)	30.0 (63.0)	16.5 (9.8)
RL52	73.4 (70.2)	99.0 (82.8)	-13.0 (14.4)
LL52	56.6 (74.5)	106.0 (100.0)	14.9 (10.6)
Mean (SD)	43.7 (73.4)	66.5 (77.8)	1.3 (31.3)

**Table 4.6:** Significant results for the group by muscle ANOVAs.

	Abdominals		Back Extensors	
	PC1	PC2	PC1	PC2
CIG/NCIG (n = 32)				
Group	p = 0.467	p = 0.011	p = 0.236	p = 0.307
Muscle	p < 0.001	<i>p</i> < 0.001	<i>p</i> < 0.001	<i>p</i> < 0.001
Group*Muscle	p = 0.252	p = 0.011	p = 0.064	p = 0.548

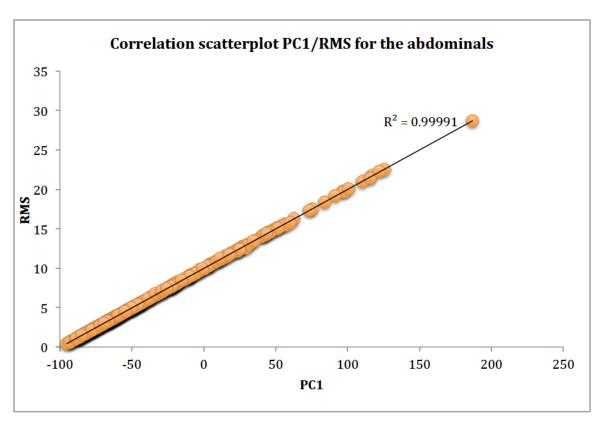
PC1 captured the overall amplitude and shape characteristics of the waveform (see Figure 4.5(a)) as demonstrated by the high correlation between the PC1 score and RMS amplitude (Figures 4.6(a) and (b)). Higher PC1 scores were associated with higher overall amplitudes (see high-low waveforms in Figure 4.5(b)). A significant group by muscle interaction was found (p < 0.10) for PC1 in the back muscles (Table 4.6) and post hoc analysis showed fewer between-muscle differences in the CIG (Table 4.8). Back extensor muscle PC1 scores divided by group are shown in Figure 4.5(c) to illustrate the significant group by muscle interaction. A significant muscle main effect was also found (p < 0.05) for PC1 in the abdominals. Post hoc analysis of the muscle main effects can be found in Table 4.7

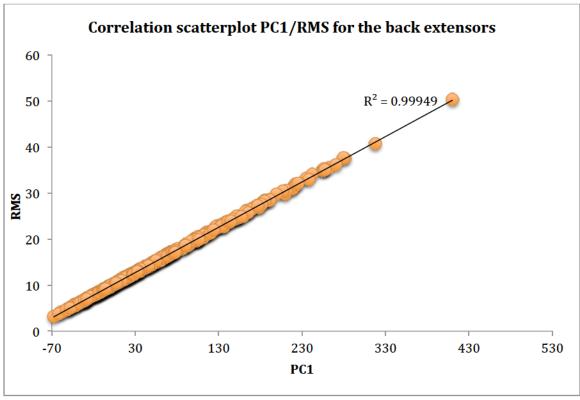


**Figure 4.5:** Principal components (features) for (a) PC1, (d) PC2. PC1 explains 90.3% of the waveform variance with PC2 explaining 6.8%. Ensemble average waveforms for the 5 highest (blue) and 5 lowest (red) scores for PC's 1 and 2 (b and e respectively) are shown to aid with interpretation. PC 1 scores for back extensor muscle interactions are graphed in c, blue is NCIG and red is CIG. PC 2 scores for abdominal muscle interactions are graphed in f, blue is NCIG and red is CIG.

**Table 4.7:** Post hoc Muscle main effects for abdominal and back muscle PC scores. PC1 abdominals and PC2 back extensors. Muscles with the same letter are not significantly different from each other (p > 0.05).

Back e	xtensors		Abdominals
	PC2		PC1
RL13	d	RLRA	def
LL13	a	LLRA	def
RL16	d	RURA	f
LL16	a	LURA	ef
RL33	cd	REO1	cde
LL33	ab	LEO1	cdef
RL36	cd	REO2	bcd
LL36	ab	LEO2	bc
RL48	c	REO3	cdef
LL48	b	LEO3	bcd
RL52	c	RIO	a
LL52	b	LIO	ab





**Figure 4.6:** Correlation scatterplots between PC1 and EMG RMS values for the abdominals (a) and the back extensors (b) with R<sup>2</sup> values.

PC2 captured the muscle response to the changing lateral flexion moment (Figure 4.5(d)). A positive score depicts a pattern of high activity initially and then a gradual decrease in activity as the lateral flexion moment moves from right to left as illustrated for the high low scores in Figure 4.5(e). A negative PC2 score indicates the opposite pattern. A significant group by muscle interaction was found (p < 0.05) for PC2 in the abdominal muscles (Table 4.6) and post hoc analysis showed fewer between muscle differences in the CIG in terms of temporal patterns responding to the right lateral flexor moment (Table 4.8). Abdominal PC2 scores divided by group are shown in Figure 4.5(f) to illustrate the significant group by muscle interaction. A significant muscle main effect was also found (p < 0.05) for PC2 in the back extensors and these results were found to be consistent with previous studies (Hubley-Kozey et al., 2014a). Post hoc analysis of the muscle main effect can be found in Table 4.7.

**Table 4.8:** Post hoc results of the group by muscle interactions for both PC1 in the back extensors and PC2 in the abdominals. Muscles with the same letter are not significantly different from each other (p > 0.05).

Back extensors			Abdominals		
	PC1 CIG	PC1 NCIG		PC2 CIG	PC2 NCIG
RL13	abcde	abc	RLRA	bcde	bcde
LL13	abcdef	abc	LLRA	bcde	bcde
RL16	abcdefg	bcdefg	RURA	bcd	bcd
LL16	cdefg	cdefg	LURA	bcde	bcde
RL33	bcdefg	abc	REO1	abc	bcd
LL33	defg	abcd	LEO1	bcde	cde
RL36	defg	bcdefg	REO2	bcde	cde
LL36	g	defg	LEO2	bcde	def
RL48	defg	efg	REO3	cdef	ef
LL48	fg	defg	LEO3	abcd	ab
RL52	abcdef	ab	RIO	a	bcd
LL52	abcdefg	a	LIO	cdef	f

# 4.3.3 Strength

Strength variables for each group, measured as the torque produced during a MVIC are found in Table 4.9. Differences were found (p < 0.05) between groups for both the flexion and extension torques when normalized to body weight, but not for the non-normalized torques nor for the flexion to extension torque ratio.

**Table 4.9:** Strength variables. Significant findings (p < 0.05) are bolded.

Variable	CIG (n = 12)	NCIG (n = 20)	P-value
Flexion Torque (Nm)	169.8 (54.5)	157.2 (40.3)	0.089
Extension Torque (Nm)	227.5 (82.2)	207.9 (89.8)	0.137
Flexion Torque normalized to BW (Nm/kg)	1.9 (0.5)	1.7 (0.4)	0.036
Extension Torque normalized to BW (Nm/kg)	2.5 (0.7)	2.1 (0.8)	0.021
Flexion/Extension ratio	0.8 (0.2)	0.9 (0.3)	0.321

# 4.4 DISCUSSION

This study showed that differences exist in objective physiologically based measures between a LBI recovered group with clinical instability based on a modified Hicks protocol and a group without. Proportion of participants from this study in the CIG (37.5%) is consistent with other studies that used the Hicks CPR (33.3% from Hicks et al. 2005 and 38.1% from Rabin et al. 2014). Since the CIG and NCIG were well matched based on the majority of the demographic characteristics, the between group differences were not likely attributed to these factors. Overall, the pain levels and disability reported from both groups were low (Table 4.2) supporting the recovery classification.

Additionally, both groups had similar gender proportions minimizing confounding factors for between group differences due to gender that have been found previously with activation amplitudes during the transfer task (Hubley-Kozey et al., 2012). Finally, the highly controlled task minimized differences in task performance, with both groups demonstrating compliance with the motion and time constraints. The greatest motion (less than 7°) was in the transverse plane (axial rotation) and resulted in a velocity of less

than 1.5 degrees/second, which would have minimal effect on the magnitude of force acting on the spine in the sagittal and frontal plane. The significant difference between groups that were found in the demographic variables was in occupational activity level, which interestingly was higher in the CIG.

The two principal patterns extracted from the PCA explained 97.2% of the variance and displayed similar shapes to previous studies (Hubley-Kozey et al., 2014a). The first principal pattern extracted from the PCA explained 90.3% of the variance and captured the overall amplitude and shape characteristics of the waveform, while the second pattern explained 6.9% of the variance and captured the muscle response to the changing lateral flexion moment. The general shape of PC1 indicates moderate activity at the beginning of the task in response to a more lateral moment. As the load is transferred at the midline, muscle activity is at its peak, responding to a more pure flexion moment reflecting an increase in PC1. A drop in activity follows this peak as it moves once again to a more lateral moment on the opposite side. As seen in Figures 4.6(a) and (b), the PC1 scores among muscles correlate highly with the normalized root mean squared amplitude as would be expected with PC1 representing the overall amplitude which is similar to previous work (Hubley-Kozey et al., 2009). Since it has been shown that both a decrease in passive stiffness (Brown & Potvin, 2005; Brown & McGill, 2008; Hodges, van den Hoorn, Dawson, & Cholewicki, 2009; Moreside, Vera-Garcia, & McGill, 2007; Stokes & Gardner-Morse, 2003) and in increase in imposed instability (Cholewicki, McGill, & Norman, 1995; Cholewicki, Panjabi, & Khachatryan, 1997; Granata & Orishimo, 2001) leads to increases in activation amplitudes in an effort to compensate for stability requirements, it was hypothesized that an increase in activation amplitudes or PC1 would

be found in our CIG. This increase in activation amplitudes (PC1) was not found in the CIG with similar amplitudes for the abdominals and lower activation amplitudes for specific back extensors muscles in the CIG compared to NCIG. Since strength can influence activation (Alkner et al., 2000; Lawrence & Luca, 1983; Woods & Bigland-Ritchie, 1983) and the CIG had greater strength compared to NCIG, the decreased activation in the back extensors would be expected. The result that cannot be explained by strength differences is the fewer amplitude differences between the muscles in the CIG compared to NCIG. As with the back extensors the CIG also had greater strength in the abdominals. Following the same logic, a decrease in activation should be expected between the groups for the abdominal muscles. Since there is no difference, the similar percentage of activation could be indicative of higher relative active stiffness and cocontraction in the CIG in response to a decrease in passive stability. The other significant finding for PC1 was the muscle main effect where the amplitude differences found between muscles for the abdominals was consistent with previous research (Hubley-Kozey et al., 2014a). These findings showed that the IO amplitude was significantly higher than all other abdominal sites and the EO sites were higher than RA (Table 4.8).

Another interesting finding was the temporal patterns that captured the responsiveness of the muscles to the lateral flexor moment (PC2) throughout the dynamic task representing coordination of activity among the muscles. The significant group by muscle interaction for the abdominals shows that the two groups responded differently to the lateral flexor moment. Specifically, the CIG had fewer temporal differences between abdominal muscles in responding to the right lateral flexor moment (Table 4.8). Fewer temporal differences is indicative of increased co-contraction (Hubley-Kozey & Vezina,

2002), which would be expected in a group with clinical instability as they increase active stiffness in response to a decrease in passive stability. The temporal differences found between muscles for the back extensors was similar to previous finding (Hubley-Kozey et al., 2014a) with significant differences between sides for each back extensor muscle site with all left sided PC2 scores positive and right sided sites negative (Table 4.7). Specifically, the more superior sites had PC2 scores significantly greater in absolute magnitude then ipsilateral inferior sites.

In summary, the significant group by muscle interactions found in PC1 scores for the back musculature and PC2 scores for the abdominals indicates that there was not a systematic difference in activation among be muscles between groups, nor were the differences in response to the lateral flexion moment for the abdominals. Rather there were specific differences between muscles that represented less variability in muscle amplitudes in the back extensors and temporal responses in the abdominals in the CIG. These differences could been seen as a compensatory mechanism to increase active stiffness in response to a decreased in passive stability as would be expected in a group with a classification of clinical instability.

The significant difference in strength between the two groups was a surprise in that it ran contrary to the original hypothesis. Previous research showed decreases in back extensor strength in both a chronic LBP population and one with clinical instability (Alston et al., 1966; Davarian et al., 2012; Descarreaux et al., 2004; Gruther et al., 2009; Mannion et al., 2001). Therefore, it was initially thought that the CIG would have decreased back muscle strength and consequently an increase in the flexion to extension ratio. However the CIG had greater strength of both the abdominals and the back

extensors when normalized to body mass compared to the NCIG and subsequently no difference in the flexion/extension ratio. Upon further investigation, it was found that the CIG also rated their jobs as significantly heavier than the other group, a factor that could explain the strength difference. Increased loading of the spine has been associated with decreased disc height, joint space narrowing and an increase in anterior/posterior shear forces (Marras & Granata, 1997; Shan, Zhang, Zhang, Chen, & Wei, 2012; Suri et al., 2014), which could lead to a decrease in passive stability. Additionally, increased occupational loads and repetition has been associated with increased passive instability due to creep and the development of tension-relaxation (Solomonow, 2009). The higher job demands in the CIG could explain the increased strength in that group as necessary to meet the specific job demands (Schibye, Hansen, Sogaard, & Christensen, 2001; Tammelin, Nayha, Rintamaki, & Zitting, 2002).

While this study showed that the LBI group could be differentiated based on the modified Hick's protocol and that trunk muscle activation patterns were different between groups, an important next step is to determine the predictive capability of the two tests. Future work could conduct a follow-up study to determine re-injury status to determine if the clinical tests used or the objective measures have predictive validity for assessing risk of LBI recurrence.

If predictive ability can be shown in the clinical tests, objective measures or some combination of the two, then it could aid in the creation and direction of treatments to not only increase their efficacy but also to possibly stop the transition of recurrent LBP into chronic.

### 4.6 CONCLUSION

In conclusion, there were differences in activation amplitude patterns for the back extensor muscles and temporal patterns for the abdominal muscles between those deemed recovered from LBP classified as having a clinical instability versus those that do not. Those with clinical instability employed different activation strategies including; increased temporal synergies of the abdominals in response to the right lateral flexor moment, increases in the activation amplitudes of specific back extensor muscles and increased co-contraction seen as greater relative overall activation amplitude in the abdominals. These findings are consistent with a hypothesis that individuals testing positive for instability on clinical tests would employ strategies such as these as a compensatory mechanism to increase active stiffness. These finding could serve to reinforce the validity of the modified Hicks CPR in determining clinically instability in individuals.

# CHAPTER 5: COMPARING TRUNK MUSCLE ELECTROMYOGRAPHIC PATTERNS IN A RECOVERED LBI POPULATION BETWEEN THOSE WHO TESTED POSITIVE OR NEGATIVE ON THE PRONE INSTABILITY TEST.

### 5.1 INTRODUCTION

Increasing emphasis is being put on groupings of tests that are designed to improve decision-making in clinical practice. Certain clinical prediction rules (CPRs) have been developed to assist with subgrouping low back injured (LBI) patients into homogeneous groups that are useful in guiding management and decision making. One such subgroup is patients who have a clinical instability in their lumbar spine. Hicks et al. in 2005 developed a CPR to determine who would be successful in an exercise stabilization program, hypothesizing that if someone was successful with stabilization exercises they probably have a clinical instability (Hicks et al., 2005). The Hicks CPR consists of four items: i) age greater than 40 years, ii) passive straight leg raise greater than 91°, iii) aberrant motion present, iv) positive prone instability test (PIT). Individuals are classified with a clinical instability if they have at least three of these items positive (Delitto et al., 2012; Hicks et al., 2005). A follow-up validation study by Rabin et al. found that a modification of the Hicks CPR, which reduced it to two tests, the PIT and aberrant motion, increased its predictive power (Rabin et al., 2014).

If the theoretical basis for the individual tests in the Hicks CPR modified by Rabin is examined; both tests (the PIT and aberrant motion) examine the ability of the active subsystem to compensate for a decrease in passive stiffness (Biely et al., 2014; Magee, 1997). The PIT achieves this by noting a reduction in symptoms with testing and

can be found in symptomatic and asymptomatic individuals (Magee, 1997), while a positive aberrant motion test occurs when symptoms increase and usually is associated in individuals who are symptomatic (Biely et al., 2014). This calls into question the utility of aberrant motion testing by itself or in a CPR on a recovered asymptomatic population, and in that instance would the PIT be just as selective as the entire CPR? This study is a first step toward addressing this question.

All participants in this study were deemed recovered from a LBI and thus are not symptomatic. Currently all CPR's for LBIs look at individuals that are symptomatic (Childs et al., 2004; Flynn et al., 2002; Hicks et al., 2005) while none have given consideration to the benefit of testing individuals who are recovered possibly for return to work or discharge from active treatment. Although clinical tests have not explored this recovered population, objective physiologically based measures such as EMG, have shown the ability to detect individuals recovered from a LBI that have altered neuromuscular responses compared to individuals that never had a LBI (Butler et al., 2012; Cholewicki et al., 2005) and also have shown modest evidence in the ability to predict recurrence in a recovered population (Hubley-Kozey et al., 2014a). Therefore, even though the PIT has not been used on a recovered population, for discharge planning, or return to work, this study aimed to explore the possibility of tests, and perhaps in the future, a CPR being used or developed for a recovered population.

With this in mind, the purpose of this study was to determine whether there are differences in trunk muscle activation patterns during a standardized functional task in those deemed recovered from a LBI when they are scored positive on the PIT versus those that scored negative. This preliminary assessment of results could determine

whether the PIT and the modified Hicks CPR result in similar differences in EMG patterns.

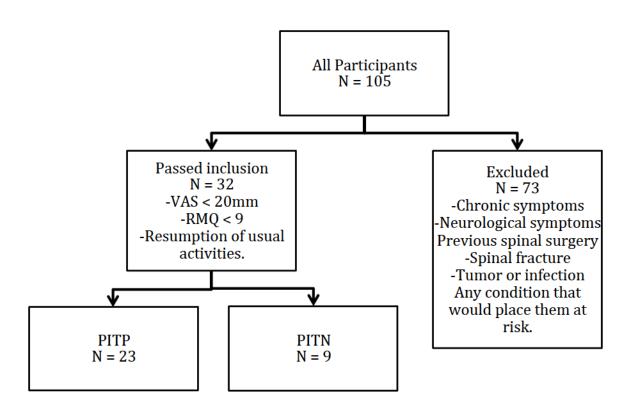
### 5.2 METHODOLOGY

# 5.2.1 Participants

The study population included 32 participants with a LBI; all were recruited from the military hospital at CFB Halifax representing an inclusive sample of all military members stationed at CFB Halifax. Inclusion criteria included being in the sub-acute phase (between 4 and 12 weeks post injury) of a LBI (pain between the lower ribs and the gluteal folds as a result of a specific event). Additionally the participants were deemed recovered as indicated by experiencing minimal pain (VAS < 20mm), minimal disability (RMQ < 8) and either already resuming usual activities or being within a week of resumption of usual activities. Excluded were those whose pain was to a specific cause such as a fracture, tumour or infection, those that had a previous spinal surgery or were experiencing chronic LBP (as defined as pain lasting longer than 12 weeks). Participants were also excluded if they had neurological symptoms or other unrelated issues that would preclude them from completing the experimental task. Figure 5.1 outlines the breakdown of participants with inclusion and exclusion criteria as well as group definitions.

In this study participants were divided into groups based on their results from the prone instability test with the two groups being positive on the prone instability test (PITP) or negative (PITN). The PIT was performed using a standardized protocol (Magee, 1997) with the subject lying prone on an examination table with their legs hanging over the end of the table and their feet resting on the floor. An orthopaedic

physiotherapist with 12 years of experience applied a posterior to anterior directed force to a painful segment of the lumbar spine. The participant was then directed to lift their legs off the floor and the same force was reapplied to the same segment. If the symptoms were reduced with the legs lifted the test was positive indicating that the muscle action compensates for the instability (Magee, 1997). Prior to testing all participants signed an informed consent that was approved by the Health Sciences Research Ethics Board, Dalhousie University and the Canadian Armed Forces through the Surgeon Generals Health Research Program.



**Figure 5.1:** Classification of groups and breakdown of participants. VAS = Visual analogue scale, RMQ = Roland Morris questionnaire, PITP = Positive prone instability test, PITN = negative prone instability test.

# 5.2.2 Test procedure

General screening was done initially over the telephone and then confirmed during testing. Participants attended two separate sessions as described in Chapter 3. The spinal stability tests conducted as part of the clinical assessment were used to define the PITP and PITN groups as stated above. The same task was performed as described in Chapter 3.

Electrodes were placed over 12 muscles sites bilaterally, all normalization trials, strength measures, motion capture set-up and surface EMG collection were done according to Chapter 3. Additionally, all processing and statistical analysis followed previously described protocols.

### 5.3 RESULTS

# 5.3.1 Demographic and Motion data

There were 32 participants classified into two group: PITP (23) and PITN (9).

Descriptive data for both groups are found in Table 5.1. No significant differences were found between the groups for any of the demographic or strength variables measured.

**Table 5.1:** Subject demographics, mean (SD).

	PITP	PITN	
Variable	(n = 23)	(n = 9)	P-value
Age (yr)	38.1 (10.2)	37.1 (9.9)	0.808
Height (cm)	173.1 (7.3)	171.6 (6.3)	0.581
Mass (kg)	93.8 (14.9)	93.2 (14.4)	0.908
$BMI (m^2/kg)$	31.2 (3.8)	31.6 (4.0)	0.802
VAS initial	9.0 (7.0)	10.8 (10.6)	0.581
VAS Final	13.9 (13.9)	13.3 (12.6)	0.914
RMQ	3.3 (3.4)	3.4 (4.6)	0.901
PCS	10.2 (11.4)	7.3 (7.9)	0.501
Time from injury to first session (days)	47.3 (21.4)	55.6 (25.2)	0.375
Time from injury to second session (days)	56.1 (22.1)	61.3 (24.7)	0.587
Time between sessions (days)	8.7 (5.6)	6.0(4.0)	0.193
Occupation activity level	2.3	1.8	0.681
Number of Female	3	2	

Data from the FOB motion sensors indicated that motion from all three sensors was less than  $7^{\circ}$  in all three directions (see Table 5.2). The greatest motion was see in the transverse plane for all three sensors, which corresponds to axial rotation and would have minimal effect on the flexor moments. The only significant effect between groups (p < 0.05) was for the iliac crest sensor indicating greater motion for the PITN group in the transverse plane (axial rotation). However, both groups had minimal total movement for the task ( $<5.5^{\circ}$ ) and the difference between groups for axial rotation of the pelvis was 1.5°. Due to the minimal overall movement and the small difference in the one significant finding, it can be considered that similar trunk and pelvic motion occurred during the testing between groups and that neither trunk nor pelvic motion contributed to any group differences observed. The total time to complete the task was  $4.5 \pm 0.3$  s for the PITP group and  $4.5 \pm 0.4$  s for the PITN group. No significant difference was found (p > 0.05) between groups for the time to complete the task.

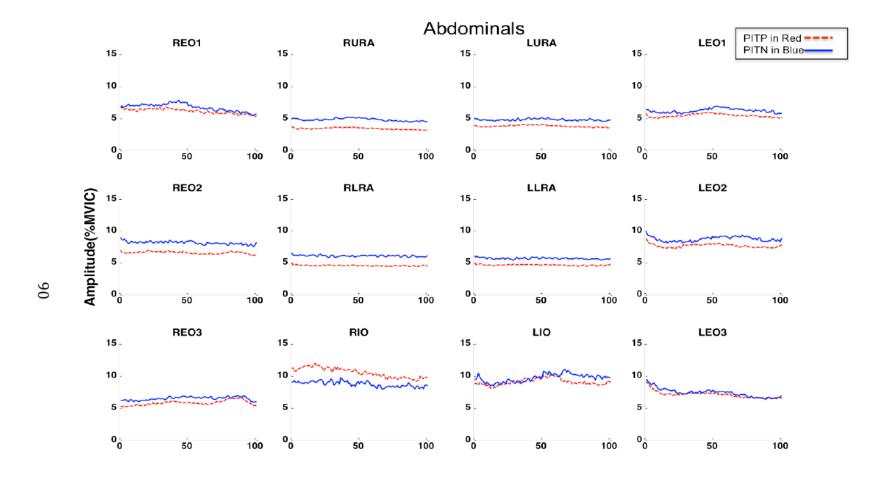
**Table 5.2:** Trunk and pelvic motion data, mean (SD). Significant differences (p < 0.05) are bolded.

	T8 sensor		
	Frontal	Sagittal	Transverse
PITP	2.4 (1.4)	3.3 (1.9)	6.6 (3.2)
PITN	2.2(0.9)	4.5 (2.4)	6.7(2.0)
		L4 senso	<u>r</u>
	Frontal	Sagittal	Transverse
PITP	1.1 (0.4)	1.2 (0.6)	3.2 (1.3)
PITN	1.2(0.5)	1.5 (1.5)	3.4 (1.3)
	<u>Il</u> :	iac crest se	nsor
	Frontal	Sagittal	Transverse
PITP	2.8 (1.4)	1.7 (1.3)	3.8 (1.7)
PITN	2.1(0.6)	1.5(0.5)	5.3 (1.6)

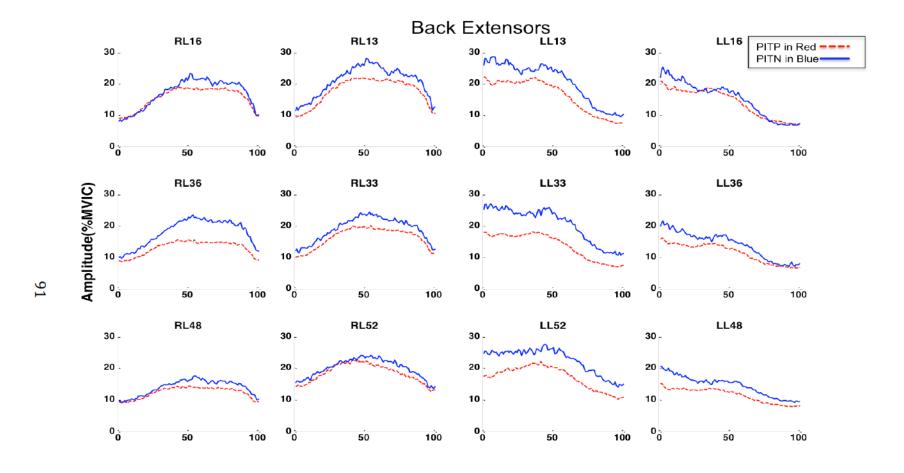
# 5.3.2 EMG waveform analysis

Average EMG waveforms for all abdominal muscle sites during the transfer task with maximal reach are depicted in Figure 5.2. Qualitatively there is no systematic amplitude difference between groups although the rectus abdominus (left and right URA and LRA) and the lateral fibers of the external obliques (LEO2, REO2) have lower activation amplitudes and the right internal oblique (RIO) has higher activation amplitudes in the PITP group. Qualitatively only the internal obliques (RIO and LIO) have deviations in temporal patterns between groups.

Average EMG waveforms for all back extensor muscle sites during the transfer task with maximal reach are depicted in Figure 5.3. Qualitatively the PITP group has lower amplitudes for the majority of the muscle sites with the exception of RL16, LL16, RL48, and RL52. Additionally, there do seem to be some pattern differences, especially with RL36 and LL52. While the waveforms for the abdominals were relatively constant, the ones for the back show more variability as would be expected as the muscles respond to changing flexion and lateral flexion moments. This is demonstrated as an increase in activation of the right-sided back muscles and a decrease in the left-sided back muscles as the mass was transferred from the right side of the body to the left.



**Figure 5.2:** Ensemble average waveforms for each of the 12 abdominal muscle sites. Averaged waveforms for the PITP group are shown in dashed red and PITN group in blue. EO1 = external obliques (anterior fibers); EO2 = external obliques (lateral fibres); EO3 = external obliques (posterior fibres); URA = upper rectus abdominus; LRA = lower rectus abdominus; IO = internal obliques



**Figure 5.3:** Ensemble average waveforms for each of the 12 back muscle sites. Averaged waveforms for the PITP group are shown in dashed red and PITN group in blue. L13, L16, L33, L36 = Lumbar erector spinae at L1 and L3 at 3 and 6 cm from the midline to represent the longissimus and iliocostalis muscle sites, respectively; L48 = quadratus lumborum at L4 at approximately 8 cm from the midline; L52 = multifidus at L5 at 1–2 cm from the midline.

Two principal patterns extracted from the PCA for the combined back and abdominal muscle sites explained 97.2% of the variance (PC1 explained 90.3%, PC2 6.9%). Means and standard deviations of PC scores for both groups are found in Table 5.3. Means and standard deviations for PC2 scores for the back extensor muscles separated by group are in Table 5.4. ANOVA results are in Table 5.5

**Table 5.3:** Mean (SD) PC scores for both groups.

Variable	PITP	PITN
PC1 abs	-39.1 (46.8)	-30.3 (41.8)
PC2 abs	-1.3 (4.5)	-2.0 (4.9)
PC1 back	47.7 (69.2)	83.9 (88.8)
PC2 back	1.7 (14.9)	1.6 (18.7)

Table 5.4: Means (SD) for PC2 scores for the back extensors separated by group.

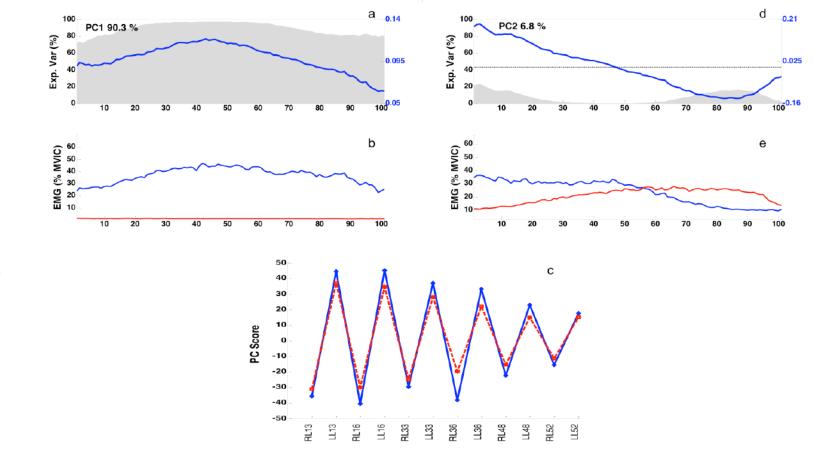
Muscle	PC2 PITP	PC2 PITN
RL13	-31.2 (16.3)	-35.7 (15.5)
LL13	37.1 (14.4)	44.6 (21.2)
RL16	-30.0 (24.9)	-40.5 (18.2)
LL16	34.6 (25.5)	45.2 (26.2)
RL33	-25.0 (14.1)	-29.6 (18.0)
LL33	28.0 (12.3)	37.0 (21.7)
RL36	-19.7 (16.4)	-38.2 (20.4)
LL36	22.2 (14.9)	33.2 (21.1)
RL48	-15.4 (7.7)	-22.4 (16.3)
LL48	15.1 (7.3)	23.0 (16.8)
RL52	-11.4 (13.8)	-15.5 (14.4)
LL52	15.2 (11.3)	17.6 (14.7)
Mean (SD)	1.6 (14.9)	1.6 (18.7)

 Table 5.5: Significant results (shaded in grey) for the group\*muscle ANOVAs

	Abdominals		Back Extensors	
	PC1	PC2	PC1	PC2
PITP/N (n = 32)				
Group	p = 0.310	p = 0.282	p = 0.186	p = 0.992
Muscle	<i>p</i> < 0.001	<i>p</i> < 0.001	<i>p</i> < 0.001	<i>p</i> < 0.001
Group*Muscle	p = 0.934	p = 0.594	p = 0.333	p = 0.012

PC1 captured the overall amplitude and shape characteristics of the waveform (Hubley-Kozey et al., 2014a) (see Figure 5.4(a)) as demonstrated by the high correlation between the PC1 score and RMS amplitude (Figures 4.6(a) and (b)) Higher PC1 scores are associated with higher overall amplitudes (see high-low waveforms in Figure 5.4(b)). No significant group main effects or group by muscle interactions were found for PC1 (p > 0.05). A significant muscle main effect was found (p < 0.05) for PC1 in both the abdominals and the back extensors with post hoc analysis of the muscle main effects found in Table 5.6.





**Figure 5.4:** Principal components (features) for (a) PC1, (d) PC2. PC1 explains 90.3% of the waveform variance with PC2 explaining 6.8%. Ensemble average waveforms for the 5 highest (blue) and 5 lowest (red) scores for PC's 1 and 2 (b and e respectively) are shown to aid with interpretation. PC 2 scores for back muscle interactions are graphed in c PITP is red, PITN is blue.

**Table 5.6:** Muscle main effects for abdominal and back muscle PC scores. PC1 abdominals and back extensors and PC2 abdominals. Muscles with the same letter are not significantly different from each other (p > 0.05).

Abdominals			Back ex	Back extensors	
	PC1	PC2		PC1	
RLRA	def	bcd	RL13	a	
LLRA	def	bcd	LL13	ab	
RURA	f	abc	RL16	abc	
LURA	ef	bcd	LL16	bcd	
REO1	cde	abc	RL33	abc	
LEO1	cdef	cde	LL33	abcd	
REO2	bcd	bcd	RL36	cd	
LEO2	bc	cde	LL36	d	
REO3	cdef	de	RL48	d	
LEO3	bcd	ab	LL48	d	
RIO	a	a	RL52	a	
LIO	ab	e	LL52	ab	

PC2 captured the muscle response to the changing lateral flexion moment (Figure 5.4(d)). A positive score depicts a pattern of high activity initially and then a gradual decrease in activity as the lateral flexion moment moves from right to left as illustrated from the high low scores in Figure 5.4(e). A negative PC2 score indicates the opposite pattern. A significant group by muscle interaction was found (p < 0.05) for PC2 in the back extensor muscles (Table 5.7) and post hoc analysis showed that while both groups had more superior back sites with a greater response to the lateral flexion moment the PITP group had slightly more differences (Table 5.7). Table 5.7 also shows us that for the PITN group the only within group difference (aside from left/right differences which are expected) was LL16 > LL52, whereas for the PITP group it was LL16, LL13 > LL48, LL52. A significant muscle main effect was also found (p < 0.05) for PC2 in the abdominals and post hoc analysis of the muscle main effect can be found in Table 5.6.

**Table 5.7:** Post hoc results of the group by muscle interactions for PC2 in the back. Muscles with the same letter are not significantly different from each other (p > 0.05).

Back extensors				
	PC2 PITP	PC2 PITN		
RL13	ef	ef		
LL13	ab	ab		
RL16	ef	f		
LL16	ab	a		
RL33	def	def		
LL33	abc	abc		
RL36	def	ef		
LL36	abc	abc		
RL48	de	def		
LL48	c	abc		
RL52	d	def		
LL52	c	bc		

# 5.3.3 Strength

Strength variables for each group, measured as the torque produced during a MVIC are found in Table 5.8. No significant differences were found (p > 0.05) between groups for either the flexion or extension torques, irrespective of if they were normalized to body weight or not. There was also no significant difference found (p > 0.05) between groups for the flexion to extension torque ratio.

**Table 5.8:** Strength variables, no significant differences (p > 0.05).

Variable	PITP (n = 23)	PITN (n = 9)	P-value
Flexion Torque (Nm)	174.1 (48.5)	153.4 (44.1)	0.276
Extension Torque (Nm)	235.6 (87.4)	202.0 (93.6)	0.346
Flexion Torque normalized to BW (Nm/kg)	1.9 (0.5)	1.7(0.4)	0.503
Extension Torque normalized to BW (Nm/kg)	2.5 (0.8)	2.1 (0.9)	0.262
Flexion/Extension ratio	0.8(0.3)	0.9 (0.4)	0.283

### 5.4 DISCUSSION

This study showed that minor differences exist in objective physiologically based measures between a group that tested positive on the PIT and one that did not. Since these two groups were well matched based on all of the demographic characteristics, the between group differences cannot be attributed to these factors. Overall, the pain levels

and disability reported from both groups were low (Table 5.1) supporting the recovery classification. Additionally, both groups had similar gender proportions minimizing confounding factors for between group differences due to gender that have been found previously with activation amplitudes during the transfer task (Hubley-Kozey et al., 2012). Finally, the highly controlled task minimized differences in task performance, with both groups demonstrating compliance with the motion constraints. The greatest motion (less than 7°) was in the transverse plane (axial rotation), which would have minimal effect on the magnitude of force acting on the spine in the sagittal and frontal plane. The sole significant difference in motion between the groups was small (1.5°) and in the transverse plane. This motion should have minimal effect on the flexion moment magnitudes as similar anthropometrics and timing characteristics between groups should result in similar moments of force.

The general shape of PC1 indicates moderate activity at the beginning of the task in response to a more lateral moment. As the load is transferred at the midline, muscle activity is at its peak, responding to a more pure flexion moment reflecting an increase in PC1. A drop in activity follows this peak as it moves once again to a more lateral moment on the opposite side. As seen in Figures 4.6(a) and (b), the PC1 scores among muscles correlate highly with the normalized root mean squared amplitude as would be expected with PC1 representing the overall amplitude. There were no differences found between groups in amplitude nor was there a group by muscle interaction. The only significant finding for PC1 was the muscle main effect where the amplitude differences found between muscles for the both the abdominals and the back extensors were similar to previous research (Hubley-Kozey et al., 2014a). These findings showed that the more

medial sites (L13, L33 and L52) had higher amplitudes than the lower and more lateral sites (L36 and L48) (Table 5.6). For the abdominal sites the IO amplitudes were higher than most other abdominal sites (Table 5.6).

The only significant difference for PC2 between the groups was a group by muscle interaction for the back musculature, where the PITN group displayed less variability of the back extensors in temporal features than the PITP group. This difference can be interpreted as greater temporal synchrony or more synergistic co-contraction in the back musculature compared to the PITP group. Both groups displayed similar differences seen as an inferior-superior muscle site difference (Table 5.7) with the PITP group showing slightly more variability. Specifically, the only finding that was different between the groups was that the higher left sided muscles (LL16 and LL13) had a greater response to the lateral flexor moment than the lower sites (LL48 and LL52) in the PITP group. The similar pattern in the PITN group was that left higher more lateral site (LL16) had a greater response to the lateral flexor moment than lowest medial site (LL52). Although, statistically significant differences were found between these two groups caution should be taken when looking at these results since whether the differences are clinically significant is less clear. The temporal differences found between muscles for the abdominals was similar to previous research (Hubley-Kozey et al., 2014a) with some right-left differences for the oblique sites (Table 5.6).

### 5.5 CONCLUSION

In conclusion, minor differences were found in the temporal back extensor activation patterns only during a standardized functional task between a group that tested positive on the PIT and one that tested negative. Upon further analysis of the post hoc

results, the clinical relevance of these findings is not evident. Therefore, it does not seem that the PIT separates a group of people recovered from a LBI such that each subgroup has distinct trunk muscle activation patterns that could impact clinical decision making.

#### **CHAPTER 6 SUMMARY AND CONCLUSIONS.**

#### **6.1 SUMMARY**

The following is a list of the specific objectives of this thesis and the findings related to them:

- To determine whether there are differences in trunk muscle activation patterns during a standardized functional task in those deemed recovered from a LBI when they are classified as having a clinical instability versus those that do not.
  - Significant differences were found between groups in both amplitude and temporal patterns. The CIG had decreased variability in the back extensors in terms of amplitude (PC1) and in the abdominals in terms of temporal features (PC2). These findings were expressed in the group by muscle interactions.
- To determine if there are differences in the isometric torque of trunk flexion and extension between those with clinical instability and those without.
  - Significant differences were identified between groups for strength
    normalized to body weight but not for absolute strength with greater strength
    in both the trunk flexors and extensors in the CIG.
- To determine if there is a difference in the ratio of abdominal to back isometric torque between those with clinical instability and those without.
  - As stated above, there were significant differences in strength in both the trunk flexors and extensors. With this difference being more or less proportional, there was no difference found in the flexion to extension ratio.

- To determine if there is a difference in EMG patterns between those deemed recovered from a LBI with a positive prone instability test (PIT) and those with a negative PIT.
  - A significant difference was found between groups in temporal EMG patterns.
     The PITP group had increased variability of back extensors in the temporal features (PC2).

The premise of this thesis was driven by the need for clinical predictors, and the recent emergence of objective biomechanical predictors for recurrence of LBI in a recovered population. Therefore, it was dedicated to exploring the relationship between clinical measures and objective measures of neuromuscular function (trunk muscle activation patterns and muscle strength). This study found that groups classified based on a battery of clinical tests for spinal stability had differences in both amplitude and temporal objective EMG measures during a standardized functional task, but those classified using a single test had only minor differences. Additionally, while strength differences were found between groups separated by the CPR, their explanation proved to be problematic due to confounding factors.

A previous study suggested that amplitude differences found between a LBI and an asymptomatic population could be explained by strength differences (Hubley-Kozey et al., 2014a), hence an objective measure of trunk flexion and extension were included in the present study to help with interpretation of the findings. The first study (Chapter 4) hypothesized that the CIG would have lower back extensor strength than the NCIG and consequently a higher flexion to extension strength ratio but this was not supported. In fact the CIG had significantly higher strength values for both flexion and extension. Since

the differences for both flexion and extension were proportional, no difference was found in the flexion to extension ratio between groups. The CIG also had a higher occupational activity level, meaning that their job demands were significantly heavier than the NCIG, which could possibly explains their higher strength readings. Higher occupational activity levels would also mean that the CIG experienced increased loading of the spine, which has been associated with decreased disc height, vertebral joint space narrowing and an increase in the anterior/posterior shear forces experienced by the spine (Marras & Granata, 1997; Shan et al., 2012; Suri et al., 2014). All these factors could lead to the decrease in passive stiffness that was detected by the CRP.

Concerning the main objective, distinct differences were found between the two groups with respect to comprehensive amplitude and temporal EMG patterns. When examining the amplitude (PC1) main effects no significant difference was found for the abdominals, although one might expect lower amplitudes (if active stiffness was consistent) based on the greater strength measures in the CIG. Active stiffness was not measured but motion parameters were also similar between groups suggesting that the CIG had greater active stiffness due to the abdominals compared to the NCIG. The group by muscle interaction found for PC1 in the back extensors demonstrated less variability in terms of amplitude in the CIG. While we would expect to see lower activation amplitude due to the CIG's increased strength, this was not a systematic difference suggesting that the difference is not due to strength alone. The interaction found in the temporal features (PC2) of the abdominals also points to less variability or more temporal synchrony (co-contraction) in the muscles of the CIG. All of these findings indicate a strategy by the CIG to increase their active stiffness in an attempt to compensate for the

passive instability (Brown & Potvin, 2005; Brown & McGill, 2008; Hodges et al., 2009; Moreside et al., 2007; Stokes & Gardner-Morse, 2003).

The theoretical foundation for the second study (Chapter 5) was that similar findings in the objective measures would be detected with the PIT as with the classification of clinical instability. This theory was not supported with the only significant finding being a group by muscle interaction in the temporal features (PC2) in the back extensors. Even with this finding of significance, the clinical relevance of it is questionable as the PITP group had only slightly more variability between muscles than the PITN group. Additionally, an increase in variability in the PITP group is opposite to what we found in the CIG (where they had a decrease in variability between muscles), which further decreases the support for this theory. Due to these finding we can state that the PIT is not sufficient by itself to dichotomize a group in terms of objective physiological or biomechanically based measures.

Speculative reasons for the difference between the two classification strategies are based on the nature of CPR's. As discovered by Hicks et al., combining multiple items increases the predictive or discriminatory abilities of the tests over if they were used in isolation(Hicks et al., 2005). Multiple tests provide another level of discrimination that is not present when relying on only one test to be selective between groups. It allows for a more robust definition of the subdivided groups thereby creating a more homogenous group with a greater likelihood to share similar characteristics.

#### 6.2 LIMITATIONS

One potential limitation when using surface EMG recordings is the potential for crosstalk, but to mitigate this, care was taken to follow published protocols in electrode

placement, validation and processing (Butler et al., 2009; Butler et al., 2010). Concerns have also been expressed with obtaining true maximal activations during the normalization procedure. These concerns were minimized in this study not only because all participants were deemed recovered and reported minimal pain (VAS < 20mm) at the time of testing, but also because standard verbal encouragement was used which has been shown to aid reliability (Ng et al., 2003).

It remains unclear from this study whether there is a strength difference between a group with clinical instability and one without due to the differences found in the occupational activity level between groups. Another study matching groups for this metric would be required to answer this question.

There is a potential for a type 1 error using an alpha of 0.1 for the interactions, however the small sample size and the correction for multiple comparisons minimizes this error. On the other hand the small sample size overall results in low statistical power and the potential of a type 2 error for other tests including muscle strength.

There are potential limitations in generalizing these results to a non-military population as the population used in this study was exclusively from the Canadian Armed Forces. There is the potential for differences related to fitness and job demands, but whether other differences would be found for a civilian population with similar job demands, demographics and fitness levels would need further exploration. In any event, generalizing the results of this study is limited to the military population.

Finally, because the testing sessions occurred on separate days, there is the possibility that further recovery could have taken place and therefore change the status of the participants in the different groups. Both because the intersession time was

approximately one week, and there was no significant difference between groups, we believe that the risk associated with this is fairly low.

#### 6.3 IMPLICATIONS

This is the first study that used EMG or an objective biomechanically based measure to test the ability of a CPR to separate a group recovered from a LBI based on clinical instability. The explanation of the specific neuromuscular pattern differences found in this study agrees with the models of spinal stability that utilize the three-subsystem paradigm. Since strength differences could not explain all the amplitude and temporal differences, it suggests that muscle strengthening should not be the sole method of treatment in this group. Another area that treatment could address is the pattern differences although whether these differences are a positive protective mechanism in an unstable spine or a negative influence that could set the person up for recurrence or a future injury remains to be seen.

These results also show that the PIT does not capture salient features that are adequate to separate a group based on similar characteristics.

#### 6.4 FUTURE RESEARCH

This study identified that there are differences in EMG between two groups separated by clinical instability. There are two future paths for this work; the first involves determining the predictive capability of these two tests. This could be in the form of a follow-up study to determine re-injury status of the participants to establish if the clinical tests used or the objective measures have predictive validity for assessing risk of LBI recurrence. The second possible path for future research is to compare these results to a control or asymptomatic group matched for demographic variables, which

would allow for a comparison between a recovered LBI population and an asymptomatic population.

Whether the differences found in this study represent a positive or negative protective mechanism was outside the scope of this study and a follow-up study as mentioned above could aid in determining this. If predictive ability can be shown in the clinical tests, objective measures or some combination of the two, in addition to determining the function of the differences found, then it could aid in the creation and direction of treatments to not only increase their efficacy but also to possibly stop the transition of recurrent LBP into chronic.

The sub-objective explored the neuromuscular differences in a group separated by the PIT but did not compare those results to the ones found in Chapter 4. This could be an avenue for a future study to confirm that the two methods of separating groups are not the same.

#### 6.5 CONCLUSION

In conclusion, the findings of this study support the main hypothesis that those with clinical instability based on a modified Hicks CPR did have altered trunk neuromuscular patterns compared to those with no instability while performing a highly controlled function task including; an increased antagonist/agonist co-activation during the transfer task compared to those without instability and more sustained activity i.e. less response to changing external moments (flexion and lateral flexion). The results did not support the hypothesis that those with clinical instability had decreased back muscle strength as the opposite was found, nor did they have an increased abdominal to back strength ratio. Finally the study does not support the hypothesis that those with a positive

PIT, which is one component of the protocol used to define clinical instability, will have different neuromuscular patterns compared to those that are negative.

This was the first study to examine how objective physiological and biomechanical based measures relate to a clinical test battery and an individual test for spinal instability. This adds to our previous knowledge that trunk amplitude and temporal patterns are different between subgroups of those recovered from a low back injury categorized by a battery of tests and confirms that all differences could not be explained by trunk muscle strength only.

#### REFERENCES

- Alkner, Tesch, & Berg. (2000). Quadriceps EMG/force relationship in knee extension and leg press. *Medicine and Science in Sports and Exercise*, 32(2), 459-463.
- Alston, Carlson, Feldman, Grimm, & Gerontinos. (1966). A quantitative study of muscle factors in the chronic low back syndrome. *Journal of the American Geriatrics Society*, 14(10), 1041-1047.
- Bayramoglu, Akman, Kilinc, Cetin, Yavuz, & Ozker. (2001). Isokinetic measurement of trunk muscle strength in women with chronic low-back pain. *American Journal of Physical Medicine & Rehabilitation / Association of Academic Physiatrists*, 80(9), 650-655.
- Bergmark. (1989). Stability of the lumbar spine. A study in mechanical engineering. *Acta Orthopaedica Scandinavica Supplementum*, 230, 1-54.
- Biely, Silfies, Smith, & Hicks. (2014). Clinical observation of standing trunk movements: What do the aberrant movement patterns tell us? *The Journal of Orthopaedic and Sports Physical Therapy*, 44(4), 262-272. doi:10.2519/jospt.2014.4988; 10.2519/jospt.2014.4988
- Brandon, Graham, Almosnino, Sadler, Stevenson, & Deluzio. (2013). Interpreting principal components in biomechanics: Representative extremes and single component reconstruction. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 23(6), 1304-1310. doi:10.1016/j.jelekin.2013.09.010; 10.1016/j.jelekin.2013.09.010
- Briner, & Benjamin. (1999). Volleyball injuries: Managing acute and overuse disorders. *The Physician and Sportsmedicine*, 27(3), 48-60. doi:10.3810/psm.1999.03.720; 10.3810/psm.1999.03.720
- Brown, & McGill. (2008). How the inherent stiffness of the in vivo human trunk varies with changing magnitudes of muscular activation. *Clinical Biomechanics (Bristol, Avon)*, 23(1), 15-22. doi:10.1016/j.clinbiomech.2007.08.011
- Brown, & Potvin. (2005). Constraining spine stability levels in an optimization model leads to the prediction of trunk muscle cocontraction and improved spine compression force estimates. *Journal of Biomechanics*, 38(4), 745-754. doi:10.1016/j.jbiomech.2004.05.011
- Brown, Vera-Garcia, & McGill. (2006). Effects of abdominal muscle coactivation on the externally preloaded trunk: Variations in motor control and its effect on spine stability. *Spine*, 31(13), E387-93. doi:10.1097/01.brs.0000220221.57213.25
- Burden. (2010). How should we normalize electromyograms obtained from healthy participants? what we have learned from over 25 years of research. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 20(6), 1023-1035. doi:10.1016/j.jelekin.2010.07.004

- Butler, Hubley-Kozey, & Kozey. (2009). Electromyographic assessment of trunk muscle activation amplitudes during a simulated lifting task using pattern recognition techniques. Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology, 19(6), e505-12.
- Butler, Hubley-Kozey, & Kozey. (2010). Characterisation of trunk muscle activation amplitude patterns during a simulated checkstand operation with continuously changing flexor and lateral moment demands. *Ergonomics*, 53(5), 685-695. doi:10.1080/00140131003671991; 10.1080/00140131003671991
- Butler, Hubley-Kozey, & Kozey. (2012). Changes in electromyographic activity of trunk muscles within the sub-acute phase for individuals deemed recovered from a low back injury. Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology, doi:10.1016/j.jelekin.2012.10.012; 10.1016/j.jelekin.2012.10.012
- Butler, Newell, Hubley-Kozey, & Kozey. (2009). The interpretation of abdominal wall muscle recruitment strategies change when the electrocardiogram (ECG) is removed from the electromyogram (EMG). *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology, 19*(2), e102-13. doi:10.1016/j.jelekin.2007.10.004
- Callaghan, Salewytsch, & Andrews. (2001). An evaluation of predictive methods for estimating cumulative spinal loading. *Ergonomics*, 44(9), 825-837. doi:10.1080/00140130118541
- Cedraschi, Nordin, Nachemson, & Vischer. (1998). Health care providers should use a common language in relation to low back pain patients. *Bailliere's Clinical Rheumatology*, 12(1), 1-15.
- Childs, Fritz, Flynn, Irrgang, Johnson, Majkowski, & Delitto. (2004). A clinical prediction rule to identify patients with low back pain most likely to benefit from spinal manipulation: A validation study. *Annals of Internal Medicine*, 141(12), 920-928.
- Cholewicki, & McGill. (1996). Mechanical stability of the in vivo lumbar spine: Implications for injury and chronic low back pain. *Clinical Biomechanics (Bristol, Avon)*, 11(1), 1-15.
- Cholewicki, McGill, & Norman. (1995). Comparison of muscle forces and joint load from an optimization and EMG assisted lumbar spine model: Towards development of a hybrid approach. *Journal of Biomechanics*, 28(3), 321-331.
- Cholewicki, Panjabi, & Khachatryan. (1997). Stabilizing function of trunk flexor-extensor muscles around a neutral spine posture. *Spine*, 22(19), 2207-2212.
- Cholewicki, Silfies, Shah, Greene, Reeves, Alvi, & Goldberg. (2005). Delayed trunk muscle reflex responses increase the risk of low back injuries. *Spine*, 30(23), 2614-2620.
- Cholewicki, & VanVliet. (2002). Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. Clinical Biomechanics (Bristol, Avon), 17(2), 99-105.

- da Silva, Arsenault, Gravel, Lariviere, & de Oliveira. (2005). Back muscle strength and fatigue in healthy and chronic low back pain subjects: A comparative study of 3 assessment protocols. *Archives of Physical Medicine and Rehabilitation*, 86(4), 722-729. doi:10.1016/j.apmr.2004.08.007
- Dagenais, Caro, & Haldeman. (2008). A systematic review of low back pain cost of illness studies in the united states and internationally. *The Spine Journal: Official Journal of the North American Spine Society*, 8(1), 8-20. doi:10.1016/j.spinee.2007.10.005; 10.1016/j.spinee.2007.10.005
- Davarian, Maroufi, Ebrahimi, Farahmand, & Parnianpour. (2012). Trunk muscles strength and endurance in chronic low back pain patients with and without clinical instability. *Journal of Back and Musculoskeletal Rehabilitation*, 25(2), 123-129. doi:10.3233/BMR-2012-0320; 10.3233/BMR-2012-0320
- Davidson, & Hubley-Kozey. (2005). Trunk muscle responses to demands of an exercise progression to improve dynamic spinal stability. *Archives of Physical Medicine and Rehabilitation*, 86(2), 216-223. doi:10.1016/j.apmr.2004.04.029
- de Vet, Heymans, Dunn, Pope, van der Beek, Macfarlane, . . . Croft. (2002). Episodes of low back pain: A proposal for uniform definitions to be used in research. *Spine*, 27(21), 2409-2416. doi:10.1097/01.BRS.0000030307.34002.BE
- Delitto, George, Van Dillen, Whitman, Sowa, Shekelle, . . . Godges. (2012). Low back pain. *The Journal of Orthopaedic and Sports Physical Therapy*, 42(4), A1-57. doi:10.2519/jospt.2012.0301
- Descarreaux, Blouin, & Teasdale. (2004). Force production parameters in patients with low back pain and healthy control study participants. *Spine*, 29(3), 311-317.
- du Bois, Weycker, Albera, Bradford, Costabel, Kartashov, . . . Wells. (2011). Forced vital capacity in patients with idiopathic pulmonary fibrosis: Test properties and minimal clinically important difference. *American Journal of Respiratory and Critical Care Medicine*, 184(12), 1382-1389. doi:10.1164/rccm.201105-0840OC; 10.1164/rccm.201105-0840OC
- Flynn, Fritz, Whitman, Wainner, Magel, Rendeiro, . . . Allison. (2002). A clinical prediction rule for classifying patients with low back pain who demonstrate short-term improvement with spinal manipulation. *Spine*, 27(24), 2835-2843. doi:10.1097/01.BRS.0000035681.33747.8D
- Fritz, Brennan, Clifford, Hunter, & Thackeray. (2006). An examination of the reliability of a classification algorithm for subgrouping patients with low back pain. *Spine*, 31(1), 77-82.
- Fritz, & George. (2000). The use of a classification approach to identify subgroups of patients with acute low back pain. interrater reliability and short-term treatment outcomes. *Spine*, 25(1), 106-114.
- Geisser, Ranavaya, Haig, Roth, Zucker, Ambroz, & Caruso. (2005). A meta-analytic review of surface electromyography among persons with low back pain and normal, healthy controls.

- The Journal of Pain: Official Journal of the American Pain Society, 6(11), 711-726. doi:10.1016/j.jpain.2005.06.008
- Granata, & Orishimo. (2001). Response of trunk muscle coactivation to changes in spinal stability. *Journal of Biomechanics*, 34(9), 1117-1123.
- Granata, & Wilson. (2001). Trunk posture and spinal stability. *Clinical Biomechanics (Bristol, Avon)*, 16(8), 650-659.
- Gross, & Battie. (2004). The prognostic value of functional capacity evaluation in patients with chronic low back pain: Part 2: Sustained recovery. *Spine*, 29(8), 920-924.
- Gross, Battie, & Cassidy. (2004). The prognostic value of functional capacity evaluation in patients with chronic low back pain: Part 1: Timely return to work. *Spine*, 29(8), 914-919.
- Gruther, Wick, Paul, Leitner, Posch, Matzner, . . . Ebenbichler. (2009). Diagnostic accuracy and reliability of muscle strength and endurance measurements in patients with chronic low back pain. *Journal of Rehabilitation Medicine : Official Journal of the UEMS European Board of Physical and Rehabilitation Medicine*, 41(8), 613-619. doi:10.2340/16501977-0391; 10.2340/16501977-0391
- Hestback, Leboeuf-Yde, & Manniche. (2003). Low back pain: What is the long-term course? A review of studies of general patient populations. European Spine Journal: Official Publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 12(2), 149-165. doi:10.1007/s00586-002-0508-5
- Heydari, Nargol, Jones, Humphrey, & Greenough. (2010). EMG analysis of lumbar paraspinal muscles as a predictor of the risk of low-back pain. European Spine Journal: Official Publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society, 19(7), 1145-1152. doi:10.1007/s00586-010-1277-1
- Hicks, Fritz, Delitto, & McGill. (2005). Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. Archives of Physical Medicine and Rehabilitation, 86(9), 1753-1762. doi:10.1016/j.apmr.2005.03.033
- Hicks, Fritz, Delitto, & Mishock. (2003). Interrater reliability of clinical examination measures for identification of lumbar segmental instability. *Archives of Physical Medicine and Rehabilitation*, 84(12), 1858-1864.
- Hodges, Kaigle Holm, Holm, Ekstrom, Cresswell, Hansson, . . . Hodges, P. (2003). Intervertebral stiffness of the spine is increased by evoked contraction of transversus abdominis and the diaphragm: In vivo porcine studies. *Spine*, 28(23), 2594-2601. doi:10.1097/01.BRS.0000096676.14323.25

- Hodges, & Richardson. (1996). Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. *Spine*, 21(22), 2640-2650.
- Hodges, van den Hoorn, Dawson, & Cholewicki. (2009). Changes in the mechanical properties of the trunk in low back pain may be associated with recurrence. *Journal of Biomechanics*, 42(1), 61-66. doi:10.1016/j.jbiomech.2008.10.001; 10.1016/j.jbiomech.2008.10.001
- Holmstrom, Moritz, & Andersson. (1992a). Trunk muscle strength and back muscle endurance in construction workers with and without low back disorders. *Scandinavian Journal of Rehabilitation Medicine*, 24(1), 3-10.
- Holmstrom, Moritz, & Andersson. (1992b). Trunk muscle strength and back muscle endurance in construction workers with and without low back disorders. *Scandinavian Journal of Rehabilitation Medicine*, 24(1), 3-10.
- Hubley-Kozey, Butler, & Kozey. (2012). Activation amplitude and temporal synchrony among back extensor and abdominal muscles during a controlled transfer task: Comparison of men and women. *Human Movement Science*, 31(4), 863-879.
- Hubley-Kozey, Hanada, Gordon, Kozey, & McKeon. (2009). Differences in abdominal muscle activation patterns of younger and older adults performing an asymmetric leg-loading task. *PM & R: The Journal of Injury, Function, and Rehabilitation*, *1*(11), 1004-1013. doi:10.1016/j.pmrj.2009.09.018; 10.1016/j.pmrj.2009.09.018
- Hubley-Kozey, Moreside, & Quirk. (2013.). Trunk neuromuscular pattern alterations during a controlled functional task in a low back injured group deemed ready to resume regular activities. *Work, Epub ahead of print*
- Hubley-Kozey, Moreside, & Quirk. (2014a). Trunk neuromuscular pattern alterations during a controlled functional task in a low back injured group deemed ready to resume regular activities. *Work (Reading, Mass.)*, 47(1), 87-100. doi:10.3233/WOR-131689; 10.3233/WOR-131689
- Hubley-Kozey, Moreside, & Quirk. (2014b). Trunk neuromuscular pattern alterations during a controlled functional task in a low back injured group deemed ready to resume regular activities. *Work (Reading, Mass.)*, 47(1), 87-100. doi:10.3233/WOR-131689; 10.3233/WOR-131689
- Hubley-Kozey, & Vezina. (2002). Differentiating temporal electromyographic waveforms between those with chronic low back pain and healthy controls. *Clinical Biomechanics* (*Bristol*, *Avon*), 17(9-10), 621-629.
- Innes, & Straker. (1999a). Reliability of work-related assessments. Work (Reading, Mass.), 13(2), 107-124.
- Innes, & Straker. (1999b). Validity of work-related assessments. Work (Reading, Mass.), 13(2), 125-152.

- Iwai, Nakazato, Irie, Fujimoto, & Nakajima. (2004). Trunk muscle strength and disability level of low back pain in collegiate wrestlers. Medicine and Science in Sports and Exercise, 36(8), 1296-1300.
- Jackson. (2003). A user's guide to principal components. Hoboken, N.J.: Wiley-Interscience.
- Jensen, Chen, & Brugger. (2003). Interpretation of visual analog scale ratings and change scores: A reanalysis of two clinical trials of postoperative pain. *The Journal of Pain: Official Journal of the American Pain Society*, 4(7), 407-414.
- Jensen, Strom, Turner, & Romano. (1992). Validity of the sickness impact profile roland scale as a measure of dysfunction in chronic pain patients. *Pain*, 50(2), 157-162.
- King, Tuckwell, & Barrett. (1998). A critical review of functional capacity evaluations. *Physical Therapy*, 78(8), 852-866.
- Kirkaldy-Willis. (1985). Presidential symposium on instability of the lumbar spine. <br/> <br/> />Introduction. spine. Spine, 10, 254.
- Kotilainen, & Valtonen. (1993). Clinical instability of the lumbar spine after microdiscectomy. *Acta Neurochirurgica*, 125(1-4), 120-126.
- Kraus, Schaffer, McArthur, & Peek-Asa. (1997). Epidemiology of acute low back injury in employees of a large home improvement retail company. *American Journal of Epidemiology*, 146(8), 637-645.
- Kumar. (1990). Cumulative load as a risk factor for back pain. Spine, 15(12), 1311-1316.
- Landry, McKean, Hubley-Kozey, Stanish, & Deluzio. (2007). Knee biomechanics of moderate OA patients measured during gait at a self-selected and fast walking speed. *Journal of Biomechanics*, 40(8), 1754-1761. doi:10.1016/j.jbiomech.2006.08.010
- Laupacis, Sekar, & Stiell. (1997). Clinical prediction rules. A review and suggested modifications of methodological standards. JAMA: The Journal of the American Medical Association, 277(6), 488-494.
- Lawrence, & Luca. (1983). Myoelectric signal versus force relationship in different human muscles. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology*, 54(6), 1653-1659.
- Lee, Hobden, Stiell, & Wells. (2003). Clinically important change in the visual analog scale after adequate pain control. *Academic Emergency Medicine: Official Journal of the Society for Academic Emergency Medicine*, 10(10), 1128-1130.
- Lee, Miara, Arnold, Biewener, & Wakeling. (2011). EMG analysis tuned for determining the timing and level of activation in different motor units. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 21(4), 557-565. doi:10.1016/j.jelekin.2011.04.003; 10.1016/j.jelekin.2011.04.003

- Lusted. (1993). Predicting return to work after rehabilitation for low back injury. *The Australian Journal of Physiotherapy*, 39(3), 203-210. doi:10.1016/S0004-9514(14)60483-9; 10.1016/S0004-9514(14)60483-9
- Macdonald, Dawson, & Hodges. (2011). Behavior of the lumbar multifidus during lower extremity movements in people with recurrent low back pain during symptom remission. *The Journal of Orthopaedic and Sports Physical Therapy*, 41(3), 155-164. doi:10.2519/jospt.2011.3410; 10.2519/jospt.2011.3410
- MacDonald, Moseley, & Hodges. (2009). Why do some patients keep hurting their back? evidence of ongoing back muscle dysfunction during remission from recurrent back pain. *Pain*, 142(3), 183-188. doi:10.1016/j.pain.2008.12.002; 10.1016/j.pain.2008.12.002
- MacDonald, Moseley, & Hodges. (2010). People with recurrent low back pain respond differently to trunk loading despite remission from symptoms. *Spine*, 35(7), 818-824. doi:10.1097/BRS.0b013e3181bc98f1; 10.1097/BRS.0b013e3181bc98f1
- Maetzel, & Li. (2002). The economic burden of low back pain: A review of studies published between 1996 and 2001. *Best Practice & Research.Clinical Rheumatology*, 16(1), 23-30. doi:10.1053/berh.2001.0204
- Magee. (1997). Orthopedic physical assessment. Philadelphia: W.B. Saunders.
- Mannion, Taimela, Muntener, & Dvorak. (2001). Active therapy for chronic low back pain part 1. effects on back muscle activation, fatigability, and strength. *Spine*, 26(8), 897-908.
- Manusov. (2012). Evaluation and diagnosis of low back pain. *Primary Care*, *39*(3), 471-479. doi:10.1016/j.pop.2012.06.003; 10.1016/j.pop.2012.06.003
- Marras, & Granata. (1997). Changes in trunk dynamics and spine loading during repeated trunk exertions. *Spine*, 22(21), 2564-2570.
- Matheson. (1982). Work capacity evaluation: A training manual for occupational therapist. Trabuco Canyon, CA.: Rehabilitation Institute of Southern California.
- Matheson, Isernhagen, & Hart. (2002). Relationships among lifting ability, grip force, and return to work. *Physical Therapy*, 82(3), 249-256.
- McGill. (1997). The biomechanics of low back injury: Implications on current practice in industry and the clinic. *Journal of Biomechanics*, 30(5), 465-475.
- McGill, Childs, & Liebenson. (1999). Endurance times for low back stabilization exercises: Clinical targets for testing and training from a normal database. *Archives of Physical Medicine and Rehabilitation*, 80(8), 941-944.
- McGill, & Cholewicki. (2001). Biomechanical basis for stability: An explanation to enhance clinical utility. *The Journal of Orthopaedic and Sports Physical Therapy*, 31(2), 96-100.
- Mirolla. (2004). The cost of chronic disease in canada. ().

- Moreside, Quirk, & Hubley-Kozey. (2014). Temporal patterns of the trunk muscles remain altered in a low back-injured population despite subjective reports of recovery. *Archives of Physical Medicine and Rehabilitation*, 95(4), 686-698. doi:10.1016/j.apmr.2013.10.003; 10.1016/j.apmr.2013.10.003
- Moreside, Vera-Garcia, & McGill. (2007). Trunk muscle activation patterns, lumbar compressive forces, and spine stability when using the bodyblade. *Physical Therapy*, 87(2), 153-163. doi:10.2522/ptj.20060019
- National Collaborating Centre for Primary Care (UK). (2009). Low back pain Early management of persistent non-specific low back pain. Retrieved 08/19, 2014, from <a href="http://www.ncbi.nlm.nih.gov/books/NBK11702/">http://www.ncbi.nlm.nih.gov/books/NBK11702/</a>
- Ng, Parnianpour, Kippers, & Richardson. (2003). Reliability of electromyographic and torque measures during isometric axial rotation exertions of the trunk. Clinical Neurophysiology: Official Journal of the International Federation of Clinical Neurophysiology, 114(12), 2355-2361.
- O'Sullivan. (2000). Lumbar segmental 'instability': Clinical presentation and specific stabilizing exercise management. *Manual Therapy*, 5(1), 2-12. doi:10.1054/math.1999.0213
- Ozguler, Leclerc, Landre, Pietri-Taleb, & Niedhammer. (2000). Individual and occupational determinants of low back pain according to various definitions of low back pain. *Journal of Epidemiology and Community Health*, 54(3), 215-220.
- Panjabi. (1992a). The stabilizing system of the spine. part I. function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders*, 5(4), 383-9; discussion 397.
- Panjabi. (1992b). The stabilizing system of the spine. part II. neutral zone and instability hypothesis. *Journal of Spinal Disorders*, 5(4), 390-6; discussion 397.
- Panjabi. (2003). Clinical spinal instability and low back pain. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 13(4), 371-379.
- Pengel, Herbert, Maher, & Refshauge. (2003). Acute low back pain: Systematic review of its prognosis. *BMJ (Clinical Research Ed.)*, 327(7410), 323. doi:10.1136/bmj.327.7410.323
- Pitkanen, Manninen, Lindgrer, Turunen, & Airaksinen. (1997). Limited usefulness of traction-compression films in the radiographic diagnosis of lumbar spinal instability. comparison with flexion-extension films. *Spine*, 22(2), 193-197.
- Pope, & Panjabi. (1985). Biomechanical definitions of spinal instability. Spine, 10(3), 255-256.
- Rabin, Shashua, Pizem, Dickstein, & Dar. (2014). A clinical prediction rule to identify patients with low back pain who are likely to experience short-term success following lumbar stabilization exercises: A randomized controlled validation study. *The Journal of Orthopaedic and Sports Physical Therapy*, 44(1), 6-B13. doi:10.2519/jospt.2014.4888; 10.2519/jospt.2014.4888

- Radebold, Cholewicki, Polzhofer, & Greene. (2001). Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine*, 26(7), 724-730.
- Reme, Hagen, & Eriksen. (2009). Expectations, perceptions, and physiotherapy predict prolonged sick leave in subacute low back pain. *BMC Musculoskeletal Disorders*, 10, 139-2474-10-139. doi:10.1186/1471-2474-10-139; 10.1186/1471-2474-10-139
- Resnik, & Dobrzykowski. (2003). Guide to outcomes measurement for patients with low back pain syndromes. *The Journal of Orthopaedic and Sports Physical Therapy*, 33(6), 307-16; discussion 317-8.
- Robertson. (2004). Research methods in biomechanics. Champaign, IL: Human Kinetics.
- Roland, & Fairbank. (2000). The roland-morris disability questionnaire and the oswestry disability questionnaire. *Spine*, 25(24), 3115-3124.
- Roland, & Morris. (1983). A study of the natural history of low-back pain. part II: Development of guidelines for trials of treatment in primary care. *Spine*, 8(2), 145-150.
- Rosenbaum. (1991). Human motor control. San Diego: Academic Press.
- Schibye, Hansen, Sogaard, & Christensen. (2001). Aerobic power and muscle strength among young and elderly workers with and without physically demanding work tasks. *Applied Ergonomics*, 32(5), 425-431.
- Schmidt. (1982). *Motor control and learning: A behavioral emphasis*. Champaign, Ill.: Human Kinetics Publishers.
- Schopflocher, Taenzer, & Jovey. (2011). The prevalence of chronic pain in canada. *Pain Research & Management: The Journal of the Canadian Pain Society = Journal De La Societe Canadienne Pour Le Traitement De La Douleur*, 16(6), 445-450.
- Shan, Zhang, Chen, & Wei. (2012). Flexion relaxation of erector spinae response to spinal shrinkage. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 22(3), 370-375. doi:10.1016/j.jelekin.2011.12.011; 10.1016/j.jelekin.2011.12.011
- Shirado, Kaneda, & Ito. (1992). Trunk-muscle strength during concentric and eccentric contraction: A comparison between healthy subjects and patients with chronic low-back pain. *Journal of Spinal Disorders*, 5(2), 175-182.
- Sjolie, & Ljunggren. (2001). The significance of high lumbar mobility and low lumbar strength for current and future low back pain in adolescents. *Spine*, 26(23), 2629-2636.
- Solomonow. (2009). Ligaments: A source of musculoskeletal disorders. *Journal of Bodywork and Movement Therapies*, 13(2), 136-154. doi:10.1016/j.jbmt.2008.02.001; 10.1016/j.jbmt.2008.02.001

- Spitzer, LeBlanc, & Dupuis. (1987). Scientific approach to the assessment and management of activity-related spinal disorders. A monograph for clinicians. report of the quebec task force on spinal disorders. Spine, 12(7 Suppl), S1-59.
- Stanton, Henschke, Maher, Refshauge, Latimer, & McAuley. (2008). After an episode of acute low back pain, recurrence is unpredictable and not as common as previously thought. *Spine*, 33(26), 2923-2928. doi:10.1097/BRS.0b013e31818a3167
- Stanton, Latimer, Maher, & Hancock. (2009). Definitions of recurrence of an episode of low back pain: A systematic review. *Spine*, 34(9), E316-22. doi:10.1097/BRS.0b013e318198d073
- Stokes, & Gardner-Morse. (2003). Spinal stiffness increases with axial load: Another stabilizing consequence of muscle action. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 13(4), 397-402.
- Stratford, Binkley, Riddle, & Guyatt. (1998). Sensitivity to change of the roland-morris back pain questionnaire: Part 1. *Physical Therapy*, 78(11), 1186-1196.
- Sullivan, Bishop, & Pivik. (1995). The pain catastrophizing scale: Development and validation. *Psychological Assessment*, 7(4), 524-532. doi:10.1037/1040-3590.7.4.524
- Suri, Hunter, Boyko, Rainville, Guermazi, & Katz. (2014). Physical activity and associations with computed tomography-detected lumbar zygapophyseal joint osteoarthritis. *The Spine Journal: Official Journal of the North American Spine Society*, doi:10.1016/j.spinee.2014.06.022; 10.1016/j.spinee.2014.06.022
- Tammelin, Nayha, Rintamaki, & Zitting. (2002). Occupational physical activity is related to physical fitness in young workers. *Medicine and Science in Sports and Exercise*, 34(1), 158-165.
- Twomey, & Taylor. (2000). Physical therapy of the low back. New York: Churchill Livingstone.
- Vander, Sherman, & Luciano. (1994). *Human physiology: The mechanisms of body function*. New York: McGraw-Hill.
- Vera-Garcia, Brown, Gray, & McGill. (2006). Effects of different levels of torso coactivation on trunk muscular and kinematic responses to posteriorly applied sudden loads. *Clinical Biomechanics (Bristol, Avon)*, 21(5), 443-455. doi:10.1016/j.clinbiomech.2005.12.006
- Vera-Garcia, Moreside, & McGill. (2010). MVC techniques to normalize trunk muscle EMG in healthy women. Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology, 20(1), 10-16. doi:10.1016/j.jelekin.2009.03.010; 10.1016/j.jelekin.2009.03.010
- Vezina, & Hubley-Kozey. (2000). Muscle activation in therapeutic exercises to improve trunk stability. Archives of Physical Medicine and Rehabilitation, 81(10), 1370-1379. doi:10.1053/apmr.2000.16349

- Wakeling. (2009). Patterns of motor recruitment can be determined using surface EMG. *Journal of Electromyography and Kinesiology: Official Journal of the International Society of Electrophysiological Kinesiology*, 19(2), 199-207. doi:10.1016/j.jelekin.2007.09.006
- Wasiak, Young, Dunn, Cote, Gross, Heymans, & von Korff. (2009). Back pain recurrence: An evaluation of existing indicators and direction for future research. *Spine*, 34(9), 970-977. doi:10.1097/BRS.0b013e3181a01b63
- Wasson, Sox, Neff, & Goldman. (1985). Clinical prediction rules. applications and methodological standards. *The New England Journal of Medicine*, 313(13), 793-799. doi:10.1056/NEJM198509263131306
- Waters, Putz-Anderson, & Garg. (1994).

  Applications manual for the revised NIOSH lifting equation. (No. 00224171). ((NIOSH) 94-110)
- White, Johnson, Panjabi, & Southwick. (1975). Biomechanical analysis of clinical stability in the cervical spine. *Clinical Orthopaedics and Related Research*, (109)(109), 85-96.
- White, & Panjabi. (1978). The basic kinematics of the human spine. A review of past and current knowledge. *Spine*, 3(1), 12-20.
- Winter. (2009). Biomechanics and motor control of human movement. Hoboken, N.J.: John Wiley & Sons.
- Woods, & Bigland-Ritchie. (1983). Linear and non-linear surface EMG/force relationships in human muscles. an anatomical/functional argument for the existence of both. *American Journal of Physical Medicine*, 62(6), 287-299.

# APPENDIX 1 COMPARISON OF ARTICLES USING LBP/LBI

An example of articles using LBP or LBI and the definitions that they provided. Articles were found through a PubMed search using Low back pain AND definition, or Low back injury AND definition.

Study	Title	LBP or LBI	Definition.
Include table number and title for table, give an indication as to how these were chosenTaylor et. Al 2014	Incidence and risk factors for first time incident LBP: a systematic review and meta analysis	LBP	No operation al definition of what LBP is.
Lizier et al. 2012	Exercises for treatment of non-specific LBP.	LBP	Defined LBP as pain localized below the margin of the last rib and above the inferior gluteal line with or without limb pain.
Sypert et al. 1986	LBP disorders: lumbar fusion?	LBP	Uses LBP to encompass all disorders of the lumbar spine i.e. mechanical (lumbar instability) pain.
Cedraschi et al. 1998(Cedraschi et al., 1998) (Cedraschi et al., 1998) (Cedraschi et al., 1998) (Cedraschi et al., 1998) (Cedraschi et al., 1998)	Health care providers should use a common language in relation to LBP patients.	LBP	Uses LBP to define any problem in the lumbar spine. Suggests a common nomenclature such as specific LBP, non-specific LBP, or common LBP.
Spitzer et al. 1987	Scientific approach to the assessment and management of activity related spinal disorders, a monograph for clinicians. Report of the Quebec task force on spine disorders.	LBP	LBP defined as pain between the lower ribs and the gluteal folds, subdivides LBP into acute, sub acute and chronic. -States that pain is the primordial, and often only, symptom of the vast majority of spinal disorders.
Maetzel et al. 2002	The economic	LBP/LBI	No definition of LBP. Also

Ozguler et al. 2000	burden of LBP: a review of studies published between 1996 and 2001.  Individual and occupational determinants of LBP according to various definitions of LBP.	LBP	Discusses how LBP is a vague definition and there is a need to clarify it but all suggestions include LBPProposes a classification scheme according to duration and if health care was
Panjabi 2003.	Clinical spinal instability and LBP.	LBP	accessed.  No definition of LBPStates specific causes for LBP are not known.
Kraus et al. 1997	Design factors in epidemiologic cohort studies of work related LBI or pain.	LBI/LBP	Uses LBI and LBP interchangeablyLBI defined as a workers' compensation claim or medical department visitDoesn't distinguish between LBI and LBP.
Lusted et al. 1993	Predicting return to work after rehabilitation for LBI	LBI	Identifies that the terms LBP, back pain, back injury and chronic back pain are frequently used without definitionUses terms interchangeablyDefines LBI as any compensable work-related back problem which was referred for rehab.
Briner et al. 1999	Volleyball injuries, managing acute and overuse disorders.	LBI	No definition of LBI providedUses LBI and LBP interchangeably.
McGill 1997	The biomechanics of LBI: Implication on current practice in industry and the clinic.	LBI	Predominantly uses LBI, seems to use LBP as a part or symptom of LBI but does not separate them.
Dasinger et al. 2001	Doctor proactive communication, return to work recommendation and duration of	LBI	Uses LBI and LBP interchangeably.

	disability after a workers' compensation LBI.		
Peek-Asa et al. 2004	Incidence of acute LBI among older workers in a cohort of material handlers.	LBI	LBI was defined as those that submitted workers compensation claims.
Kraus et al. 1997	Epidemiology of acute LBI in employees of a large home improvement retail company.	LBI	Uses terms interchangeablyAcknowledges that there are multiple terms used to describe it.
Bigos et al. 1980	Back injuries in industry: A retrospective study III Employee-related factors.	LBI	Terms not defined and used interchangeablyTaken from injury claim data.

## **APPENDIX 2**

**Physiotherapy Assessment:**Posture, Neurological and Stability assessment checklist

PARTICIPANT NAME:						
PARTICIPANT ID:						
DATE:						
Physiotherapist:						
SPINE		.1	b		•	
Index of Kyphosis:	(a/b)*100		a			
Lordosis Height (cm):	(c)	'			c	
Check one of the following:						
Ideal posture Kyphosis Lu Sway Back Flat Back Observable Scoliosis- EXCLUDI						
FROM STUDY	Repeated	l Flexio	n			
Articular Assessment:	Repeated I	extension	n			
	Con	pressio	n			
Dorsolumbar						
LSF F RSF	RR					
PELVIS						
Pelvic Tilt Angle:						
Check one of the following (if app	licable):					
Anterior Pelvic Tilt	Posterior Pelvic Tilt	Neutra	l Pelvis			
<b>MYOTOMES</b>						
Examiner matches participant's staparticipant response: L2: Hip flexion (sitting)	rength for 5 seconds. ( RIGHT: 0	Check ap 1	pplicable 2	e box l	pelow for g	grading 5

	LEFT: 0	1	2	3	4	5
L3: Knee extension (sitting)	RIGHT: 0	1	2	3	4	5
	LEFT: 0	1	2	3	4	5
L4: Dorsiflexion (sitting)	RIGHT: 0	1	2	3	4	5
	LEFT: 0	1	2	3	4	5
L5: Big toe extension (sitting)	RIGHT: 0	1	2	3	4	5
	LEFT: 0	1	2	3	4	5
S2: Knee flexion (sitting)	RIGHT: 0	1	2	3	4	5
	LEFT: 0	1	2	3	4	5
S1: Plantar flexion (standing)	RIGHT: 0	1	2	3	4	5
	LEFT: 0	1	2	3	4	5

#### **REFLEXES**

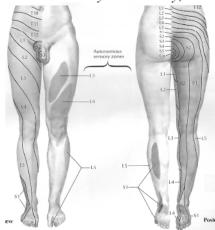
0 = Absent, 1 = Diminished, 2 = Normal, 3 = Hyperactive without clonus, 4 = Hyperactive with clonus.

Test with tendon on stretch. Check applicable box below for grading participant response:

L3/4: Patellar tendon	RIGHT: 0	1	2	3	4
	LEFT: 0	1	2	3	4
S1: Achilles tendon	RIGHT: 0	1	2	3	4
	LEFT: 0	1	2	3	4

#### **DERMATOMES**

Compare side to side. Check box(es) below if any numbness or tingling. Netter FH: Atlas of Human Anatomy. New Jersey, CIBA-GEIGY Corporation, 1989.



RIGHT: L1 L2 L3 L4 L5 S1 S2/S3 (Saddle paresthesia)

LEFT: L1 L2 L3 L4 L5 S1 S2/S3 (Saddle

paresthesia)

**COMMENTS** (if needed):

#### "STABILIZATION" CLASSIFICATION TESTS (FRITZ ET AL, 2007; HICKS ET AL 2005)

	Troit There is in the bring provided by
Test	Result (+ vs - , side or segmental level
	involved, score for FMS movements)
Predictors of clinical success with a spi	inal stabilization exercise program
Aberrant movement during lu-	mbar
Flexion/Extension	
<ol><li>Straight leg raise range of mot</li></ol>	ion
3. Posterior-to-anterior mobility	testing
4. Prone Instability Test	
Additional tests related to spine instabi	ility
5. Posterior Pelvic Pain Provocat	tion Test

6.	ASLR Test:	
•	Neutral	
	TA (anterior) compression	
•	MF (posterior) compression	
7.	Modified Trendelenburg Test	
8.	Passive Lumbar Extension Test (PLE)	
Functio	onal Movements	
9.	Hurdle step	
10.	In-line lunge	
11.	Rotary Stability	
12.	Push-up	

In order to develop our prediction model we will include the general category of the type of work demand that you have as individuals with different demands have different risks of injury.

#### What type of work do you do?

Sedentary: Exerting 10lbs (4.54 Kg) of force occasionally and/or negligible amount of force frequently
Light: Exerting 20lbs (9 Kg) of force occasionally and/or 10lbs frequently and/or negligible amount of
force constantly
Medium: Exerting 50lbs (25Kg) of force occasionally and/or 20lbs frequently and/or up to 10lbs
constantly
Heavy: Exerting 100lbs (45 Kg) of force occasionally and/or 50lbs frequently and/or up to 20lbs
constantly
Very Heavy: Exerting in excess of 100lbs of force occasionally and/or in excess of 50lbs frequently
and/or in excess of 20lbs constantly

The following 13 tests will be performed in the order presented according to the following descriptions.

#### 1. Presence of aberrant movement during sagittal lumbar AROM (Hicks et al., 2005)

- 1.1. Procedure:
  - 1.1.1. The participant starts in a relaxed standing position.
  - 1.1.2. The participant then flexes forward as far as possible to reach towards their toes.
  - 1.1.3. They then return to the standing position.
- 1.2. Observe for any anomalies from a smooth performance. Positive if any of the following or other dysfunction noted:
  - 1.2.1. Instability Catch (sudden twitch or lateral movement with flexion).
  - 1.2.2. *Painful Arc* (repeatable onset of pain only during a specific ROM).
  - 1.2.3. Thigh Climbing or Gower's Sign (uses hand on thighs to return to standing).
  - 1.2.4. *Reversal of lumbo-pelvic rhythm* (ie. lumbar spine extends first upon returning to standing instead of pelvis rotating).

#### 2. Passive Straight Leg Raise (SLR) (Hicks et al, 2005)

- 2.1. Procedure:
  - 2.1.1. The participant is supine.
  - 2.1.2. An inclinometer is placed on tibial crest just below the tibial tubercle.
  - 2.1.3. The examiner raises the leg passively and slowly to the maximum tolerated SLR (not the onset of pain).
  - 2.1.4. The examiner's other hand palpates the ipsilateral pelvis to ensure that no pelvic occurs.
- 2.2. Test is terminated and hip flexion is recorded when:
  - 2.2.1. The participant cannot tolerate any additional movement.
  - 2.2.2. The examiner detects pelvic ROM.
  - 2.2.3. Positive if  $> 91^{\circ}$

#### 3. Posterior-to-anterior mobility testing (Hicks et al., 2005)

- 3.1. Procedure:
  - 3.1.1. The participant is prone.
  - 3.1.2. The examiner uses the hypothenar eminence to apply a posterior-to-anterior pressure to the lumbar spinous process at each level, L1 L5.
- 3.2. The examiner makes a judgment for each of the lumbar segments (L1 L5) of:
  - 3.2.1. Normal, hypomobile, or hypermobile as determined from clinical experience.

#### 4. Prone Instability Test (Hicks et al, 2003):

- 4.1. Procedure:
  - 4.1.1. The participant lies prone with their body on the examining table and legs over the edge with feet resting on the floor.
  - 4.1.2. While the participant rests in this position, the examiner applies posterior-to-anterior pressure to the lumbar spinous processes at each level, L1 L5.
  - 4.1.3. When provocation of pain is reported at a specific level, the examiner makes a note of the painful level.
  - 4.1.4. The participant then lifts the legs off the floor and the examiner applies the posterior-to-anterior pressure at the same level noted in 4.1.3.
- 4.2. Test is positive if pain is present in resting position but absent in the legs raised position.

#### 5. Posterior Pelvic Pain Provocation Test (Ostgaard et al., 1994):

- 5.1. Procedure:
  - 5.1.1. The participant is supine.
  - 5.1.2. The examiner passively flexes the participant's hip to 90°.
  - 5.1.3. The examiner applies a posteriorly directed force through longitudinal axis of the femur.
- 5.2. The test is positive if participant reports a deep pain in the gluteal/SI area during the test.
- 6. ASLR Test (Mens et al., 2001; Mens et al., 1999):
  - 6.1. Procedure:
    - 6.1.1. The participant is supine with legs straight and feet 20cm apart.
    - 6.1.2. The participant is instructed to lift the right leg and then the left approximately 20cm above the table without bending the knees.
    - 6.1.3. The participant is to maintain each leg in the up position for 5 seconds.
  - 6.2. The participant is asked to score the difficulty of the task on a 6-point scale, where any score > 0 is a positive test, the following are the score descriptions:

No difficulty	0
Minimally difficult	1
Somewhat difficult	2
Fairly difficult	3
Very difficult	4
Unable to do	5

- 6.3. The leg lift is repeated with anterior pelvic compression to mimic transversus abdominis activity and graded.
  - 6.3.1. Anterior pelvic compression is applied via a horizontal force directed medially to both ASIS.
- 6.4. The leg lift is repeated with posterior pelvic compression to mimic multifidus activity and graded.
  - 6.4.1. Posterior pelvic compression is applied via a horizontal force directed laterally to both ASIS by the examiner.

#### 7. Modified Trendelenburg Test (Albert et al., 2000):

- 7.1. Procedure:
  - 7.1.1. The examiner is located behind the standing participant.
  - 7.1.2. The participant is asked to stand on the left foot while flexing the right knee and hip to 90°.
  - 7.1.3. The participant is asked to stand on the right foot while flexing the left knee and hip to 90°.
- 7.2. The test is positive if the pelvis descends on the flexed side.

- 8. Passive Lumbar Extension Test (PLE): (Kasai, Phys Ther. 2006; 86:1661).
  - 8.1. Procedure:
    - 8.1.1. The participant is in the prone position.
    - 8.1.2. The examiner gently tractions both legs simultaneously while passively raising both legs about 30 cm from the bed, keeping knees extended.
  - 8.2. The test is positive when:
    - 8.2.1. During elevation of both legs during the test, the participant complains of strong pain in the lumbar region.
    - 8.2.2. The pain disappears when the legs are returned to the starting position.
  - 8.3. Participant's complaints of an abnormal sensation, such as mild numbness or a prickling sensation during this test are not considered abnormal.
- 9. Hurdle Step: (Cook et al. 2006, Butler et al. 2012):
  - 9.1. Procedure:
    - 9.1.1. The participant assumes the starting position by aligning the toes touching the base of the hurdle.
    - 9.1.2. The hurdle is then adjusted to the height of the tibial tuberosity.
    - 9.1.3. The dowel is positioned across the shoulders below the neck.
    - 9.1.4. The participant is asked to step over the hurdle with the right leg and touch the heel to the floor while maintaining the stance leg in an extended position.
    - 9.1.5. The right leg is then returned to the starting position.
    - 9.1.6. The same procedure 9.1.4 9.1.5 is repeated for the left leg.
    - 9.1.7. The participant can have as many as three trials to complete the task. Once they complete the task as described above, they do not need to continue.
  - 9.2. Scoring: it is possible to obtain a maximum of 18 points (9 points per side); scoring is as follows (named according to moving leg):

#### Right

Foot clears cord (does not touch).	5
Hips, knees, and ankles remain aligned in the sagittal plane.	2
Minimal to no movement is noted in the lumbar spine.	1
Dowel and hurdle remain parallel.	1
Left	
Foot clears cord (does not touch).	5
Hips, knees, and ankles remain aligned in the sagittal plane.	2
Minimal to no movement is noted in the lumbar spine.	1
Dowel and hurdle remain parallel.	1

#### 10. In-Line Lunge: (Cook et al. 2006, Butler et al. 2012):

#### 10.1. Procedure:

- 10.1.1. A 100 cm tape measure is fixed to the floor and the participant is asked to stand with the right foot on the tape with the toes at the 0 cm mark.
- 10.1.2. The examiner measures the participants' tibial length from the ground to knee joint line.
- 10.1.3. This distance is marked on the tape.
- 10.1.4. A dowel is placed vertically behind the back touching the head, thoracic spine, and sacrum.
- 10.1.5. The participant is instructed to grasp the dowel at the level of the cervical spine with the left hand and at the level of the lumbar spine with the right hand.
- 10.1.6. The participant is instructed to step forward with the left foot so that their heel touches the mark that was placed on the tape.
- 10.1.7. The participant then lowers the right knee enough to touch the floor behind the heel of the left foot.
- 10.1.8. The participant then returns to the starting position.
- 10.1.9. The same procedure 10.1.1 10.1.8 is repeated with the left foot on the tape, the right hand grasping the dowel at the level of the cervical spine, the left hand grasping the dowel at the level of the lumbar spine and the right foot stepping forward.

- 10.1.10. The participant can have as many as three trials to complete the task. Once they complete the task as described above, they do not need to continue.
- 10.2. Scoring: it is possible to obtain a maximum of 20 points (10 per side); scoring is as follows (named according to the forward leg):

Left	
Knee touches behind heel	2
Dowel and feet remain in sagittal plane	2
Dowel contacts maintained	2
Dowel remains vertical	2
No torso movement noted	2
Right	
Knee touches behind heel	2
Dowel and feet remain in sagittal plane	2
Dowel contacts maintained	2
Dowel remains vertical	2
No torso movement noted	2

#### 11. Rotary Stability (Cook et al. 2006, Butler et al. 2012):

#### 11.1. Procedure:

- 11.1.1. The participant assumes a quadruped position with their shoulders and hips at 90° relative to the torso.
- 11.1.2. The knees are positioned at 90° and the ankles should remain dorsiflexed.
- 11.1.3. The participant then flexes and elevates the right shoulder and extends/lifts the right hip and knee. The leg and hand are only raised enough to clear the floor by approximately 15 cm.
- 11.1.4. The right shoulder is then extended and the right hip and knee are flexed enough for the elbow and knee to touch.
- 11.1.5. The participant then returns to the starting position.
- 11.1.6. The same procedure 11.1.1 11.1.5 is repeated on the left side.
- 11.1.7. If the participant is unable to perform the maneuver with the ipsilateral arm and leg, they attempt it with a diagonal pattern using the opposite shoulder and hip as described in 11.1.1 11.1.5.
- 11.2. Scoring: it is possible to obtain a maximum of 12 points (6 per side). Scoring is as follows:

# Right Unilateral repetition 6 Diagonal repetition 2 Failure of diagonal repetition 0 Left Unilateral repetition 6 Diagonal repetition 2 Failure of diagonal repetition 0

#### 12. Trunk Stability Push-Up (Cook et al. 2006, Butler et al. 2012):

#### 12.1. Procedure:

- 12.1.1. The participant assumes a prone position with the feet together and the hands shoulder width apart at the appropriate position as described in 12.2.
- 12.1.2. The knees are then fully extended and the ankles dorsiflexed.
- 12.1.3. The participant is instructed to perform one push-up in this position. The body should be lifted as a unit; no lag should occur in the lumbar spine.
- 12.1.4. If the participant cannot perform the push-up in this position, the hands are lowered to the appropriate position as described in 12.2.
- 12.2. Hand position starts with the most difficult, with difficulty decreasing if the participant is unable to finish the movement:

- 12.2.1. Males perform one repetition with thumbs aligned with the top of the forehead. Females perform one repetition with thumbs aligned with the chin.
- 12.2.2. Males perform one repetition with thumbs aligned with the chin. Females perform one repetition with thumbs aligned with the clavicle.
- 12.3. Scoring: it is possible to obtain a maximum of 12 points, the scoring is as follows:

M	en

Thumbs at forehead level	12
Thumbs at chin level	5
Failure at chin level	0
Women	
Thumbs at forehead level	12
Thumbs at chin level	5
Failure at chin level	0

- 13. Active Straight Leg Raise from the Functional Movement Screen (ASLR FMS) (Cook et al. 2006, Butler et al. 2012):
  - 13.1. Procedure:
    - 13.1.1. The participant first assumes the starting position by lying with the arms in an anatomical position and head flat on the floor.
    - 13.1.2. The tester identifies mid-point between the ASIS and mid-point of the patella.
    - 13.1.3. A dowel is placed at the position identified in 13.1.2 perpendicular to the ground.
    - 13.1.4. The participant is instructed to lift the test leg as high as possible with a dorsiflexed ankle and an extended knee.
    - 13.1.5. During the test the opposite knee should remain in contact with the ground, the toes should remain pointed upward, and the head flat on the floor.
    - 13.1.6. Once the end range position is achieved, and the malleolus is located past the dowel then the score is recorded per the established criteria in 13.2.
    - 13.1.7. If the malleolus does not pass the dowel then the dowel is aligned along the medial malleolus of the test leg, perpendicular to the floor and scored per the established criteria.
    - 13.1.8. The test is repeated on the opposite leg.
    - 13.1.9. The ASLR test should be performed as many as three times bilaterally.
  - 13.2. Scoring: it is possible to obtain a maximum of 12 points (6 per side), the scoring is as follows:

#### Right

Malleolus resides between mid-thigh and ASIS	
Malleolus resides between mid-thigh and joint line	
Malleolus resides below joint line	
Left	
Malleolus resides between mid-thigh and ASIS	6
Malleolus resides between mid-thigh and joint line	
Malleolus resides below joint line	0