Evaluating The Effect Of A 10-Week Stabilization Exercise Program On The Postural
Stability And The Neuromuscular Control Of The Spine In Subjects With Subacute
Recurrent Low Back Pain

Dissertation

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Abstract

Low back pain (LBP) is one of the most common medical problems, afflicting around 80% of the world’s population at one time or other in their lifetime. The LBP episodes usually resolve quickly but a high number of individuals often relapse and suffer repeated episodes of LBP. This leads to a loss in work productivity and a decrease in their quality of life. Hence, it becomes imperative to understand and possibly correct the underlying factors responsible for recurrent LBP. Alterations in trunk muscle response patterns such as delays in muscle onset times, increased agonist/antagonist co-activation levels, and loss of anticipatory control have been observed in subjects with LBP. Altered muscle responses contribute to impaired postural control and instability which may be a contributing factor to the recurrence of LBP. Stabilization exercise programs have been shown to decrease the risk of LBP recurrence in a select group of patients with acute, first episode LBP. However, there is little evidence to demonstrate if the stabilization exercise program influences the underlying neuromuscular control of the spine and postural stability. Hence, the purpose of this study was to determine whether a 10-week stabilization exercise program improved postural stability or altered
the neuromuscular control in subjects with LBP. Two experiments were designed for that purpose: 1) A sudden perturbation test to assess the trunk muscle reflex responses, and 2) A seated postural control task to assess postural stability and trunk equilibrium control. 30 subjects (15 subjects with sub acute recurrent LBP, 15 healthy) aged between 18-55 years participated in the study. The LBP group was tested before (PRE) and after (POST) the 10-week physical therapy intervention program. The healthy control (CNTL) group was tested on one occasion.

In the sudden perturbation test it was observed that the subjects with LBP at PRE therapy had delayed and dampened trunk muscle reflexes compared to the CNTL group subjects. Postural stability assessed using a fractional Brownian motion analysis of the force plate data from the wobble chair revealed that the subjects with LBP at PRE therapy had increased postural sway and large trunk displacements. In addition, the subjects with LBP displayed compensatory flexor activation during the seated postural task compared to the CNTL group subjects. At POST therapy in the subjects with LBP, the reflex responses remained delayed but there was a significant increase in the reflex amplitudes. In the seated postural task, there was a decrease in the postural sway and a reduction in the compensatory trunk flexor activity. In addition compared to the CNTL group, the LBP group at POST therapy demonstrated increased extensor activity. In
conclusion, a 10-week stabilization program improved the neuromuscular control of the spine and postural stability in subjects with LBP.
Dedication

This work is dedicated to my parents, who have been a source of inspiration and strength throughout this project.
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Chapter 1 Introduction

1.1 Low back pain

Low back pain (LBP) is usually defined as pain, muscle tension, or stiffness localized below the costal margin and above the inferior gluteal folds, with or without leg pain.\(^1\)

In 2002, approximately one quarter of U.S. adults reported having LBP lasting at least one whole day, and 7.6\% reported at least 1 episode of severe acute LBP within a year period.\(^2\) Acute LBP episodes usually resolve quickly but often individuals relapse, evolving into chronic or recurrent conditions. It is estimated that at least 60\% to 86\% of the individuals with an acute LBP episode will experience at least one recurrence within a year period.\(^3\) Ricci and colleagues\(^4\) reported that employers spend an estimated $ 7.4 billion/year on workers between the ages of 40 to 65 years with LBP related expenses. Individuals with chronic or recurrent LBP have substantially higher total medical and indemnity costs and longer periods of work absences. Apart from loss in work productivity, recurrent LBP is also associated with functional impairment, social and physical activity limitations and reduction in the quality of life.
The causes of LBP remain elusive, however an increasing number of studies suggest that LBP and the high recurrence rate may be associated with instability in the spinal system.\textsuperscript{5-10} According to the American Academy of Orthopaedic Surgeons,\textsuperscript{11} spinal instability has been defined as the abnormal response to applied loads, characterized by movement in the motion segments beyond normal constraints. The motion segment here is the smallest functional spinal unit which exhibits the overall characteristics of the spine. The motion segment consists of two adjacent vertebrae, an intervertebral disc, various ligaments and apophyseal joints. The stability to the motion segment is provided by the intervertebral discs, ligaments and facet joints which restrict the range of movements for the segment. These structures are largely defined as the passive components of the spinal system. Apart from the passive components, spinal stability is also governed by what are referred to as active components,\textsuperscript{5} which consist of the spinal muscles and the neural control which governs their actions. Muscles have intrinsic stiffness and resist deformation to stabilize the joints. The neural control initiates active stiffness and force generation in the muscles allowing them to move the joints or resist loads as required.

Damage to any of these passive or active components or deficits in the actions of the active components will potentially impair their ability to restrict the segmental motion resulting in an abnormal response to normal physiological loads. The abnormal response in the motion segments can result in excessive strains or deformations in the spinal
structures causing injuries. McGill et al\textsuperscript{12} suggests that these excessive deformations may result in injuries to the spinal structures and may be a source of pain. Damage to the structures which include the intervertebral disc, facet joints, vertebral segments, muscles, ligaments, tendons, nerves/receptors can be a source of pain.\textsuperscript{13} One of the hypotheses behind the high recurrence rate in the episodes of LBP is that although the pain from a LBP episode may resolve, the deficits or impairments in the active components remain, leaving the individual susceptible to further injuries.\textsuperscript{14} Hence, in order to understand LBP and the possible causes for the high recurrence rate, understanding spinal stability and the role of the active components in spinal stability becomes crucial.

Earlier studies to characterize spinal stability have focused on the stability of the spine during static conditions which is referred to as static stability.\textsuperscript{15} Static stability refers to a state of equilibrium where the sum of the forces acting on the system is zero and the body or object is not in motion. Spinal stability can also be quantified under dynamic conditions; where in the stability of the system is evaluated as a function of time, when the object or body is in motion\textsuperscript{16,17}. The role of the active components becomes significant when maintaining spinal stability during dynamic conditions and thus evaluating stability during conditions where rapid corrections are required allows one to interpret and evaluate the role of the active components in the overall spinal stability system. Figure 1 illustrates the role of the passive and active components in
maintaining spinal stability. The different parts of the model and the findings related to LBP will be discussed in depth in the following sections.
Figure 1 Spinal Stability Model. The model illustrates both the passive and active components of the spinal stability system. The passive component box represents the individual vertebral segments, intervertebral discs, facet joints and ligaments. The active components consist of the muscles and the neuromuscular control which governs their
actions. A time delay also exists for the intrinsic properties of the muscle due to its visco-elastic properties, but the delays are considerably smaller compared to that of the reflex and voluntary responses and hence not represented here.

1.2 Spinal Stability Components

1.2.1. Passive components:
The passive components consist of the individual vertebral segments, the intervertebral discs, the ligaments and the facet joints (Figure 2). The vertebral segments articulate with each other and with the intervertebral discs and ligaments allowing for forces to be transferred between segments. The intervertebral disc is a load bearing structure located between the vertebrae of the spine and allows for the transfer of compressive forces. The ligaments are uniaxial structures and resist tensile forces while buckling under compressive loads. A spine consisting only of the passive components and devoid of the active components buckles under a mere compressive load of 90 N, a load which is significantly lower than the weight of the upper torso. Injuries to the passive structures such as fractures in the vertebral segments, disc ruptures, and injuries to the ligaments would create instability in the spinal system leading to increased range of motion. In such scenarios, the active components could theoretically compensate for the instabilities by providing increased stiffness and hence reducing the range of motion in these segments.
Figure 2 Passive components of the spinal stability system. Image obtained from the public domain of Wikipedia Commons and the image is free from copyright.
1.2.2 Active components

The active components consist of the muscles surrounding the spine and the neuromuscular control which governs their actions. The active components stabilize the spinal structures by providing stiffness and modulating force production in the spinal muscles. The main focus of this dissertation is in the LBP associated with the lumbar region of the spine. Hence, the main content in the description of the muscles of the spine will focus on the abdominal and lumbar muscles.

1.2.2.1 Abdominal and Lumbar Muscles:

The abdominal trunk muscles (Figure 3) include the external oblique, internal oblique, rectus abdominis and the transverse abdominis. The lumbar muscles (Figure 4) include the psoas major, intertransversarii lateralis, quadratus lumborum, interspinalis, intertransversarii mediales, multifidus, and erector spinae (longissimus lumborum, iliocostalis lumborum, and the spinalis dorsi). The intertransverse and the interspinalis are small intersegmental muscles which lie between the transverse processes and the spinous processes respectively.
Figure 3: Figure illustrating the trunk abdominal muscles. Image obtained from the public domain of Wikipedia Commons and the image is free from copyright.
Figure 4 Figure illustrating the muscles of the back. Image obtained from the public domain of Wikipedia Commons and the image is free from copyright.
Among the trunk back muscles, the lumbar multifidus has received great attention in recent studies because of its purported role in intricately controlling the motion of the individual vertebral segments.\textsuperscript{23-27} The multifidus is one of the largest intrinsic muscles of the lumbar spine with its fascicles (bundles of muscle fibers) spanning 2 to 5 joint segments. The fascicles vary in length with the longest and the most superficial passing from one vertebrae to the third or fourth above, while the deepest fibers connecting two contiguous vertebrae.\textsuperscript{27} A one to one relationship exists between the vertebral segment and the innervations of the multifidus at the particular segment with each fascicle of the lumbar multifidus and the corresponding facet joint being innervated by the same medial branch of the dorsal ramus enabling it to have a tight control over the motion of the segment.\textsuperscript{28} In addition, the multifidus also has a high proportion of type I muscle fibers and is highly vascularized which enables it to be a very efficient, fatigue resistant postural stabilizer.\textsuperscript{27} These evidences have led researchers to believe that the lumbar multifidus may be an important local stabilizer of the vertebral segments and any deficits in its activity may compromise the local segmental stability.\textsuperscript{14} Animal studies\textsuperscript{28} have shown that after an injury to the disc on one side, loss of cross sectional area and infiltration of adiopocytes occurs at the ipsilateral segmental level of multifidus, while a denervation to the multifidus muscle causes changes in the multifidus ipsilateral to the lesion across multiple segments. These results might explain the observed atrophy in the lumbar multifidus ipsilateral to the pain in subjects with LBP.
within 24 hours after their first episode.\textsuperscript{29} It was also observed that the cross sectional area of the multifidus muscle on the ipsilateral side was smaller than the contralateral side even after the patients recovered from their first episode of LBP and had resolution of pain and restoration of function with conventional medical and physical therapy interventions. It has been suggested that the persistent dysfunction observed in the multifidus may contribute to loss of stiffness in the spinal system and make the individual susceptible to future injuries.\textsuperscript{14}

In addition to the lumbar multifidus, the transverse abdominis, one of the trunk abdominal muscles has also been suggested to be an important contributor to local segmental stability. It was demonstrated in healthy control subjects that the transverse abdominis tended to exhibit anticipatory control with a pre-activation being observed in the muscle before a rapid arm movement.\textsuperscript{30} This anticipatory activation in contrast was observed to be missing in individuals with LBP. However the pre-activation observed in the muscle may be task and direction dependant. Hodges and colleagues\textsuperscript{30} demonstrated the pre-activation in only the contralateral side of the arm being raised and did not measure the ipsilateral side and assumed a non-direction specific activation in the transverse abdominis. Based on this, Richardson, Hodges, and colleagues\textsuperscript{14} developed a theory and corresponding LBP rehabilitation program, which states that a deep musculofascial corset or hoop is formed by the transversely oriented fibers of the transverse abdominis, the middle and posterior layers of the lumbar fascia, the lower
portions of the internal oblique, the lumbar multifidus and the lumbar spine. The exercise program includes the abdominal “hollowing exercise” which trains individuals to preferentially activate the transverse abdominis. This action along with a conscious co-activation of the lumbar multifidus induces activation of the diaphragm and the pelvic floor muscles which altogether forms a pressurized cylinder, thus in theory contributing to increased stiffness and reduction in the joint segmental laxity. This “corset-like” mechanism theory is the basis of the rehabilitation program used to train individuals in the study reported in this dissertation.

The theory developed by Hodges relies on the assumption that the transverse abdominis acts uniformly on both the ipsilateral and contralateral sides thus lending credence to the hoop formation. However, Allison and colleagues measured both the ipsilateral and the contralateral transverse abdominis and found that the pre-activity was found only in the transverse abdominis contralateral to the arm being raised. In addition, the loss of anticipatory response observed in individuals with LBP has also been demonstrated in individual without a history of LBP when they are stressed. These findings thus warrant further study on the overall effects of the rehabilitation program developed by Richardson, Hodges, and colleagues in individuals with LBP.
1.2.2.2 Neuromuscular control:

The CNS co-coordinates and modulates the forces developed in the different muscles for movement and to resist disturbances while maintaining balance. Sensory information related to muscle state, postural equilibrium, joint stability are all projected on to the CNS. Three levels of control exist within the CNS; the motor areas of cerebral cortex, the brain stem and cerebellum, and the spinal cord which coordinate and regulate reflex (M1,M2) and voluntary (M3) muscle responses to achieve the desired state and maintain a stable equilibrium. This is a highly simplified view of the CNS, with each level having multiple sub components that are beyond the scope of this dissertation.

Figure 5 illustrates the simplified hierarchical role of the different levels which exist in the neuromuscular control of the spine. The spinal level of motor control integrates and processes proprioceptive information unconsciously to elicit muscle activation through reflex and neural pathways. The motor areas of cerebral cortex represent the highest level of control integrating voluntary movement with automatic pre-planned motor control patterns based on somatosensory, acoustic, nociceptive, and other continuous afferent information to coordinate muscle responses. The cerebellum continuously receives sensory information and regulates the information that it relays to the cerebrum and brainstem. The motor centers of the brain stem integrates the sensory information with afferent information from the vestibular and the visual centers.
along with integrated outputs from the cerebellum and cortex to control the preplanned task of maintaining postural equilibrium.\textsuperscript{43}

Figure 5 Hierarchical role of the different levels of control within the CNS in the neuromuscular control of the spine. Any perturbation to the spine initiates a change in the length of the muscle and elicits a quick M1 stretch reflex through the spinal cord. Sensory information related to muscle and joint position is also relayed to the different
centers of the brain which responds with appropriate M2 and M3 responses in order to maintain stability with inputs from pre-established motor control patterns.

There are two control mechanisms through which the CNS coordinates the muscle forces, the feedforward mediated control and the feedback mediated control. The interplay between the feedforward and feedback mechanisms allows for an efficient and optimum control paradigm. Feedforward control refers to the anticipatory mechanisms which exists where in the muscles are pre activated by the CNS either in preparation of movement or in anticipation of an external perturbation. Feedback control refers to the mechanisms which exist to react to changes in muscle state, joint stability or equilibrium conditions. The feedback mediated control occurs through a series of reflexive and voluntary responses. Figure 6 depicts the feedback response of a muscle to a perturbation which occurs through reflex and voluntary responses. The reflex responses consist of a short latency reflex (M1) mediated at the spinal level of the motor control and a medium latency reflex (M2) mediated through the higher centers which include the cerebellum, brain stem and the motor cortex. Reflex responses are characterized by the time delay and the amplitude of the response referred to as the reflex gain.
Figure 6 Feedback Control Responses. A typical feedback response to a perturbation observed in a muscle consists of a short latency M1 response, followed by a medium latency M2 response and finally followed by the M3 voluntary responses.

Neuromuscular impairments in the feedforward and feedback mechanisms have been reported in patients with LBP. Anticipatory activations seen in the back extensor
muscles during arm raising tests in healthy subjects have been observed to be delayed in subjects with LBP. Delays in trunk muscle reflexes and longer muscle shut off times have been observed when subjects with LBP experienced rapid perturbations to the trunk.\textsuperscript{53-55} The delayed muscle reflex responses have been suggested to be a significant predictor for low back injuries.\textsuperscript{53} Although, the reflex responses in these studies have not been classified as being either M1 or M2, the onset times of the reflex responses for the subjects with LBP reported in the studies are in the ranges of the 50-80 ms which correspond to the onset times of the M2 responses. Hence, the delays observed in the reflex responses of the subjects with LBP are most likely delays in their M2 reflex responses. In addition, alterations in muscle recruitment patterns such as increased agonist/antagonist co-activation and increased flexor activations during functional tasks have also been repeatedly observed in subjects with LBP.\textsuperscript{56-60} Agonist/antagonist co-activations significantly and rapidly increases the active stiffness in the spinal structures enhancing stability and has been suggested to be a strategy adopted to compensate for the deficits in the neuromuscular control.\textsuperscript{61, 62} However, the co-activations comes at the cost of increased compressive loads on the intervertebral disc which may be detrimental to its health.\textsuperscript{63}

Pain brought on by injuries to the spinal structures can have potent effects on the motor control functions of the CNS and perhaps may have led to the observed impairments.\textsuperscript{10, 64, 65} Nociceptive information is well integrated at the higher levels of the
CNS and can have a direct impact on the motor responses. An antalgic gait, decreased ranges of movement, loss of the spinal curves are some of the more obvious pain avoidance motor patterns. Less obvious are the underlying physiological changes at both the peripheral and central level. Altered patterns of muscle recruitment to protect the involved joint may be initiated during the acute stage of pain and may persist as the pain becomes more chronic. A prolonged period of nociceptive input has been observed to alter the behaviors of spinal cord neurons and induce structural changes at the motor cortex level. Additionally, experimentally induced pain in the structures has been shown to cause muscle atrophy, muscle inhibition and delayed muscle activation. As these physiological changes occur, loss of control over active stability may occur and a susceptibility to instability may occur at the involved segment as a consequence, which may contribute to further pain or further injury.

Van Dieen et al suggest that given these differences in the active components, patients with LBP would be unable to rapidly develop and sustain adequate trunk muscle forces thus limiting their capacity to protect against perturbations to their trunk equilibrium. These deficits and impairments would show up as increased sways during postural tasks which has encouraged researchers to use measures of postural control as a means to characterize the overall status of the underlying neuromuscular system
1.3 Postural Stability in subjects with LBP

Traditionally, measuring standing postural sway has been used to characterize postural stability. The postural stability can be characterized through analysis of the center of pressure (COP) data obtained through a force plate and includes parameters such as mean center of pressure, 95% ellipse area, root mean squared displacement, stabilogram diffusion analysis, rescaled range analysis, detrended fluctuation analysis and Lyapunov analysis. Subjects with LBP typically display higher numbers in these measurements. These higher numbers have been interpreted as the inability to restrict movements or fine tune displacements which should represent a state of instability. However, during standing postural tasks, the COP measure is the net effect of the motions at the trunk as well as the joints of the lower extremities. Recent studies to isolate the trunk postural control have developed methods, and instruments to study seated postural sway. In a recent study, Radebold and Cholewicki conducted seated stability tests on the lumbar spine using hemispherical balls attached to the bottom of a seat. This apparatus isolated motion to the lumbar and pelvic region. The location of the COP of the subject was measured during the test using a force plate. Trunk postural control was analyzed using the stabilogram diffusion analysis (SDA) which characterizes the behavior of the COP as a correlated random walk. The analyses relate mean squared displacements of the COP and the corresponding periods of time. The authors observed increased trunk displacements during the short intervals of time in the subjects with LBP. The authors also observed delayed trunk muscle reflexes in the
same set of subjects during a sudden force-release task and found that the increased postural sway was associated with the delays in the trunk muscle reflexes. This relation will be further explored in the subsequent chapters.

In summary, subjects with LBP tend to display impairments in their neuromuscular control and postural stability. Evidence from recent studies suggest that although the pain from the episodes of LBP resolves, the underlying neuromuscular control remains impaired, which makes the individuals susceptible for further low back injuries. Hence, the newer approaches in the treatment of LBP have focused on identifying these impairments and designing therapy programs to address them.

1. 4 Specific Stabilization Exercise (SSE) therapy as an intervention for acute, recurrent LBP

Among the many non-surgical methods of treatment of LBP which include physical therapy programs, chiropractic interventions, and medical management practices (e.g. medications, advice on bed rest and activity, and abstention from work) the SSE program has garnered a lot of interest in the research community. The SSE program aims to improve the neuromuscular control factors which influence spinal stability. The development of the SSE program is based on the corset mechanism theory described previously. This theory relies on the concept that the overall stability is the sum of the
stability of its parts. Hence the intersegmental control or stability at the individual segmental level is crucial and contributes to the overall spinal stability.

Concepts of SSE were followed in this study. The program was based on a progression of training which occurs through three stages and is described in detail in Appendix D. In brief, stage 1 includes general stretching exercises for the trunk and leg extensors, general conditioning exercises, and motor control exercises which involve teaching the patient isolated contractions of the lumbar multifidus and the transverse abdominis through ultrasound feedback. In addition, Stage 1 also emphasizes patient education and pain management. General conditioning exercises include treadmill walking at reported perceived exertions. In the next stages, the complexities and the intensity of the tasks increase with the motor control exercises emphasizing co-activation of the multifidus and transverse abdominis in other complex functional activities such as in walking, lifting, and bending. General conditioning activities in the second and third stages include swimming, treadmill training and aerobics which increase in intensity during the final stage. The detailed protocol for the progression in the SSE program is listed in Appendix D.

Hides et al\textsuperscript{82} demonstrated that the SSE program was effective in reducing pain and preventing recurrence in a select population of subjects with acute, first episode of nonspecific, unilateral LBP. However, there is little evidence to demonstrate if this SSE
program influences the underlying neuromuscular control of the spine and postural stability. Moreover, it is unknown if this exercise approach is able to affect the cycle of recurrence in individuals who have experienced episodic LBP. The SSE approach is being widely used today by many physical therapists as an intervention program for treating LBP without truly understanding how this program might prove beneficial in a wider range of patient conditions. Hence, it’s important to address the gap in the existing knowledge of whether the SSE training improves the neuromuscular control factors in subjects with LBP and if it perhaps influences the recurrence rate of episodes of LBP.

1.5 Summary of findings related to patients with LBP, and the SSE program

Patients with LBP tend to exhibit altered neuromuscular responses such as delayed trunk muscle reflexes and altered muscle activations levels as compared to subjects without LBP. Subjects with LBP also tend to exhibit postural instability which might be associated with the impairments observed in their neuromuscular control. Alterations in the neuromuscular control and postural instability are thought to contribute to the high recurrence rate in episodes of LBP.

The SSE program is aimed at individuals who demonstrate clinical symptoms of impaired neuromuscular control as being a major contributing factor to their pain and functional limitations. The developers of the SSE program claim that the program improves neuromuscular control factors; however, there is little evidence to demonstrate that.
The present study aims to evaluate the effect of a 10-week SSE program on the neuromuscular control factors and postural stability in subjects with subacute recurrent LBP. Two experiments were designed for that purpose: 1) A sudden perturbation test to measure trunk muscle reflexes, and 2) A seated postural task to assess postural control and muscle recruitments. Subjects were screened and recruited for the LBP group and the control group according to the inclusion and exclusion criteria (Appendix A). Subjects in the LBP group attended the physical therapy twice/week for 2 weeks (weeks 1 and 2), then once per week for four weeks (weeks 3-6), then once every 2 weeks (weeks 8 and 10) for the 10-week SSE program. The consent form and the detailed research protocol are listed in Appendix B and Appendix C respectively, and the SSE protocol as followed during the physical therapy program is listed in Appendix D. The subjects in the LBP group were tested at the beginning of the 10-week period for a baseline recording and at the completion of the SSE training (10 weeks). Experimental measurements on the healthy control subjects were recorded only once. The hypotheses for the study are as follows:

**Hypothesis 1** Patients with subacute recurrent LBP before therapy would demonstrate impaired trunk muscle reflexes and impaired postural control as compared to healthy subjects

**Hypothesis 2** A 10-week stabilization exercise program would lead to improved trunk muscle reflexes and trunk postural control in subjects with subacute recurrent LBP
1.6 Document organization

This dissertation has been divided into chapters, with chapters 2, 3 and 4 serving as independent manuscripts. Chapter one introduces the topic of LBP, neuromuscular control and postural stability. Chapter two compares trunk muscle reflexes in subjects with LBP before and after a specific stabilization exercise program and matched healthy control subjects. Chapter three compares stability indices during a seated postural control task in the subjects with LBP before and after a specific stabilization exercise program and matched healthy control subjects. Chapter four compares muscle activation levels and neuromuscular patterns during the seated postural task in subjects with LBP before and after a specific stabilization exercise program and matched healthy control subjects. In Chapter five, conclusions of the present study and ideas for future studies are presented.
Chapter 2 A stabilization exercise program influenced the amplitudes but not the latencies of trunk muscle reflexes in patients with subacute, recurrent LBP

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2.1 Abstract

**Study design:** A perturbation test was used to compare trunk muscle reflexes in 15 patients with subacute, recurrent LBP, before and after 10 weeks of trunk stabilization exercise (SE) program and 15 matched control subjects (CNTL).

**Objective:** It was hypothesized that patients with LBP would have delayed and dampened trunk extensor muscle reflex responses compared to the CNTL subjects and a 10-week SE program would improve the reflexes in the patients with LBP.

**Summary of background data:** Delayed muscle onsets have been observed in patients with LBP in experiments involving sudden loading of the trunk. Altered muscle responses contribute to impaired postural control and instability which may be a contributing factor to the recurrence of LBP. SE programs have been shown to decrease the risk of LBP recurrence in a select group of patients with acute, first episode LBP. It is not known if SE programs influence the trunk muscle response patterns to sudden perturbations.

**Methods:** Subjects kneeled in a frame while connected via a harness and cable system to a servomotor. The cable tension applied repeated flexion loads at approximately the T6-T7 level of the trunk to elicit reflexes. Electromyograms were recorded bilaterally from the L5 multifidus and L3 erector spinae. Reflex latencies and amplitudes were calculated.
**Results:** The LBP group at PRE therapy had delayed reflex responses in the multifidus and the erector spinae compared to the CNTL group. POST therapy reflex latencies did not change but reflex amplitudes increased.

**Conclusions:** Increased reflex amplitudes after the 10-week stabilization exercise program in patients with LBP could limit excessive movement of the spine when perturbed; potentially reducing strain on the ligaments, providing a protective benefit.

**Keywords:** Stabilization Exercise, Low back pain, Sudden loading, Reflex Gain, Multifidus

**Key points:**

- Trunk muscle reflexes elicited based on a sudden loading protocol were compared in patients with LBP before and after a 10 week stabilization exercise program and matched healthy control subjects.
- Patients with LBP had delayed reflexes in the lumbar multifidus and erector spinae compared to the control subjects. Additionally reflex gains in the multifidus measured using fine wire electromyography were lower in patients with LBP.
- After the 10-week stabilization exercise program the patients with LBP demonstrated increased reflex gains in their multifidus and erector spinae while still retaining their delayed onset times.
• Delayed reflexes contribute to spinal instability and have been suggested as a predisposing factor for LBP. Increased reflex amplitudes might compensate for the delay in the onset time and provide for a strong and robust response to achieve spinal stability.

Mini abstract:

Patients with LBP demonstrated stronger trunk muscle reflexes after a 10-week stabilization exercise program. However the onset times of the trunk muscle reflexes remained delayed in the patients with LBP compared to matched healthy control subjects.
2.2 Introduction

Persons with LBP respond differently to sudden changes in loading conditions on the torso as compared to persons without a history of LBP. Studies have demonstrated LBP is associated with delayed trunk muscle responses to perturbations,\textsuperscript{30, 55, 83} delayed muscle shut-off times after an external load has been removed,\textsuperscript{54} and increased co-activation during complex tasks.\textsuperscript{84}

Impairments in the trunk muscle responses to sudden perturbations may affect the active (muscle) control of the spine, and predispose individuals to injury or re-injury.\textsuperscript{53} There are three components in the trunk muscle reflex response to a rapid perturbation; a short latency reflex (M1), a medium latency reflex (M2), and a long latency component (M3).\textsuperscript{55} M1 is dominated by the monosynaptic stretch reflex, initiated by the muscle spindle afferents.\textsuperscript{85} The M2 response is thought be pre-programmed and mediated by several circuits which include the brain stem,\textsuperscript{39} cerebellum,\textsuperscript{86} and primary motor cortex.\textsuperscript{87} There is also evidence that the M2 may depend heavily on signals originating in mechanoreceptors of the interspinous ligaments.\textsuperscript{88} The M2 response thus requires more neural processing and longer pathways than the M1, and hence longer latencies are observed. Onset times as fast as 19.3 (SD 2.1) ms for the M1 and 44.6 (SD 2.5) ms for the M2 have been observed in the erector spinae of healthy subjects.\textsuperscript{49} The M3 response with onset times in the ranges of 120-180 ms is considered a voluntary response initiated by cortical motor areas.\textsuperscript{89}
Studies involving sudden unexpected perturbations to the trunk\textsuperscript{53, 54, 90, 91} have reported onset times of 60-120 ms in their trunk muscle reflex responses, which correspond to M2 response times. The lack of M1 responses in these studies may be explained by the setup. With perturbations applied at the T9-T10 level, lumbar muscles may not have been rapidly stretched. For M2 responses, Radebold et al\textsuperscript{54} reported average onset times of 85 (SD 25) ms in the back muscles in patients with LBP in reaction to a sudden load as compared to an average onset time of 69 (SD 8) ms in the control subjects. Cholewicki and colleagues\textsuperscript{53} in a 2 year prospective study demonstrated in 292 college athletes that, on average, the athletes who went on to sustain a low back injury (LBI) had a 14 ms longer muscle shut-off time after an external load was removed as compared to athletes who did not sustain an LBI. In addition, there were no significant changes in the muscle shut-off times before and after the LBI in the athletes. The authors concluded that delays in reflex latencies were a significant predictor and a preexisting condition for LBP.

Pain,\textsuperscript{92} damage to the nerves / receptors, muscle wasting leading to loss of type II fibers,\textsuperscript{26} fat infiltration,\textsuperscript{93} and reduced endurance in the muscles\textsuperscript{54} may all contribute to the greater variability seen in the amplitude of the reflex response in patients with LBP compared to healthy subjects as observed by Danneels and colleagues.\textsuperscript{95} These altered muscle response patterns are thought to contribute to the eventual impaired postural control and poor balance observed in persons with LBP.\textsuperscript{54, 77, 96} Moreover, a persistent
imbalance in muscle response patterns may alter the motor control and be a potential reason for recurrent low back pain.\textsuperscript{10}

The question arises then: is it possible to change these muscle response patterns to make them quicker and stronger? In theory this may reduce the risk of LBI. Rehabilitation programs aimed at retraining motor control of the back muscles such as the specific stabilization exercise (SSE) have demonstrated some success in reducing the incidences of recurrence in LBP in persons with first episode, acute LBP, but there is little evidence to document the changes, if any, in the muscle response patterns.\textsuperscript{14} Understanding the influence of the SSE program on the muscle response patterns may provide insight as to how and why this program could be beneficial to patients at risk for LBI or re-injury.

The purpose of this pilot study was twofold. The first was to quantify differences in the reflexes of the back muscles between the subjects with subacute, recurrent LBP and healthy controls (CNTL). The second was to determine if a 10-week physical therapy (PT) program incorporating the SSE approach influenced the trunk muscle reflexes in the patients with LBP. It was hypothesized that the LBP group before therapy (PRE) would demonstrate delayed reflexes and reduced amplitude as compared to the CNTL group; and a PT intervention aimed at altering the neuromuscular control in the LBP group would lead to quicker reflexes and increase in the reflex amplitudes after therapy (POST).
2.3 Methods

2.3.1 Subjects

15 subjects (6 female) with a primary complaint of subacute, recurrent LBP, and 15 subjects (6 female) with no history of significant LBP in the past 2 years volunteered for the study. Subjects with LBP were included if the duration of pain in the current episode was less than or equal to 8 weeks, and had experienced at least 1 separate episode in the past year which would have had seemingly resolved. Subjects were excluded if they had a prior history of lumbar surgery and presence of sciatica or medical conditions which might affect spinal control such as cauda equina syndrome, neurological disorders, fracture, cancer, infection, or systemic disease. The subjects in the control group were matched to the subjects in the group with LBP by their age, gender, height and weight. Informed consent was obtained from each subject following procedures approved by the Biomedical Institutional Review Board at the University.

2.3.2 Experiment protocol

Subjects provided demographic information, and all subjects underwent a baseline examination by one of the investigators. Their trunk and hip range of motion were recorded. Subjects with LBP filled out a numerical rating scale for their best and worst pain in the past 24 hours, with 0 being the lowest pain and 10 being the worst. The numerical rating scale has been shown to have good sensitivity and reliability.97
Trunk muscle activity was recorded with surface electromyography (EMG) using the system from Motion Labs (Baton Rouge, LA) bilaterally from the L3 erector spinae (ES S) at 3 cm from the midline at the L3 spinous process and the L5 multifidus (Mult S) at 3 cm from midline at the L5 spinous process. In addition, fine wire EMG recorded activity from the deep fibers of the L5 multifidus (Mult FW) using paired fine wire electrodes (0.002 x 8’’ Nickel alloy insulated wires, Chalgrin Inc, CA). The fine wire electrodes were inserted adjacent to the lamina of the L5 vertebrae based on measurements made using ultrasonography (Titan, Sonosite, Bothell, WA). For normalization of the EMG signals from the multifidus and erector spinae, subjects performed the Biering-Sorensen test.

For the reflex perturbation test, the subject was seated in a kneel chair (Figure 2) with a belt across the lower torso to restrict pelvic motion. A metal bar was placed across the thorax at the inferior angles of the scapulae (T6-T7). A harness from the bar was attached to a cable from the servomotor (Pacific Scientific, Rockford, IL). Tension in the cable provided a constant isotonic flexion preload of 100 N. Pseudorandom force perturbations of +/- 30 N were superimposed on the 100 N preload. The loads were measured by a torque transducer (Omega TQ301 series, 0-45 Nm, Stamford, CT) attached to the shaft of the motor. Six perturbation trials of 10 s each were performed for each subject. The LBP group was tested PRE and POST the 10-week SSE program, while the CNTL group was tested on one occasion using the same protocol.
The SSE PT intervention was modeled using the program by Richardson and colleagues. Subjects attended 10 visits, each lasting 45 minutes, distributed over a 10-week period. The subjects were also instructed to carry out a home exercise program for 30 min/day during the 10-week program. Initial part of the SSE program involved exercising the abdominal and low back muscles with specific exercises for the multifidus.
and the transverse abdominis. Initial training for recruitment of these muscles was augmented with biofeedback via rehabilitation ultrasound imaging and exercises were progressed to recruit the rest of the trunk muscles. Patients also performed an aerobic activity such as walking, biking, or swimming.

2.3.3 Data analysis

The raw EMG and torque transducer signals (Figure 3) were collected and sampled at 2000 Hz using LabVIEW (National Instruments, Austin, TX) and processed using Datapac (RunTech Inc., Mission Viejo, CA). The EMG signals were low pass filtered at 100Hz using a 5th order zero lag Butterworth filter and rectified. A single file was created by concatenating the six trials for each subject. The onset time for each perturbation was detected and a -100msec to +400 msec window was created around the onset time. Within this window, the muscle activity was averaged for each of the channels and peak muscle responses were identified (Figure 4). The latency of the muscle activity was detected as the time when the magnitude increased by 2 standard deviations above the baseline activity. The area under the reflex peak was calculated as the reflex gain. Mean data from a corresponding window of the Biering-Sorensen test was used to normalize the reflex gain to provide a standardized comparison between muscles, times, and subjects.
Figure 8 Raw EMG response patterns to the pseudo-random perturbations for a CNTL group subject

2.3.4 Statistical analysis
Subject characteristics (age, height, and weight) were compared between groups using an independent t test (Excel 2007, Microsoft, Redmond, WA). The average of the best and worst pain rating scores was compared PRE and POST therapy in the group with LBP using a paired samples t test.
A mixed effects regression modeling framework (SAS, v9.1, SAS Institute Inc., Cary, NC) was used to model the reflex latency and gain computed for each muscle. A random intercept for each subject was included in order to separate within-subjects and between-subjects variability. Empirical “sandwich” estimators of the variance-covariance matrix for the fixed effects parameters (e.g. group, PRE/POST) were used to alleviate the impact of any departures from the defined covariance structure. Data is presented as means and standard deviations. For all statistical tests, a p <= 0.05 was considered significant.

2.4 Results

2.4.1 Subjects
Subjects in the LBP and the CNTL groups were similar in age, height and weight (Table 1). Average pain ratings improved (P= 0.0039) for the LBP group POST therapy and were clinically important (Δ in pain >= 1.5).97

Five out of the 15 subjects in the LBP group did not complete the study. Two attended only the first PT session while 2 more attended only 3-4 sessions, and 1 attended all PT sessions, but could not be contacted for the POST test. Reflexes were not elicited in one of the remaining 10 subjects with LBP at the PRE or POST therapy sessions, and in two of the subjects in the CNTL group. Hence the PRE analysis included 13 subjects for the LBP group and 13 matched CNTL subjects, and 9 subjects for the LBP
PRE therapy and POST therapy analysis. Averaged reflex response for a control subject (Figure 4A), a subject with LBP at PRE therapy (Figure 4B) and the same subject at POST therapy (Figure 4C) are shown.

Table 1 Subject characteristics for the sudden perturbation test experiment

<table>
<thead>
<tr>
<th></th>
<th>CNTL n=15 (6 female)</th>
<th>LBP n=15 (6 female)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>35 (SD 10.1)</td>
<td>32.3 (SD 8.2)</td>
<td>0.31</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.75 (SD 0.12)</td>
<td>1.75 (SD 0.08)</td>
<td>0.43</td>
</tr>
<tr>
<td>Weight(kg)</td>
<td>84.5 (SD 19.9)</td>
<td>81.2 (SD 19.6)</td>
<td>0.32</td>
</tr>
<tr>
<td>BMI(Kg/m2)</td>
<td>27.3 (SD 4.2)</td>
<td>26.4 (SD 6.0)</td>
<td>0.31</td>
</tr>
<tr>
<td>Pain Rating</td>
<td>N/A</td>
<td>PRE: 4.03 (SD 1.19)</td>
<td>0.0039</td>
</tr>
<tr>
<td></td>
<td></td>
<td>POST: 2.27 (SD 1.07)</td>
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</table>
2.4.2 LBP group at PRE therapy VS. CNTL group

The average latencies for the LBP group at PRE therapy were in the range of an M2 response. These latencies were delayed in all the three measurements of Mult FW, Mult S and ES S compared to the CNTL group (Figure 5A). There was a significant interaction between the group and side for the Mult FW (p = 0.0017), and ES S (p = 0.0083) but not for Mult S. The left side Mult FW in the LBP group at PRE therapy had longer (p = 0.0177) mean latencies [47.8 (SD 7.7) ms] compared to the CNTL group [35.4 (SD 9.3) ms]. The mean latencies for the right side Mult FW were not different (p = 0.49) for the LBP group at PRE therapy [40.0 (SD 6.4) ms] and the CNTL group [39.6 (SD 9.5) ms]. For Mult S there was no difference (p = 0.63) between the left and right side latencies. The mean reflex latencies for the LBP group at PRE therapy were 5.4 ms greater (p = 0.016) than that of the CNTL group. For ES S, the mean reflex latencies for the LBP group at PRE therapy were 6.7 ms greater (p = 0.001) than the CNTL group; however within the CNTL group the right side (p = 0.0053) was quicker [36.4 (SD 4.9) ms] than the left side [40.4 (SD 6.3) ms]. Reflex gains (Figure 5A) for Mult FW were lower (p = 0.030) for the LBP group at PRE therapy compared to the CNTL group. Reflex gains were not different between the two groups for Mult S (p = 0.071) or ES S (0.098).
Figure 9 Reflex responses in the individual low back muscles to the perturbations for
A) a CNTL subject; B) a LBP subject at PRE therapy; C) and the same LBP subject at POST
therapy
2.4.3 LBP group at PRE therapy VS. LBP group at POST therapy

There was no change in the reflex latencies for Mult FW (p = 0.915), Mult S (p = 0.561) and ES S (p = 0.549), between the two sessions at PRE and POST therapy (Figure 5B). The magnitude of the latencies remained in the order of an M2 response. However, there was an interaction (p = 0.0005) for the reflex latencies for Mult FW on the left side with 4 subjects demonstrating longer latencies and 5 subjects demonstrating shorter latencies at POST therapy compared to PRE therapy.
Figure 10 Comparing mean and standard deviations for reflex latencies and reflex gains for the low back muscles between A) CNTL group subjects and LBP subjects at PRE therapy; and between B) LBP subjects at PRE therapy and POST therapy.
A significant increase was observed in the reflex gains for Mult FW (p = <0.0001), Mult S (p = 0.0056) and ES S (p = 0.0018) at POST therapy for the LBP group. At the POST therapy session the reflex gains for Mult FW, Mult S and ES S were 5.4, 3.2 and 4.3 units higher than at PRE therapy, respectively.

2.5 Discussion

The findings of this study show that the subjects with LBP exhibited delayed latencies in the Mult S, Mult FW and ES S as compared to the healthy controls. These results follow similar trends observed in previous studies. Additionally, the reflex gains were lower for the Mult FW in the LBP subjects.

Thirteen LBP subjects were considered for the PRE PT analysis of which eight of them reported pain on the left side, perhaps explaining the delays observed on the left side for Mult FW in the LBP group at PRE therapy [47.8 (SD 7.7) ms] as compared to the CNTL group [35.4 (SD 9.3) ms]. Within the CNTL group, the right ES S was quicker than the left ES S. Handedness has been demonstrated to influence trunk muscle reflexes and may have contributed to the differences observed, but was not recorded in the study, nor was there a balanced group of left and right handed persons to test this hypothesis.

Trunk exercise programs such as the SSE program are directed towards recruitment of muscles and restoring the cross-sectional area, particularly in the
multifidus\textsuperscript{14} which is considered to be one of the important stabilizers for the intersegmental control of the spine.\textsuperscript{101} The subjects with LBP after completing the SSE program demonstrated stronger reflexes with higher reflex amplitudes with no changes in the reflex latencies. On the one hand, the reflexes became stronger and robust allowing for an effective response to an external perturbation, thus limiting excessive movement which might have led to excessive strain on the ligaments and hence injury. On the other hand, Franklin et al\textsuperscript{52, 102} observed that large reflex delays required smaller reflex amplitudes in order for the system to remain stable. However those authors only examined reflex delays greater than 60 ms. The need for smaller reflex gains in order to maintain stability became largely pronounced only as the delays approached 100 ms. The reflexes observed in the subjects with LBP in the present study were in the ranges of 39 to 57 ms which may or may not follow the above findings.

Penderson and colleagues\textsuperscript{103} trained healthy individuals to react to a variety of sudden trunk loading activities. The authors measured reflex latencies in the ES and trunk stopping time, before and after the training. The training did not alter the reflex latencies in the ES but did reduce the trunk stopping time by 7.8\% as compared to a control group which did not receive any training. The reduction in the trunk stopping time was achieved by a large burst seen in the ES response reflecting the increased reflex amplitude seen in the subjects with LBP post therapy in the present study. Training programs apart from general strengthening and flexibility exercises should also
perhaps incorporate reacting to sudden unexpected perturbations which might alter the reflex response to be stronger and more robust.

Limitations of this study include the small sample size and the number of subjects who did not complete the SSE program and POST test. These factors limit the ability to translate the findings to a larger population. Additionally, similar to the previous studies, M1 reflexes were not elicited. The possible benefits of the SSE program on the segmental reflexes (M1) might be worth exploring in future.

In conclusion, for the M2 response, longer latencies were observed for the subjects with LBP PRE therapy compared to CNTL subjects. A 10-week SSE rehabilitation program did not influence the latencies of the long latency reflexes but increased the reflex gain making them stronger and more robust.
Chapter 3 A stabilization exercise program improved seated postural control in patients with subacute, recurrent LBP

3.1 Abstract

Objective: This study evaluated the differences in seated postural control between healthy subjects and patients with subacute, recurrent low back pain (LBP), and investigated whether a 10-week physical therapy (PT) program incorporating specific stabilization exercises led to changes in those postural stability indices.

Summary of background data: Increased postural sway and greater trunk displacements have been observed in patients with LBP. One of the treatment options for LBP is the specific stabilization exercise program which is aimed at rehabilitating the neuromuscular control of the trunk muscles, and consequently hopes to improve postural stability. However, it is not known how and if this rehabilitation program improves seated postural control.
**Methods:** 20 subjects (10 subjects with subacute recurrent LBP, 10 healthy) aged between 18-55 years participated in the study. Force plate data were collected while subjects attempted to maintain sitting balance on a chair with an unstable platform. The LBP group was tested before (PRE) and after (POST) a 10-week physical therapy intervention program. The healthy control (CNTL) group was tested on one occasion. A stabilogram diffusion analysis was performed with the force plate data to discern differences in the postural stability indices between the two groups. Additionally, summary statistics such as total distance travelled and 95% ellipse area were also analyzed and compared between the two groups.

**Results:** The subjects with LBP at PRE therapy had larger trunk displacements and higher short term diffusion coefficients compared to the healthy subjects in the seated postural task. POST the 10-week stabilization exercise program, subjects with LBP demonstrated decreased trunk displacements and a reduction in their short term diffusion coefficients.

**Conclusions:** Subjects with subacute, recurrent LBP who completed a stabilization exercise program, which initially included specific exercises for the lumbar multifidus and the transverse abdominis, and then progressed to general trunk stabilization exercises, showed improved seated postural control. The POST therapy performance was consistent with that of matched healthy controls.
3.2 Introduction

Postural control is a complex task. Coordinated muscle forces are required at various joints to maintain stability. Three levels of control, the motor cortex with flexible, voluntary control over a variety of movements, the brain stem with the ability to rapidly integrate inputs for automatic responses, and the spinal cord with rapid reflex responses to local perturbations, all contribute to the regulation of muscle forces and maintenance of postural stability.

Postural control has typically been assessed in previous studies in standing as well as seated positions. Seated postural control is achieved with control over the hip and the trunk, unlike standing where stability can be achieved by coordinating movements at the level of the trunk, hip, knee, and at the ankle. Postural control in standing is usually assessed with a force platform by quantifying center of pressure (COP) trajectories in both anteroposterior and mediolateral directions. Traditionally, COP summary statistics such as total distance traveled and average ellipse area have been used to characterize postural sway. However, these measurements are unable to capture the dynamic characteristics of postural control such as energy transitions or acceleration between adjacent points. Understanding these time dependant characteristics of the COP would lead to a better understanding of the dynamics of neuromuscular control.
Collins and De luca\textsuperscript{113,114} proposed a new method, called stabilogram diffusion analysis (SDA), to analyze COP trajectories. The SDA is based on the notion of a fractional Brownian motion\textsuperscript{115,116} where the mean square displacements of the COP can be related to the corresponding time interval. Mean square displacements of the COP are calculated over different time intervals and plotted to create a stabilogram. Studies have demonstrated that the stabilogram can discriminate between the postural control of subjects based on age,\textsuperscript{114} visual feedback,\textsuperscript{117} and disease states.\textsuperscript{54} A typical SDA from a postural control task shows a two-part form, which are labeled as the short term and the long term region. One of the theories postulated for the two-part form is the existence of a two-part control mechanism; that is to say, there is an open loop control over the short time interval and a closed loop control over the longer time interval. The semantics can be argued since an open loop control moniker suggests that information regarding the state of the system is not relayed or fed back into the controller. However, sensory information related to muscle state,\textsuperscript{118} postural equilibrium,\textsuperscript{112} joint stability\textsuperscript{88} are all projected on to the CNS. Hence, rather than referring to the control mechanisms within the two regions as open loop and closed loop control, the control mechanism can be referred to as short term and long term corrections. The slopes of the two regions determine the short term and long term diffusion coefficients. The diffusion coefficients (mm\textsuperscript{2}/s) reflect the stochastic activity or COP energy within the designated regions.
A previous study comparing seated postural control between LBP subjects and healthy controls found differences in the short term diffusion coefficients. The authors used a chair developed by Cholewicki to assess trunk postural control which involved the subject sitting on a hemisphere. The base of support for the subjects could be reduced by reducing the diameters of the hemisphere. The LBP patients had higher short term diffusion coefficients suggesting greater stochastic activity within the short time region. The implication is that within the short time regions, the LBP patients tend to have greater COP displacements with greater accelerations. Additionally, increased seated postural sway and increased average distance travelled was also found in the LBP patients. An inability to confine and restrict COP displacements reflects an impaired postural control system. The authors also found that the subjects had delayed reflexes observed during a sudden force release task and suggested an association between impaired postural control and delayed onset times of the muscle reflexes. However, there are several other factors which could contribute to the impaired postural control observed in the LBP subjects. Pain, poor proprioception, muscle wasting, injured receptors/nerves, fatigue and reduced muscle endurance could all impair the ability in the individuals to precisely control movements.

Impaired trunk postural control is considered by many to be a sign of underlying instability of the spinal system. One of the theories for trunk postural control and potential instability of the spine has been the concept of a local vertebral segmental
control mechanism contributing to the overall global stability of the trunk. Local segmental stability refers to the control of the individual vertebral segments and the surrounding structures. Local instability of the vertebral segments is characterized by an abnormal response, in terms of large displacements of the vertebral segments, to normal physiological loads. Large displacements with greater accelerations would increase the strain and the stress on the structures of the spine leading to low back injury and/or may further exacerbate an existing condition. If the underlying neuromuscular impairments are not resolved, a continued state of instability may result, leading to a persistent, chronic state of LBP. It has been hypothesized that local segmental instability may contribute to an overall global instability which would be reflected in the observed increased postural sway of subjects with LBP.

Based on the same concept of the chair developed by Cholewicki, Kevin Granata at the Kevin P. Granata Musculoskeletal Biomechanics Lab at Virginia Tech designed the wobble chair apparatus which has springs underneath the chair which can be adjusted to change the base of support. Several studies have successfully used the wobble chair to evaluate trunk postural control in subjects without a history of LBP and was used in this study to compare and evaluate trunk postural control in the subjects with LBP and healthy subjects.

Among the many treatment options available, the specific stabilization exercise (SSE) is one of the therapeutic treatment options aimed at improving the local
segmental stability. The exercise program used in this study was developed based on
the concepts of the SSE. The initial part of the exercise program involved exercising the
abdominal and low back muscles with specific exercises for the multifidus and the
transverse abdominis. Training for recruitment of these muscles is augmented with
biofeedback via rehabilitation ultrasound imaging. The exercises are progressed to
recruit the rest of the trunk muscles via trunk stabilization exercises in supine, prone,
quadrupled, sitting, standing and, finally, during daily and sports activities. The exercise
program also includes general stretching of the trunk and leg extensors and aerobic
activity such as walking and swimming. The intensity and duration of the exercises are
increased with time as determined by the physical therapist. For this study, the SSE
program involved 10 visits, each lasting 45 minutes, distributed over a 10-week period.
In addition, the subjects were also instructed to carry out a home exercise program
during the 10-week program. The SSE has been shown to reduce the incidences of
recurrent LBP in a specific group of subacute LBP patients. However, it is not known
how the treatment affects postural control of the trunk.

The purpose of this study was twofold. The first objective was to determine if
the wobble chair could detect differences in postural control between patients with LBP
and healthy controls (CNTL) during performance of the seated postural control task. The
second objective was to determine if 10 weeks of physical therapy (PT) incorporating
the SSE program led to changes in the postural control parameters in patients with
subacute, recurrent LBP. It was hypothesized that the SSE intervention would improve postural control, leading to smaller postural sway and reduced diffusion coefficients in the patients with subacute, recurrent LBP like those observed in the healthy controls.

3.3 Methods

3.3.1 Subjects

10 subjects (5 female) with a primary complaint of subacute, recurrent LBP, and 10 subjects (5 female) with no history of significant LBP in the past 2 years participated in the study. Subjects were excluded if they had a prior history of lumbar surgery and presence of sciatica or medical conditions which might affect spinal control such as cauda equina syndrome, neurological disorders, fracture, cancer, infection, or systemic disease. Subjects with LBP were also included if the duration of pain in the current episode was less than or equal to 8 weeks and had experienced at least 1 separate episode in the past year which would have had seemingly resolved. The subjects in the control group were matched to the subjects in the group with LBP by their age, gender, height and weight. Informed consent was obtained from each subject following procedures approved by the Biomedical Institutional Review Board at the University.

3.3.2 Instrumentation

The wobble chair instrument (Figure 11) used to assess postural control consists of a seat mounted on a platform with a single central pivot point and 4 radially located springs. Placement of the springs relative to the pivot point allowed for adjustability of
the balancing task. Moving the springs closer to the center decreased the stability of
the chair and makes the task of maintaining a stable posture more challenging for the
neuromuscular system of the spine. The subjects sat with their arms crossed and a foot
rest integrated with the chair ensured that the subjects relied primarily on postural
adjustments of the trunk to steady the wobble chair. The wobble chair rested on a force
plate (Bertec, Worthington, OH) which allowed the 3-dimensional ground reaction
forces to be measured.

Figure 11 The experimental setup with the wobble chair resting on the force plate
with detailed views in 11A, 11B and 11C. 11D demonstrates a subject attempting to
maintain balance on the wobble chair
3.3.3 Experiment protocol

All subjects underwent a baseline physical examination by one of the investigators who was a physical therapist. Range of motion and demographic information were recorded. Subjects then completed a numerical rating scale for their best and worst pain in the past 24 hours with 0 being the lowest pain and 10 being the worst. The numerical rating scale has been shown to have good sensitivity and reliability.¹²⁴

The LBP group, using the same experimental protocol, was tested before (PRE) and after (POST) the 10-week SSE program. The CNTL group was tested on one occasion. Spring distance settings for the wobble chair were determined for each subject following previously established calibration procedures.¹²⁴ In brief, the subject sat on the wobble chair and ground reaction forces were recorded first with the chair positioned approximately 10 degrees in the backward direction (Θ₁) then with the chair positioned approximately 10 degrees in the forward direction (Θ₂). The moments generated in the backward (M₁) and forward direction (M₂) in the frontal plane were used to solve for the potential energy (Mgh) of the subject as shown in equation 1.

\[ Mgh = \frac{(M₁ - M₂)}{(-\sin (Θ₁)-\sin (Θ₂))} \]

A 100% stability setting was achieved when the moment generated by the springs was in static equilibrium with the mass and weight distribution of the subject. As the
stiffness constant (K) for the springs was known the linear distance from the pivot point for the springs at which the 100% stability setting was achieved was calculated using equation 2.

\[ L = \left( \frac{Mgh}{K} \right)^{1/2} \]  

(2)

The springs were then moved closer to the central pivot point for each of the three different stability levels (80%, 65% and 50%) used for the testing. During testing, subjects were instructed to maintain an upright posture with their arms crossed during three trials, each lasting 60 seconds, at each of the stability settings. All the subjects were tested in the same order starting from the 80% stability setting and progressing to the more difficult 65% and 50% settings. After each trial a rest period of 1 minute was provided to the subjects to avoid fatigue. A trial was considered a failed trial and repeated from the beginning if the subject could not maintain equilibrium and a contact occurred between the base of the chair and the platform. Two attempts at a trial were allowed. Two investigators visually monitored the task to detect a failed trial.

### 3.3.4 Data analysis

The COP trajectories were quantified using the 95% ellipse area (COP_EA), total distance travelled in the radial direction (RMSr) and the SDA. The 95% COP_EA (Figure 12) was representative of the area of the region of typical movement as illustrated by
Prieto et al.\textsuperscript{125} The SDA was performed for COP displacements in the radial direction \((r)\) accounting for the interactions between the anterior-posterior \((x)\) and the medial-lateral \((y)\) movements, \(<\Delta r^2> = <\Delta x^2> + <\Delta y^2>\).
Figure 12 COP trajectories for a CNTL group subject at the 80% (A) and 50% (B) stability level are shown; in addition the ellipse area at the 50% stability level (B) is also demonstrated. The corresponding SDA calculated for the CNTL group subject at the 80% (C) and the 50% (D) are also shown. The dashed lines delineate the short term and long term regions.
For the SDA, a stabilogram was created for each trial by plotting averaged squared distances traveled by the signal $<\Delta \text{COP}^2>$ for increasing time intervals $\Delta t$, starting at 0.01s. The COPx versus COPy plot and the corresponding stabilogram are shown in Figure 12 for a CNTL group subject at the 80% and 50% stability level. Two distinct regions are identified and a least squares method was used to fit two straight lines in the two regions (Figure 13). The slopes of the two regions form the short term and long term diffusion coefficients ($D_S$, $D_L$), described in equation 3. The transition point between the two regions was defined as the critical point. The Hurst exponents $H_S$ and $H_L$ were computed from the log-log slope of $<\Delta \text{COP}^2>$ versus $\Delta t$ described in equation 4.

\[
<\Delta \text{COP}^2> = 2D_{(S,L)} \Delta t \quad (3)
\]

\[
<\Delta \text{COP}^2> \sim \Delta t^{2H(S,L)} \quad (4)
\]

Hurst exponent values greater than 0.5, indicate a persistent behavior, i.e. if the COP was moving in a particular direction, it will likely, continue to move in the same direction.\textsuperscript{126, 127} Exponent values less than 0.5 indicate an antipersistent behavior, i.e. if the COP was moving in a particular direction for a time $t_0$, it would be likely to move in an opposite direction for $t>t_0$. An exponent value of 0.5 indicates a pure random behavior.
Brownian motion, i.e. the increments in the COP displacement are statistically independent.

Figure 13 A SDA plot for a typical LBP group subject at PRE therapy for the 50% stability level. The solid lines show the computed fits from which the slope was calculated.
3.3.5 Statistical analysis

Subject characteristics (age, height, and weight) were compared between groups using an independent t test (Excel 2007, Microsoft, Redmond, WA). The average of the best and worst pain rating scores were compared PRE and POST in the group with LBP using a paired samples t test. A p <= 0.05 was considered statistically significant.

A mixed effects regression modeling framework (SAS, v9.1, SAS Institute Inc., Cary, NC) was used to model the COP_EA, RMSr, Ds, DL, HS and Hr. A random intercept for each subject was included in order to separate within-subjects and between-subjects variability. Empirical “sandwich” estimators of the variance-covariance matrix for the fixed effects parameters (e.g. group, Level, PRE/POST) were used to alleviate the impact of any departures from the defined covariance structure. Data are presented as means and standard deviations.
3.4 Results

3.4.1 Subjects

The subjects in the LBP and the CNTL groups were similar in age, height and weight (Table 2). The average pain ratings improved ($P= 0.0039$) for the LBP group POST therapy and the changes were clinically important ($\Delta$ in pain $\geq 1.5$) per Childs and colleagues.\(^97\)

<table>
<thead>
<tr>
<th></th>
<th>CNTL n=10 (5 female)</th>
<th>LBP n =10 (5 female)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>35 (8.3)</td>
<td>32.6 (11.1)</td>
<td>0.28</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.75 (0.09)</td>
<td>1.74 (0.13)</td>
<td>0.49</td>
</tr>
<tr>
<td>Weight(kg)</td>
<td>79 (22.3)</td>
<td>84.2 (19.9)</td>
<td>0.29</td>
</tr>
<tr>
<td>BMI(Kg/m2)</td>
<td>26.1 (7.1)</td>
<td>27.3 (4.54)</td>
<td>0.30</td>
</tr>
<tr>
<td>Pain Rating</td>
<td>N/A</td>
<td>PRE: 4.03 (SD 1.19)</td>
<td>0.0039</td>
</tr>
<tr>
<td></td>
<td></td>
<td>POST: 2.27 (SD 1.07)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Subject characteristics for the seated postural sway experiment
3.4.2 LBP group at PRE therapy VS. CNTL group

At the 50% stability level, the COP_EA (p = 0.015) and the RMSr (p = 0.011) were greater for the LBP group at PRE therapy compared to the CNTL group (Figure 14A and 14B, Table 3). The short term diffusion coefficients (D_s) were also greater at the 50% stability level for the LBP group at PRE therapy compared to the CNTL group indicating greater trunk displacements for the subjects with LBP during the short term region (Figure 14C, Table 3). The short term scaling exponents (H_s) were not different between the two groups (p = 0.235), however the values were greater than 0.5 for both the groups indicating a persistent behavior. The long term diffusion coefficients (D_L) and the long term scaling exponent (H_L) were not different (p = 0.058, p=0.633) between the two groups. The values for the long term scaling exponents were less than 0.5 for both the groups indicating an antipersistent behavior. The level of difficulty on the wobble chair was a significant factor with the 50% stability level different from both the 65% and 80% stability levels by all measures.
Figure 14 Comparing postural stability indices between the CNTL group, the LBP group at PRE therapy, and the LBP group at POST therapy. The symbol * indicates that the values for the LBP group at PRE therapy were significantly different from the CNTL group. The symbol ¥ indicates that the values for the LBP group at POST therapy were significantly different from the values at the PRE therapy session.
3.4.3 LBP group at PRE therapy VS. LBP group at POST therapy

At the 50% stability level, the COP EA (p = 0.018) and RMSr (p = 0.014) decreased for the LBP group at the POST therapy session compared to at the PRE therapy session (Figure 14A and 14B, Table 3). Similarly, the short term diffusion coefficients (D_s) also decreased (p=0.035) at the POST therapy session for the LBP group (Figure 14C, Table 3). The long term diffusion coefficients (D_L) were not different (p=0.087) between the two sessions. The short term scaling exponents (H_s) were not different (p=0.163) between the two sessions and the values remained greater than 0.5 indicating the persistent behavior. The long term scaling exponents (H_L) were also not different (p = 0.221) between the two sessions and the values remained less than 0.5 indicating an antipersistent behavior.

3.4.4 CNTL group VS. LBP group at POST therapy

The COP EA (p = 0.198) and RMSr (p = 0.214) were similar for the LBP group at the POST therapy session and the CNTL group (Figure 14A and 14B, Table 3). Similarly, the short term diffusion coefficients (D_s) and the long term diffusion coefficients (D_L) were similar (p=0.675, p=0.436) between the CNTL group and the LBP group at the POST therapy session. The short term (H_s) and long term scaling exponents (H_L) were also not different (p = 0.221, p=0.087) between the CNTL group and the LBP group at POST therapy session, with the short term scaling exponents greater than 0.5 and the long term scaling exponents less than 0.5 for both.
### Table 3 Summary statistics and SDA results for the seated postural task for the CNTL group, and the LBP group at PRE and POST therapy.

<table>
<thead>
<tr>
<th></th>
<th>STABILITY LEVEL</th>
<th>CNTL GROUP</th>
<th>PRE THERAPY LBP</th>
<th>POST THERAPY LBP</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>COP_EA (cm²)</strong></td>
<td>Level 80</td>
<td>4.70(SD 2.5)</td>
<td>12.98(SD 17.7)</td>
<td>4.63(SD 2.8)</td>
</tr>
<tr>
<td></td>
<td>Level 65</td>
<td>5.26(SD 2.9)</td>
<td>19.46(SD 27.0)</td>
<td>5.64(SD 3.9)</td>
</tr>
<tr>
<td></td>
<td>Level 50</td>
<td>10.39(SD 3.6)</td>
<td>36.91(SD 33.1)</td>
<td>10.66(SD 8.1)</td>
</tr>
<tr>
<td><strong>RMSr(mm)</strong></td>
<td>Level 80</td>
<td>6.28(SD 2.17)</td>
<td>10.87(SD 7.28)</td>
<td>7.07(SD 1.81)</td>
</tr>
<tr>
<td></td>
<td>Level 65</td>
<td>7.37(SD 1.95)</td>
<td>13.09(SD 8.66)</td>
<td>7.52(SD 2.41)</td>
</tr>
<tr>
<td></td>
<td>Level 50</td>
<td>10.5(SD 2.03)</td>
<td>16.67(SD 13.5)</td>
<td>10.17(SD 3.66)</td>
</tr>
<tr>
<td><strong>D₃ (mm²/s)</strong></td>
<td>Level 80</td>
<td>0.039(SD 0.02)</td>
<td>0.143(SD 0.11)</td>
<td>0.054(SD 0.05)</td>
</tr>
<tr>
<td></td>
<td>Level 65</td>
<td>0.054(SD 0.03)</td>
<td>0.180(SD 0.19)</td>
<td>0.065(SD 0.06)</td>
</tr>
<tr>
<td></td>
<td>Level 50</td>
<td>0.116(SD 0.06)</td>
<td>0.380(SD 0.23)</td>
<td>0.150(SD 0.12)</td>
</tr>
<tr>
<td><strong>D₅ (mm²/s)</strong></td>
<td>Level 80</td>
<td>1.26(SD 1.21)</td>
<td>3.38(SD 1.08)</td>
<td>1.43(SD 1.11)</td>
</tr>
<tr>
<td></td>
<td>Level 65</td>
<td>1.29(SD 1.04)</td>
<td>5.35(SD 1.02)</td>
<td>1.24(SD 1.69)</td>
</tr>
<tr>
<td></td>
<td>Level 50</td>
<td>3.79(SD 2.13)</td>
<td>6.82(SD 1.08)</td>
<td>3.96(SD 1.93)</td>
</tr>
<tr>
<td><strong>H₅</strong></td>
<td>Level 80</td>
<td>0.51(SD 0.17)</td>
<td>0.56(SD 0.15)</td>
<td>0.53(SD 0.09)</td>
</tr>
<tr>
<td></td>
<td>Level 65</td>
<td>0.56(SD 0.10)</td>
<td>0.61(SD 0.14)</td>
<td>0.55(SD 0.07)</td>
</tr>
<tr>
<td></td>
<td>Level 50</td>
<td>0.62(SD 0.06)</td>
<td>0.64(SD 0.12)</td>
<td>0.61(SD 0.06)</td>
</tr>
<tr>
<td><strong>H₆</strong></td>
<td>Level 80</td>
<td>0.078(SD 0.05)</td>
<td>0.065(SD 0.06)</td>
<td>0.057(SD 0.05)</td>
</tr>
<tr>
<td></td>
<td>Level 65</td>
<td>0.063(SD 0.03)</td>
<td>0.071(SD 0.03)</td>
<td>0.060(SD 0.04)</td>
</tr>
<tr>
<td></td>
<td>Level 50</td>
<td>0.081(SD 0.02)</td>
<td>0.067(SD 0.05)</td>
<td>0.070(SD 0.04)</td>
</tr>
</tbody>
</table>
3.5 Discussion

This study found differences in seated postural control between subjects with subacute recurrent LBP and a matched group of healthy CNTL’s. The subjects with LBP at PRE therapy had larger COP ellipse areas and higher diffusion coefficients in the short term region compared to the healthy subjects. The implication of these findings is that within the short term region the patients with LBP had larger trunk displacements at greater velocities compared to the healthy subjects and suggest impairments in seated postural control.

These results are consistent with those obtained by others during a similar seated postural task. Radebold et al\textsuperscript{54} found that the subjects with LBP had greater COP ellipse areas and greater short term diffusion coefficients compared to healthy subjects. The authors in the study also observed that the subjects with LBP not only had increased COP displacements, but also delayed trunk muscle reflexes observed during a seated sudden force-release task. It has been suggested that an association between impaired reflex responses and impaired postural stability may exist.\textsuperscript{54}

Reflex responses are the first active responses to disturbances in the postural control whether brought on by external or internal perturbations.\textsuperscript{48,49} Reflex responses are characterized by the onset time and by the amplitude of the response.\textsuperscript{52,91} The subjects of this current study also underwent a test in which the trunk muscle reflexes were elicited via sudden perturbations to the trunk. The reflexes were quantified in
terms of their timing and amplitude (gain). The sudden perturbation experiment is
described in detail in the previous chapter. The subjects with LBP had delayed and
dampened reflexes in the lumbar multifidus and the erector spinae muscles compared
to the healthy subjects. Longer delays in the onset time of the reflex responses as well
as decreased amplitudes in the reflex responses could theoretically impair the ability of
an individual to rapidly and appropriately correct for changes in the equilibrium
conditions. The multifidus appears to play an important role in trunk postural
control. In a study that quantified the contribution of trunk extensor muscles to
stability, Wilke et al showed that when compared with other muscles in close
proximity to the L4-L5, the multifidus muscle contributed two thirds of the increased
stiffness imparted by contraction of the muscles. This evidence has led researchers to
believe that the lumbar multifidus may be an important local stabilizer of the vertebral
segments and any deficits in its activity may compromise the local segmental stability.
In theory loss of local segmental stability could contribute to larger trunk displacements
during performance on the wobble chair and may explain the increased postural sway
observed in the subjects with LBP. Apart from impaired reflexes, other factors such as
pain, reduced muscle strength, loss of anticipatory control, impaired proprioception
may also hamper the ability of the subjects with LBP to generate adequate muscle
forces in order to restrict trunk displacements. The increased postural sway and loss of
stability observed in the subjects with LBP at PRE therapy is indicative of deficits in the
neuromuscular control system and has the potential to exacerbate existing conditions or injuries in the subjects with LBP. Hence, perhaps the objective of an effective exercise program may be to address these deficits and to improve postural stability.

After the 10-week stabilization exercise program, the subjects with LBP at POST therapy had reduced their overall displacements and also saw a reduction in their diffusion coefficients in the short term region, indicating that the subjects with LBP were now able to limit or restrict their overall trunk displacements.

The 10-week stabilization therapy program aimed to improve local segmental stability and global postural control. The stabilization exercise program included motor control exercises in addition to general strengthening and flexibility exercises. Part of the motor control paradigm was to train the individuals to effectively recruit the lumbar multifidus and the transverse abdominis. At POST therapy the reflex responses in the lumbar multifidus and the erector spinae became stronger and more robust (Chapter 2). Franklin and Granata\textsuperscript{52} developed a dynamic spine model to investigate the role of both the reflex gain and reflex delay in spinal stability and demonstrated that a spine with reduced intrinsic stiffness could be stabilized by a proportionally increased reflex gain. The increase in the reflex amplitudes observed in the trunk muscles in the subjects with LBP could theoretically have contributed to improved control over the local segment. This, in addition to perhaps increased strength and improved recruitment of the trunk muscles could explain the observed improvements in the postural control parameters in
the subjects with LBP at POST therapy. On comparing the LBP group at POST therapy to
the CNTL group, no differences were observed in either the summary statistics of overall
displacements or in the SDA, indicating that the improvements in the postural stability
indices of the subjects with LBP after therapy were in the direction of the values for the
healthy CNTL group. This finding may represent improvements in the neuromuscular
control of the spine in the LBP group following the 10-week, SSE intervention. Further
study is needed to determine if the improvements observed are unique to the SSE
intervention or are a consequence of reduced pain, improved mobility, increased
strength, or other factors associated with recovery from an episode of LBP episode.
Several limitations in the study design are acknowledged. First, there was no follow up
test conducted for the subjects with LBP to determine if the improvements in their
postural stability indices were retained over longer periods of time. A future study
which includes long term follow up measurements may prove beneficial as it may shed
light on whether this exercise program initiates lasting changes in postural stability and
the neuromuscular control of the spine. Second, the study design also did not include a
CNTL group which received training or an untrained LBP group which was tested after 10
weeks. The lack of these two groups makes it difficult to conclusively attribute the
changes seen in the LBP group at POST therapy purely as the effect of the therapeutic
program. Third, the small sample size in the study might limit the ability to translate the
findings to a larger population. Finally, another limitation of the study lies with the
design of the wobble chair, which only tests the ability of the subject to balance in the absence of an external perturbation. In reality, subjects may either anticipate or react to applied perturbations, possibly creating different loading conditions and motor control strategies than were observed in this study. Understanding these mechanisms might provide further clues of understanding the nuanced differences between subjects with LBP and healthy subjects during postural stability tasks.

In conclusion, subjects with LBP at PRE therapy demonstrated increased postural sway on the wobble chair compared to CNTL subjects and a 10-week stabilization exercise program decreased the postural sway in subjects with LBP suggesting improved postural control. Clinically, these results suggest that exercise programs which include motor control exercises for the lumbar multifidus and the transverse abdominis, apart from general strengthening and conditioning exercises, may benefit the subjects with LBP by improving their postural control of the trunk.
Chapter 4 Effect of a 10-week stabilization exercise program on the muscle activation levels and neuromuscular patterns in patients with subacute, recurrent LBP during a seated postural task

Abstract

Objective: The purpose of this study was twofold: 1) To evaluate the differences in the muscle activation levels and neuromuscular control strategies during a trunk postural control task between healthy subjects and patients with sub acute, recurrent LBP, and 2) To determine if a 10 week physical therapy (PT) program incorporating specific stabilization exercises led to changes in the muscle activation levels in the patients with sub acute, recurrent LBP.

Summary of background data: Altered trunk muscle recruitment patterns and impaired postural control of the lumbar spine have been observed in patients with LBP. One of the treatment options for LBP is the specific stabilization exercise program which is aimed at rehabilitating the neuromuscular control of the trunk muscles. It is not known how and if this rehabilitation program alters the neuromuscular control factors for the trunk.
**Methods:** 20 subjects (10 subjects with sub acute recurrent LBP, 10 healthy) aged between 18-55 years participated in the study. Surface electromyography signals were recorded bilaterally from the L5 multifidus, L3 erector spinae, internal oblique and external oblique, while subjects attempted to maintain sitting balance on a chair with an unstable platform. The LBP group was tested before (PRE) and after (POST) a 10 week physical therapy intervention program. The healthy control (CNTL) group was tested on one occasion. Muscle activation levels were compared between the groups across their ipsilateral and contralateral side. The ipsilateral side was the side which the subjects with LBP identified as their more painful side, and the contralateral side was the side opposite to the painful side. For the CNTL group the activity across both their sides was averaged and considered for both the ipsilateral and contralateral comparisons.

**Results:** The LBP group at PRE therapy had increased levels of flexor activity in their ipsilateral side during the seated postural task compared to the CNTL group subjects. The LBP group at POST therapy had decreased levels of flexor activity in their ipsilateral and contralateral sides during the seated postural task compared to their PRE therapy session. Compared to the CNTL group, the LBP group at POST therapy had significantly higher activity levels in both their ipsilateral and contralateral extensors.

**Conclusions:** A 10-week stabilization exercise program influenced the muscle activation patterns from a compensatory flexor activity to a higher extensor activity in subjects with sub acute recurrent LBP relative to the CNTL group.
Keywords: LBP, neuromuscular postural control, lumbar stabilization exercises

4.2 Introduction

In 2002, approximately one quarter of U.S. adults reported having LBP lasting at least one whole day and 7.6% reported at least 1 episode of severe acute LBP within a year period.\(^2\) LBP may be associated with spinal instability, which is characterized by an abnormal response with increased trunk displacements to normal physiological loads. The abnormal response in the motion segments can result in excessive strains or deformations in the spinal structures causing injuries.\(^{74,129}\) McGill et al\(^{12}\) suggests that these excessive deformations may result in injuries to the neurological structures and may be a source of pain. Spinal stability is acted upon by three subsystems as defined by Panjabi.\(^5\) One is the passive subsystem which consists of the spinal column with the vertebrae, disks and ligaments which provides passive stiffness for stabilizing the spine, the second is the active subsystem which consists of steady state muscle activity and the third is the neural control subsystem which regulates active and voluntary responses. The passive components alone cannot provide sufficient stability to the spine. For example, the spine devoid of muscles buckles under a 90 N load.\(^{20}\) The active components on the other hand play a critical role in stabilizing the spine. The active components provide stability by providing active stiffness while modulating force production through a combination of anticipatory, reflex and voluntary control.\(^{130,131}\)
Neuromuscular impairments in the active components have been observed in patients with LBP. These impairments include alterations in the feed forward timing, \(^{31, 72}\) delayed reflex responses, \(^{53, 132}\) increased levels of agonist/antagonist co-contraction \(^{60, 133}\) and reduced force in the trunk muscles. \(^{94, 134}\) In addition, localized dysfunction of the multifidus muscle ipsilateral to the location of pain in terms of muscle wasting, and fat infiltration has been observed in subjects with LBP. Van Dieen et al.\(^ {74}\) propose that persons with LBP utilize the increased co-contraction and altered recruitment of muscles as a learned, compensatory strategy. This greater reliance on the active control subsystem by increasing co-contraction would stiffen the spine; and, in the face of unexpected perturbations, may prevent excessive lengthening of muscle, ligaments, and the posterior aspect of the annulus of the intervertebral disk. Van Dieen et al.\(^ {74}\) have proposed 3 reasons for the adaptive strategy of increased active control to stabilize the spine in persons with LBP. Injuries to the ligaments or disk reduce the passive stiffness of the spine. Muscle force is reduced, which limits the ability to protect the spine from perturbations. The sensorimotor system is impaired which limits or delays protective feedforward and feedback control. These changes in control increase the potential for instability in the spine and make the low back more vulnerable to injury from events that may have otherwise had innocuous consequences.

One of the approaches for treatment for LBP includes exercises aimed at rehabilitating the motor control of trunk muscles. The specific stabilization exercise
(SSE) program is one such intervention. \(^{14}\) The initial part of the SSE program involves exercising the abdominal and low back muscles with specific exercises for the multifidus and the transverse abdominis. Training for recruitment of these muscles is augmented with biofeedback via rehabilitation ultrasound imaging. The exercises are progressed to recruit the rest of the trunk muscles. Patients also perform trunk and lower extremity stretching exercises and aerobic activity such as walking and swimming. The intensity and duration of the exercises are increased with time as determined by the physical therapist. The exercise program adapted for the present research study involved 10 visits each lasting 45 minutes distributed over a 10 week period. In addition, the subjects also carried out a home exercise program for 30 min/day during the 10-week program. \(^{14}\) The SSE program was effective in reducing pain, restoring muscle cross sectional area, and reduced the risk of recurrence of LBP in a select sample of patients with first time acute LBP. \(^{82}\) It is not known how and if the SSE protocol alters the neuromuscular control of the trunk muscles which could contribute to improved spinal stability in subjects with recurrent LBP. One of the ways to assess the neuromuscular control of the trunk is to test the subject’s equilibrium control during a task involving sitting on a chair with an unstable platform or “wobble chair”. \(^{76,135,136}\) As demonstrated in Chapter 3 of this dissertation, measures of postural control in this apparatus were able to distinguish between LBP and healthy controls (CNTL), and also distinguished the effects of the therapeutic exercise program on patients with LBP. In theory, differences
in muscle recruitment patterns should accompany these differences in performance on
the wobble chair, and the effects of weaknesses and strengthening exercises should be
evident in the altered muscle recruitment patterns.

The purpose of this study was twofold. The first objective was to determine if
there were differences in muscle activation levels patients with LBP and healthy controls
(CNTL) during performance of a postural control task using a wobble chair. The second
objective was to determine if a 10-week physical therapy (PT) program incorporating
specific stabilization exercises led to changes in the trunk muscle activations in patients
with sub acute recurrent LBP. It was hypothesized that 1) the muscle activation levels
for the painful and non painful sides in the LBP group would be different compared to
the muscle activation levels in the CNTL group, and 2) that a 10-week trunk stabilization
exercise program aimed at altering the neuromuscular control would lead to changes in
the muscle activation levels in the LBP group.

4.3 Methods

4.3.1 Subjects

Ten subjects (five female) with a primary complaint of sub acute, recurrent LBP,
and ten subjects (five female) with no history of significant LBP in the past 2 years
participated in the study. Subjects were excluded if they had a prior history of lumbar
surgery and presence of sciatica or medical conditions which might affect spinal control such as cauda equina syndrome, neurological disorders, fracture, cancer, infection, or systemic disease. Subjects in LBP were included if the duration of pain in the current episode was less than or equal to 4 weeks and had experienced at least 1 separate episode in the past year which would have had seemingly resolved. The subjects in the control group were matched to the subjects in the group with LBP by their age, gender, height and weight. Informed consent was obtained from each subject following procedures approved by the Biomedical Institutional Review Board at the University.

4.3.2 Instrumentation

The wobble chair apparatus (Figure 15), designed by Kevin Granata at the Kevin P. Granata Musculoskeletal Biomechanics Lab at Virginia Tech, was modeled after a chair developed by Cholewicki. The wobble chair has been used in several studies to evaluate trunk postural control in subjects without a history of LBP. The wobble chair consists of a seat mounted on a platform with a single central pivot point and 4 radially located springs. Placement of the springs relative to the pivot point allowed for adjustability of the balancing task. Moving the springs closer to the center decreased the stability of the chair and makes the task of maintain a stable posture more challenging for the neuromuscular system of the spine. A foot rest integrated with the chair ensured that the subjects relied primarily on postural adjustments of the
trunk, achieved by recruiting the abdominal and low back muscles. The wobble chair rested on a force plate (Bertec, Worthington, OH) which allowed the 3-dimensional ground reaction forces to be measured.

Figure 15 Experimental setup for the seated postural task with different views of the wobble chair in A, B, and C. Figure 15D demonstrates a subject attempting to balance on the wobble chair.
Trunk muscle activity was recorded bilaterally from the L5 multifidus, L3 erector spinae, internal oblique and external oblique muscles using surface electromyography (EMG) (Motion Labs, Baton Rouge, LA) and sampled at 2000 Hz. The location of the electrode placements are listed below

- **Internal Oblique**: Over the area limited by the inguinal ligament, the lateral border of the rectus abdominis sheath, and a line joining the two anterior superior iliac spines.\(^{60}\)

- **External Oblique**: Just below the ribcage on a line between the most inferior point of the costal margin and the contra lateral pubic tubercle.\(^{60}\)

- **Lumbar erector spinae**: 3 cm from midline at L3 spinous process \(^{98}\)

- **Multifidus**: 3 cm from midline at L5-S1 spinous process.\(^{22}\)

### 4.3.3 Procedures

The LBP group, using the same experimental protocol, was tested before (PRE) and after (POST) the 10-week physical therapy intervention program. The CNTL group was tested on one occasion. All subjects underwent a baseline examination by one of the investigators, who was a physical therapist, and their range of motion and demographic information were recorded. Subjects were then asked to fill out a
numerical rating scale for their best and worst pain in the past 24 hours, with 0 being the lowest pain and 10 being the worst. The numerical rating scale has been shown to have good sensitivity and reliability. Subjects were also asked to identify the side at which their pain was more dominant. This was done to identify ipsilateral and contralateral sides. For normalization of the EMG signals, subjects performed the Biering-Sorensen test for the back extensors and a maximum voluntary contraction for the abdominal muscles.

Spring distance settings for the wobble chair were determined for each subject following previously established calibration procedures. In brief, the subject sat on the wobble chair and the chair was then positioned approximately 10 degrees in the backward direction ($\Theta_1$) and ground reaction forces were collected, then approximately 10 degrees in the forward direction ($\Theta_2$) and force plate data was collected again. The moments generated in the backward ($M_1$) and forward direction ($M_2$) in the frontal plane were used to solve for the potential energy ($Mgh$) of the subject as shown in equation 1.

$$Mgh = (M_1 - M_2)/(-\sin(\Theta_1) - \sin(\Theta_2))$$ (1)

A 100% stability setting was achieved when the moment generated by the springs was in static equilibrium with the mass and weight distribution of the subject. As the stiffness constant ($K$) for the springs was known the linear distance from the pivot point
for the springs at which the 100% stability setting was achieved was calculated using equation 2.

\[ L = (Mgh/K)^{1/2} \]  

(2)

The springs were then moved closer to the central pivot point for each of the three different stability levels (80%, 65% and 50%) used for the testing. Subjects were instructed to maintain an upright posture with their arms crossed during three trials, each lasting 60 seconds, at each of the stability settings. All the subjects were tested in the same order starting from the 80% stability setting and progressing to the more difficult 65% and the 50% settings. After each trial a rest period of 1 minute was provided to the subjects to avoid fatigue. A trial was considered a failed trial and repeated from the beginning if the subject could not maintain equilibrium and a contact occurred between the base of the chair and the platform. Two investigators visually monitored the task to detect a failed trial.

4.3.4 Data Analysis

The EMG signals were collected and processed using Datapac (RunTech Inc., Mission Viejo, CA). The EMG signals were band pass filtered between 10-450 Hz using a 4th order, zero lag Butterworth filter, and then low pass filtered at 100Hz using a 4th order zero lag Butterworth filter. The EMG signals were then rectified and smoothed using a RMS window of 10 msec.
Stable and unstable regions (Figure 20) for the 50% stability level were identified from the force plate data, and a 3 second window was chosen within those regions. The stable regions were identified as the regions of quiet period from the force plate data. The unstable regions were identified as the regions where the COP activity was 3 standard deviations higher than the activity measured during the quiet stable regions. The integrated EMG data was calculated within those regions for each of the muscles measured and then normalized. A log transformation of the normalized EMG was then undertaken to reduce the effects of between subjects variability. The individual muscle activities computed were then grouped for the LBP group at both PRE and POST sessions based on their ipsilateral and contralateral locations. The individual muscle activities were averaged over the two sides for the CNTL group to give a single value for each subject, which was then compared with the ipsilateral and contralateral muscle activities from the LBP group. In addition, for the LBP group, the ipsilateral multifidus and erector spinae were grouped to form the ipsilateral extensors while the contralateral multifidus and erector spinae were grouped to form the contralateral extensors. The internal oblique and external oblique were grouped similarly to form the ipsilateral and contralateral flexors. These grouped muscles were then compared with the group muscle activities averaged over both sides for the CNTL group. Additionally, total muscle activity was also compared between the groups, to compare overall muscle
activation levels; all the extensors and flexors were combined to obtain total muscle activity and compared across groups and sessions.

4.3.5 Statistical Analysis

Subject characteristics (age, height, and weight) were compared between groups using an independent one tailed t test (Excel 2007, Microsoft, Redmond, WA). The average of the best and worst pain rating scores was compared PRE and POST in the group with LBP using a paired samples one tailed t test. A p <= 0.05 was considered statistically significant.

A mixed effects regression modeling framework (SAS, v9.1, SAS Institute Inc., Cary, NC) was used to model the muscle activation data for the individual muscles and the grouped muscles. A random intercept for each subject was included in order to separate within-subjects and between-subjects variability. Empirical “sandwich” estimators of the variance-covariance matrix for the fixed effects parameters (e.g. group, PRE/POST) were used to alleviate the impact of any departures from the defined covariance structure.

4.4 Results

4.4.1 Subjects:

The subjects in the LBP and the CNTL group were similar in age, height and weight. The mean and standard deviations are listed for the subject characteristics in Table 4.
There were no differences in terms of height, weight and age between the CNTL group and the LBP group. The average pain ratings improved (P= 0.0039) for the LBP group POST therapy and were clinically important (Δ in pain >= 1.5) per Childs and colleagues.97

<table>
<thead>
<tr>
<th></th>
<th>CNTL n=10 (5 female)</th>
<th>LBP n =10 (5 female)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yrs)</td>
<td>35 (8.3)</td>
<td>32.6 (11.1)</td>
<td>0.28</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.75 (.09)</td>
<td>1.74 (.13)</td>
<td>0.49</td>
</tr>
<tr>
<td>Weight(kg)</td>
<td>79 (22.3)</td>
<td>84.2 (19.9)</td>
<td>0.29</td>
</tr>
<tr>
<td>BMI(Kg/m2)</td>
<td>26.1 (7.1)</td>
<td>27.3 (4.54)</td>
<td>0.30</td>
</tr>
<tr>
<td>Pain Rating</td>
<td>N/A</td>
<td>PRE: 4.03 (SD 1.19)</td>
<td>P =0.0039</td>
</tr>
<tr>
<td></td>
<td></td>
<td>POST: 2.27 (SD 1.07)</td>
<td></td>
</tr>
</tbody>
</table>

Table 4 Subject characteristics for the seated postural sway experiment
4.4.2 EMG Analysis:

The EMG activation patterns for the 8 different muscles along with the COP displacements in the X and Y direction are shown in Figure 16 for a subject from the CNTL group, Figure 17 for a subject from the LBP group at PRE therapy, and Figure 18 for the same LBP group subject at POST therapy.

Within the LBP PRE therapy group subjects, 8 subjects identified their left side as their painful side, one subject identified their right side as their painful side while one identified both their right and left sides as being their painful side. Hence, for the 8 subjects who had pain on their left side, the left side became the ipsilateral side, while their right side became the contralateral side. In contrast for the one subject with pain on his/her right side, the right side became the ipsilateral side and the left side became the contralateral side. One subject reported pain on both sides, and hence was not considered for this analysis. For the CNTL group subjects, the muscle activity was averaged over both the left and right sides and compared with the LBP group.
Figure 16 EMG activity and COP displacements for a 30 second window from a representative CNTL group subject while the subject balanced at the 50% stability level.
Figure 17 EMG activity and COP displacements for a 30 second window from a representative LBP group subject at PRE therapy while the subject balanced at the 50% stability level.
Figure 18 EMG activity and COP displacements for a 30 second window from a representative LBP group subject at POST therapy subject while the subject balanced at the 50% stability level. Stable and Unstable periods are labeled.
4.4.3 Individual muscle activation levels during stable periods

a) LBP group at PRE therapy vs. CNTL group:
Within the LBP group at PRE therapy, there was no difference between the ipsilateral and contralateral activation levels of MULT (p = 0.486), ES (p =0.281), EO (p =0.605) or IO (p = 0.124). Comparing the CNTL group and the LBP group at PRE therapy, group (Figure 19) was not significant for either the ipsilateral activation levels of MULT (p = 0.273), ES (p =0.404), EO (p =0.336) or IO (p = 0.190) or the contralateral MULT (p = 0.262), ES (p =0.252), EO (p =0.386) or IO (p = 0.233).

b) LBP Group at PRE therapy vs. LBP group at POST therapy:
Within the LBP group at POST therapy, there was no difference between the ipsilateral and contralateral activation levels of MULT (p = 0.447), ES (p =0.465), EO (p =0.126) or IO (p = 0.085). Comparing the LBP group at POST therapy group and the LBP group at PRE therapy, group was not significant (Figure 19) for either the ipsilateral activation levels of MULT (p = 0.177), ES (p =0.296), EO (p =0.163) or IO (p = 0.072) or the contralateral activation levels of MULT (p = 0.186), ES (p =0.498), EO (p =0.134) or IO (p = 0.156).

c) CNTL group vs. LBP group at POST therapy:
Comparing the LBP group at POST therapy and the CNTL group, group was not significant (Figure 19) for either the ipsilateral activation levels of MULT (p = 0.148), ES
(p = 0.302), EO (p = 0.291) or IO (p = 0.390) or the contralateral activation levels of MULT (p = 0.151), ES (p = 0.197), EO (p = 0.243) or IO (p = 0.252).
Figure 19 Comparing ipsilateral and contralateral muscle activations during the stable periods for the CNTL group, the LBP group at PRE and POST therapy.
4.4.4 Individual muscle activation levels during unstable periods

a) LBP group at PRE therapy vs. CNTL group:

Within the LBP group at PRE therapy, there was no difference between the ipsilateral and contralateral activation levels of MULT (p = 0.432), ES (p = 0.561), EO (p = 0.395) or IO (p = 0.098). Comparing the CNTL group and the LBP group at PRE therapy, group (Figure 20) was not significant for either the ipsilateral activation levels of MULT (p = 0.222), ES (p = 0.603), EO (p = 0.378) or IO (p = 0.531) or the contralateral activation levels of MULT (p = 0.186), ES (p = 0.223), EO (p = 0.249) or IO (p = 0.264).

b) LBP Group at PRE therapy vs. LBP group at POST therapy:

Within the LBP group at POST therapy, there was no difference between the ipsilateral and contralateral activation levels of MULT (p = 0.401), ES (p = 0.366), EO (p = 0.216) or IO (p = 0.071). Comparing the LBP group at POST therapy group and the LBP group at PRE therapy, group was not significant (Figure 20) for either the ipsilateral activation levels of MULT (p = 0.197), ES (p = 0.293), EO (p = 0.091) or IO (p = 0.098) or the contralateral activation levels of MULT (p = 0.112), ES (p = 0.162), EO (p = 0.171) or IO (p = 0.216).

c) CNTL group vs. LBP group at POST therapy:

Comparing the LBP group at POST therapy and the CNTL group, group was not significant (Figure 20) for either the ipsilateral activation levels of MULT (p = 0.214), ES
(p = 0.367), EO (p = 0.111) or IO (p = 0.667) or the contralateral activation levels of MULT (p = 0.186), ES (p = 0.147), EO (p = 0.268) or IO (p = 0.291).
Figure 20 Comparing ipsilateral and contralateral muscle activations during the unstable periods for the CNTL group, the LBP group at PRE and POST therapy
4.4.5 Grouped extensor and flexor activation levels during stable and unstable periods at the 50% Stability Level

a) LBP group at PRE therapy vs. CNTL group:

During the stable periods the LBP group at PRE therapy had higher flexor activity levels on their ipsilateral side (p = 0.005) as compared to the CNTL group (Figure 21B) with no difference (p =0.183) on their contralateral side. There was no difference in the activity levels of the ipsilateral extensors (p=0.151) or the contralateral extensors (p = 0.587) between the two groups (Figure 21A) during the stable periods.

During the unstable periods, both the contralateral (p =0.013) and ipsilateral (p=0.010) flexors, had higher activity levels in the LBP group at PRE therapy (Figure 21D) compared to the CNTL group. There was no difference in the activity levels of the contralateral (p= 0.562) or the ipsilateral extensors (p = 0.098) between the two groups (Figure 21C).

b) LBP Group at PRE therapy vs. LBP group at POST therapy:

During the stable periods there was no difference between the two sessions in either the ipsilateral (p = 0.557) or contralateral (p=0.482) extensors muscle activity levels (Figure 21A). There was also no difference between the two sessions in either the ipsilateral (p = 0.336) or contralateral (p=0.365) flexors muscle activity levels during the stable periods (Figure 21 B).
During the unstable periods, both the contralateral (p = 0.034) and ipsilateral (p=0.045) flexors had lower muscle activity levels in the LBP group at POST therapy as compared to at PRE therapy (Figure 21 D). However, there was no difference in the activity levels of either the contralateral (p = 0.095) or the ipsilateral (p = 0.114) extensors between the two sessions (Figure 21 C).

c) CNTL group vs. LBP group at POST therapy:

During the stable periods there was no difference between the two groups in either the ipsilateral (p = 0.107) or contralateral (p=0.195) flexors muscle activity levels (Figure 21 B). However, both the contralateral (p = 0.011) and ipsilateral (p = 0.013) extensors in the LBP group at POST therapy had higher activity levels as compared to the CNTL group (Figure 21 A).

During the unstable periods, there was no difference between the two groups in either the ipsilateral (p = 0.111) or contralateral (p=0.198) flexors muscle activity levels (Figure 21 D). However, again both the contralateral (p = 0.021) and ipsilateral (p = 0.018) extensors in the LBP group at POST therapy had higher activity levels as compared to the CNTL group (Figure 21 C).
Figure 21 Comparing ipsilateral and contralateral extensors and flexors activations during the stable and unstable periods between the CNTL group, the LBP group at POST therapy, and the LBP group at PRE therapy. The symbol * indicates that the values for the LBP group at PRE therapy were significantly different from the CNTL group. The
symbol # indicates that the values for the LBP group at POST therapy were significantly different from the values at the PRE therapy session. The symbol ¥ indicates that the values for the LBP group at POST therapy were significantly different from the CNTL group.

4.5 Discussion:

The primary findings of this study were: 1) subjects with LBP at PRE therapy had increased levels of ipsilateral flexor activation during the unstable periods of the postural task compared to the healthy subjects; and 2) 10 weeks of stabilization exercise training decreased the activation levels of the ipsilateral and contralateral flexors during the unstable periods of the postural control task in the subjects with LBP at POST therapy. In addition, relative to the healthy CNTL subjects, the subjects with LBP at POST therapy had increased activation levels in both the ipsilateral and contralateral extensors during the stable and unstable periods of the postural control task.

The subjects with LBP at PRE therapy had increased levels of flexor activation on their ipsilateral side during the seated postural task. Animal studies\(^\text{28}\) have shown that after an injury to the disc on one side, loss of cross sectional area and infiltration of adiopocytes occurs at the ipsilateral segmental level of multifidus, while a denervation...
to the multifidus muscle causes changes in the multifidus ipsilateral to the lesion across multiple segments. Similarly, it has been observed in subjects with LBP that within 24 hours after their first LBP episode, the lumbar multifidus ipsilateral to the pain atrophies. The decreased cross sectional area may result in decreased strength in the muscle. In addition, delays in the reflex responses in the back extensor muscles of the subjects with LBP have also been reported. Decreased strength and recruitment coupled with delayed and dampened reflexes in the trunk extensor muscles may all contribute to an inability to adequately control or restrict the movement of the vertebral segments. The increased postural sway observed in the subjects with LBP during the postural task as described in Chapter 3 may be reflective of the inability to adequately control the movement of the individual vertebral segments. The increase in the activity levels of their flexors in the LBP group at PRE therapy could be indicative of a compensatory strategy to either avoid pain and/or to overcome the deficits brought on by impairments in the activations of their extensors. These results are similar to the results observed by Silfies et al where the authors observed higher activation levels in the flexors in a chronic LBP group compared to a healthy CNTL group during a functional reaching task in the sagittal plane.

After the 10-week stabilization exercise program, the subjects with LBP demonstrated a decrease in the activity levels in both their ipsilateral and contralateral flexors during the unstable periods of the seated postural task. There was no significant
change in the activity levels of either the ipsilateral or contralateral extensors between the two sessions. However, relative to the CNTL group, the LBP group at POST therapy had significantly higher activity levels in both their ipsilateral and contralateral extensors during the seated postural task. This suggests that the therapy program improved the recruitment of the extensor muscles in the LBP group relative to a healthy CNTL group which did not receive any intervention. This change along with the improved reflexes (Chapter 2) in the LBP group perhaps allowed them to have improved control over the vertebral segments, which is reflected in the decreased postural sway during the task as demonstrated in Chapter 3. These changes in recruitment were evident only when the muscle activity levels were grouped. At the level of individual muscles, the effects were not significant. EMG is a signal with a high degree of variability, and the dynamic nature of the balancing task could only add to that. Hence, it is not surprising in retrospect that only by pooling the data from the extensor versus flexor groups on each side were we able to detect a significant difference. In theory, since the multifidus was trained most specifically, it should have shown the most change. It is possible that this does indeed occur, and our measurements could not detect this in light of the variability. The other aspect to consider it that the rehabilitation program included the specific multifidus exercise training in conjunction with a broader rehabilitation program aimed at muscles throughout the core of the trunk and with that it is possible that the muscles did indeed change their recruitment patterns but overall as a group.
In a review to study the effects of different interventions such as activity modifications (changes in work practices or physical activities), personal appliances (lumbar supports and shoe inserts) and clinical approaches (exercise and education) in preventing LBP, the authors\textsuperscript{139} observed that the clinical approaches which included exercise intervention programs were the most effective in preventing self-reported LBP episodes in the subjects. Exercise therapy interventions\textsuperscript{140-142} in subjects with LBP have demonstrated increased activity levels as measured by EMG with increased strength and endurance\textsuperscript{140} observed in the erector spinae muscle after therapy. Apart from the general benefits of exercise such as increased strength, endurance and mobility, the added benefit of the subjects feeling more confident and active may add to the overall benefits of an exercise therapy intervention. The only other study\textsuperscript{143} which has looked at changes in muscle activity levels after a similar SSE program was done in healthy subjects, so a true comparison cannot be made. However, the authors\textsuperscript{143} did demonstrate that trunk muscle levels can be altered with increased flexor activity levels post training during bridging and four point kneeling exercises. In contrast, the subjects with LBP in this study demonstrated decreased flexor activity levels after 10-weeks of training. The differences in outcomes between the two studies could be explained by the differences in the tasks which were used to measure the muscle activity, the different subject population which was being tested. The wobble chair task involves balancing on a smaller base of support which requires rapid corrections over short
periods of time. In contrast, the four point kneeling and bridging tasks have a larger base of support and require fewer rapid corrections. Impaired postural control with an inability to make rapid corrections to perturbations has been suggested to increase the susceptibility of an individual to low back injuries.\textsuperscript{54} A task which allows one to assess the ability to make rapid and dynamic corrections might prove helpful in better understanding the effect of the exercise program on postural control. The subjects with LBP at POST therapy improved their postural stability during the wobble chair task (Chapter 3) which may be a reflection on the ability to more precisely control the movement of the vertebral segments.

Several limitations with regards to the study are acknowledged. The first limitation of the study was it remains unknown whether the use of the ipsilateral flexors in the LBP at PRE therapy represents a compensatory strategy in presence of pain or is a predisposing factor present before the onset of the pain. Future studies that include the testing of this hypothesis are warranted. The second limitation of the study was the small sample size. Given the fact that there were only 10 subjects in each group, it would be difficult to translate the findings of this study to the general population. In addition, no correction was undertaken for the multiple comparisons that were made for the individual muscles, and extensors and flexors, between the groups; in this regard, the study must be seen as exploratory. Another possible limitation of the study lies with the normalization procedure. For normalizing the EMG for the back muscles,
the mean activity from the Sorenson test was used in contrast to the MVC data for the flexors. The Sorenson test allows the subjects to be tested against gravity and accounts for the differences in their anthropometrics. This was primarily done to address the difficulty in eliciting a true maximum in the back muscles from LBP subjects due to pain or inhibition. For the flexors, it can be argued that since pain is not expected to be an issue with either the LBP group or the CNTL group, the normalization of the EMG data from their flexors using their MVC would not be an issue.

In conclusion, differences were observed in the neuromuscular control patterns between the healthy CNTL subjects and the subjects with LBP before PT with increased compensatory flexor activity being observed in the LBP group. The overall effect of the training program has been to shift the LBP group subjects from an increased flexor activity strategy at PRE therapy to an increased extensor activity strategy at POST therapy with the change resulting in improved postural stability.
Chapter 5 Discussions and Conclusions

The primary purpose of the study was to understand and compare the neuromuscular control of the trunk between subjects with LBP and subjects without LBP. The secondary purpose was to assess if a 10-week stabilization exercise program improved the neuromuscular control in subjects with LBP. Two experiments were designed: 1) A sudden perturbation test to assess the trunk muscle reflex responses, and 2) A seated postural control task to assess trunk equilibrium control.

In the first experiment with the sudden perturbation test, the subjects with LBP at PRE therapy exhibited delayed and dampened M2 reflex responses in the lumbar multifidus and erector spinae compared to the subjects without LBP. In the second experiment with the seated postural control task, the subjects with LBP at PRE therapy demonstrated increased postural sway compared to the subjects without LBP. In addition, an analysis of the trunk muscle activity during the same seated postural control task revealed increased activation of the flexors in the subjects with LBP at PRE therapy compared to the subjects without LBP. Similar findings have been observed in
the literature. Delayed trunk muscle reflex responses,\textsuperscript{53-55} larger trunk displacements\textsuperscript{54, 56, 74, 80, 144} and altered muscle activation patterns\textsuperscript{56, 74} have all been observed in subjects with LBP. The differences observed have been suggested to be representative of an impaired neuromuscular control system contributing to spinal instability and making the person susceptible for sustaining future low back injuries.

Radebold et al\textsuperscript{54} demonstrated a correlation between increased seated postural sway and increased delay in the onset times of the trunk muscle reflex responses. Delays in the reflex responses could potentially limit the subject’s ability to decrease trunk movement.\textsuperscript{48, 49} Reeves et al\textsuperscript{145} in an attempt to elucidate the link between seated postural sway and trunk muscle reflexes created a model for the seated postural task. The authors observed that an additional delay of 20ms in the trunk muscle reflexes increased trunk displacements by approximately 15%. However the addition of the 20ms delay created spinal instability in only 5 out of 25 trials, suggesting that the delay alone could not account for differences in postural control between those with and without LBP. The reflex responses are dictated not only by the time delay but also by the amplitude of the response which is referred to as the reflex gain. Differences in the reflex gain between subjects with and without LBP have not been very well documented in the literature. Franklin et al\textsuperscript{52} created a dynamic spine model to investigate the role of both the reflex gain and reflex delay in spinal stability. The authors observed that a spine with reduced intrinsic stiffness could be stabilized by a proportionally increased
reflex gain. In the current study the subjects with LBP exhibited not only delayed reflex responses but also smaller reflex gains. The delayed and dampened trunk muscle reflex responses could explain the greater postural sway observed in the subjects with LBP. Consequently, the observed increase in the activity levels of the flexors in the LBP group subjects at PRE therapy during the seated postural task could be indicative of a compensatory strategy to overcome the impairments in the extensors. It is also possible that the altered strategy may be a learned strategy to avoid pain. Pain induces widespread neuroplastic changes at many levels within the nervous system\(^{146}\) which include the primary afferent neurons, spinal cord, brain stem, thalamus, limbic system and the motor cortex. Chronic pain particularly is linked with regions related to cognition and emotions.\(^{147-149}\) The altered patterns of muscle recruitment seen in the subjects with LBP may have been initiated during the acute stage of pain and become a learned strategy with continued pain. Overall, these findings indicate deficits in the neuromuscular control system and spinal instability for the subjects with LBP with a susceptibility to sustain further injuries.

The 10-week therapy program (Appendix D) in addition to the strengthening and stabilization exercises also included initial training for the recruitment of the lumbar multifidus and transverse abdominis. After the 10-week stabilization therapy program, in the sudden perturbation test, the subjects with LBP POST therapy exhibited stronger reflex responses with an increase in their reflex amplitudes in their trunk extensors.
(lumbar multifidus and erector spinae). During the seated postural control task, the subjects with LBP at POST therapy had decreased postural sways along with a decrease in the activity levels of the flexors (internal and external oblique’s) during the stable periods compared to at their PRE therapy session. In addition, relative to the CNTL group, the LBP group at POST therapy had higher activation levels in the extensors during the same task.

These results demonstrate that the therapy strengthened and improved recruitment in the trunk extensors. The improved strength coupled with the more robust reflexes in the trunk extensors would allow the subjects with LBP to better control the movement of the vertebral segments and restrict overall trunk displacements. The improved neuromuscular control was reflected in the decreased postural sway observed in the subjects with LBP during the seated postural task. These results were the desired outcomes as envisaged during the design of the study. However, the onset times of the reflex responses in the subjects with LBP did not change after the therapy program, and the reflexes remained delayed as compared to the CNTL group. Changes in the onset times of the reflex responses could occur if a previously injured or compressed nerve became healthier or remyelination occurred due to restoration of an appropriate control over the segment and could show up as reduced latency. However, this phenomenon has been primarily demonstrated in the segmental stretch reflex through the H-reflex, and the F wave, which operate only at the
In this study, the segmental reflex in the form of the M1 wave was not observed. Hence any improvement in a short segment would be hard to detect when observing the M2 reflex which constitutes the full pathway including the supraspinal centers.\textsuperscript{49,50} However, the M2 responses require integration through polysynaptic circuits.\textsuperscript{39,50} Assuming part of the therapeutic change involved more excitable neurons at one or more stages of this circuit, then each post-synaptic cell would be close to threshold and quicker to respond.\textsuperscript{14,151-154} However, this could only add to a fraction of a millisecond per synapses. The increased amplitude of the responses, however, is exactly what would be expected in this situation,\textsuperscript{155} and this was demonstrated in the present study. The final means to substantially reduce latencies would be for a previously absent segmental response to re-emerge after therapy. This could lead to large, easily detectable reductions in latency between groups. However, again segmental responses were not observed even in the CNTL group subjects so this hypothesis could not be tested.

One of the objectives of rehabilitation and exercise programs is often to cause long standing changes in movement patterns towards a more efficient or optimum paradigm.\textsuperscript{146} The improved recruitment in the trunk muscles and improved strength of the trunk reflexes along with the decreased postural sway in the subjects with LBP after therapy was in the direction of the results observed in the healthy subjects and demonstrates improvement in the overall control paradigm. The outcomes from the
present study does support the notion that a 10-week stabilization program with initial training for the recruitment of the lumbar multifidus and transverse abdominis benefits subjects with subacute recurrent LBP. However, it remains to be seen whether the changes observed are sustained over longer periods of time and lead to reduction in the recurrence rate in the episodes of LBP.

5.1 Summary

1. Subjects with subacute recurrent LBP before therapy displayed delayed and dampened trunk muscle reflexes as compared to CNTL subjects. After a 10-week stabilization exercise program, the subjects with sub acute recurrent LBP demonstrated delayed but stronger trunk muscle reflexes.

2. Subjects with subacute recurrent LBP before therapy displayed increased postural sway and higher displacements in their short term region during a seated postural task as compared to CNTL subjects. After a 10-week stabilization exercise program, the subjects with subacute recurrent LBP demonstrated decreased postural sway and smaller displacements in their short term region.

3. Subjects with subacute recurrent LBP before therapy displayed increased compensatory flexor activity during a seated postural task as compared to CNTL subjects. After 10-week stabilization exercise program, the subjects with subacute recurrent LBP had decreased levels in their compensatory ipsilateral flexor
activity and had higher ipsilateral and contralateral extensor activity relative to the CNTL group.

5.2 Future work

Many questions remain unanswered at the end of this study. The role of the M1 short latency reflex in postural stability and the impact of the SSE program on its amplitude and onset time were not investigated. M1 reflexes have been typically elicited by tapping directly on the muscle or by sudden perturbations to the trunk which apply a direct stretch to the muscle and these protocols can be used to investigate M1 reflexes. Visual feedback and verbal encouragement has been shown to influence the H-reflexes, and may be used as a training protocol to potentially alter the M1 reflex responses.

Secondly, it remains to be seen whether the onset times of the M2 reflex responses can be altered. Training individuals to react to sudden perturbations or sudden changes in their loading conditions has been hypothesized as a potential method to alter the onset times of muscle reflexes. Pederson et al.\textsuperscript{103} trained individuals to react to sudden unexpected perturbations for 10 sessions, each lasting 45 minutes, spread over 4 weeks. The onset times of the reflex responses did not change, however the amplitudes did increase. Providing visual and auditory clues as part of the training protocol might prove beneficial and needs to be explored further.
Thirdly, alterations if any in the feedforward and anticipatory control were not measured in this study, the contribution of these mechanisms and the influence of the stabilization exercise program on them would be worth studying in the future. The transverse abdominis has been projected as an important muscle in the feedforward control loop; however its exact role remains to be conclusively established. The subjects with LBP were trained to recruit both the transverse abdominis and the lumbar multifidus during the initial part of the 10-week stabilization exercise program. However, the activity of the transverse abdominis was not recorded during the experiments in the present study. A future study establishing the activation patterns of the transverse abdominis, before and after the SSE program during self-perturbation tests such as raising the arm or during more complex postural control tasks such as the wobble chair is required.

Lastly, a future study to establish the effects of the training program over longer periods of time is warranted. A future study could include testing the subjects after 6 months or after 1 year with the same experiments, to observe if the altered muscle responses established by the training protocol were retained or if the subjects returned back to their PRE therapy muscle response patterns. In addition, a screening tool to assess the susceptibility of individuals to back pain based on delayed reflexes and altered neuromuscular patterns may also be worth exploring. If indeed, a predisposing
factor for LBP exists, preventive therapy programs may be prescribed to individuals to correct for those observed deficiencies in their neuromuscular control.

5.3 Conclusions

In conclusion, differences were found between healthy subjects and subjects with LBP. Subjects with LBP had delayed and dampened trunk muscle reflexes, increased postural sway and increased compensatory flexor activation levels as compared to healthy control subjects.

A 10-week stabilization exercise program improved the strength of the trunk muscle reflexes, decreased the postural sway and influenced muscle activations in the subjects with LBP. The stabilization exercise program did not influence the onset times of the reflex responses. Overall, the 10-week stabilization exercise program improved the neuromuscular control of the spine in the subjects with LBP in the direction as observed in healthy subjects.
References


146. Chudler EH, Dong WK. The role of the basal ganglia in nociception and pain. *Pain.* 1995;60:3-38.


Appendix A: Inclusion and exclusion criteria

Subacute Recurrent LBP Group: 52 Subjects will be recruited. Subjects will be aged 18-55 years to ensure that subjects are of similar age to those of the workforce who typically develop LBP. All subjects must be currently employed (or actively engaged in daily activities such as homemaking or studying). In addition to the duration (< 8 weeks) and recurrence of pain (at least 1 separate episode in past year), subjects must meet other criteria for the ‘immobilization’ classification in the TBC system. These include: 1) pain predominately in the lumbosacral area and is musculoskeletal in nature; 2) an absence of a clear directional preference (e.g. flexion or extension) for range of motion or activities that increase or decrease the intensity or distribution of pain; and, 3) at least moderate lumbosacral flexibility (e.g. > 35° lumbar flexion).

Patients will be excluded if they: 1) are involved in litigation or are off work, receiving worker’s compensation benefits; 2) have sciatica identified by corroborating clinical signs/symptoms such as pain radiating below the knee, muscle weakness or sensory loss in a dermatome pattern, and/or reduction of deep tendon reflexes; 3) show signs of a neurological disorder; 4) have a cardiovascular disorder that would limit their
ability to perform aerobic exercise such as walking, swimming, or stationary biking;
5) have a vertebral fracture, tumor, spondylolisthesis, or spinal stenosis; 6) have a
history of back surgery; 7) have systemic infection or disease; 8) are pregnant; 9) have
had surgery in the past 3 months; 10) have a primary psychiatric disease or alcoholism;
11) are morbidly obese (body mass index > 40 kg/m²) which would decrease the
reliability of the muscle CSA and EMG measurements; and 12) taking schedule II
medications for their pain (e.g. Demerol, morphine, or Percocete) – other medications,
such as nonsteriodal anti-inflammatory drugs (NSAIDs) are permitted as usual care. No
exclusions will be made with respect to race, ethnicity, or gender.

Subjects with LBP will be informed that a part of participation is the willingness to
refrain from receiving other treatments (e.g. chiropractic, massage) during the 10-week
intervention. To assess compliance with this request, each week the PT will 1) inquire
regarding current mediations and use of alternative interventions, 2) record responses,
and 3) remind the subjects to refrain from activities that are outside the boundaries of
the research protocol. If protocol violations occur, this information will be evaluated for
potential covariates in the statistical analyses. In return for the subject’s participation,
they will receive 10-weeks of free PT (see letter of support by Robert Vanecko, Director
of Rehabilitation) and $25 per visit ($75) to the OSU Human Movement Performance
Lab to compensate for their time. Compensation will be pro-rated if both visits are not
completed.
**Control Group:** 26 Subjects will be recruited. Subjects in the control group will be included if they: 1) are healthy adults between the ages of 18-55 years; 2) have a physical examination (range of motion, sensation, strength, and reflexes) of the trunk and lower extremities that is within normal limits; and, 3) have at least moderate lumbosacral flexibility (e.g. > 35 degrees lumbar flexion). Subjects will be excluded if they: 1) meet any of the exclusion criteria defined in the LBP group; 2) have a history of back pain within the past 2 years significant enough to receive medical care; 3) received stabilization exercise training in the past. Efforts will be made to obtain a control group who are similar to the LBP group in terms of age, gender, BMI, and activity levels. Between group differences in the baseline characteristics will be evaluated for potential covariates in the statistical analyses, if necessary. Subjects will be compensated $25 for time at the laboratory.
Appendix B: Consent form

The Ohio State University Consent to Participate in Research
Subjects with Low Back Pain

Study Title: Efficacy of Therapeutic Exercise for Recurrent Back Pain
Principal Investigator: Deborah Givens Heiss, PT, PhD, DPT, OCS
Sponsor: National Institutes of Health - National Center for Medical Rehabilitation Research (NCMRR), NICHD

- This is a consent form for research participation. It contains important information about this study and what to expect if you decide to participate. Please consider the information carefully. Feel free to discuss the study with your friends and family and to ask questions before making your decision whether or not to participate.

- Your participation is voluntary. You may refuse to participate in this study. If you decide to take part in the study, you may leave the study at any time. No matter what decision you make, there will be no penalty to you and you will not lose any of your usual benefits. Your decision will not affect your future relationship with The Ohio State University. If you are a student or employee at Ohio State, your decision will not affect your grades or employment status.

- You may or may not benefit as a result of participating in this study. Also, as explained below, your participation may result in unintended or harmful effects for you that may be minor or may be serious depending on the nature of the research.

- You will be provided with any new information that develops during the study that may affect your decision whether or not to continue to participate. If you decide to participate, you will be asked to sign this form and will receive a copy of the form. You are being asked to consider participating in this study for the reasons explained below.

1. Why is this study being done?
We are interested in learning about the fitness and coordination of the low back muscles in individuals who have had episodes of low back pain in the past year that have improved, only to have another episode within the past 8 weeks. There are two purposes to our research. The first is to compare the muscle fitness and coordination of individuals who are having this type of back pain to a similar group of subjects who do not have back problems. The second is to determine if a 10-week exercise program improves the fitness and coordination of the low back muscles as well as improves the pain and ability to perform in daily, work, and recreational activities.

2. **How many people will take part in this study?**
   There are 52 subjects with low back pain and 26 individuals without a recent history of low back troubles.

3. **What will happen if I take part in this study?**

   **Testing in the Laboratory**
   You will come for testing on two dates at the Human Movement Performance Lab in 236 Atwell Hall, 453 W. 10th Ave, Columbus, OH. The first date is at the start of the study, the second is at 11 weeks at the end of the study. Each session will last 4.0 hours and you will be compensated $25.00 for each session. First you will complete a series of questionnaires including:
   1) A questionnaire to obtain information about the history of your low back problem and your confidence in the proposed physical therapy program. We will ask about your medical history for heart, lungs, and other illnesses that may prevent you from being able to safely participate in the exercise program.
   2) The Modified Habitual Physical Activity Questionnaire. This questionnaire will ask you to rate how active you are during daily, work, and recreational activities.
   3) The Oswestry Low Back Disability Questionnaire – to rate how much your low back pain affects your ability to do activities such as walking, sitting, or lifting.
   4) Fear Avoidance-Beliefs Questionnaire - to rate how much you believe that physical activity and work affect your low back pain.
   5) Visual Analog Scale – to rate how much pain you are experiencing now and also your pain at its worst (most intense) and best (mildest).
After completing the questionnaires, the following procedures will be performed:

1) You will have your height and weight measurements taken using a scale similar to those used in a doctor's office.

2) You will undergo an examination by a physical therapist to determine the strength, endurance, and flexibility of your low back. You will have the range of motion of your low back, hips, and legs measured with a handheld device similar to a level. A test which is commonly used in the clinic will be performed to determine the endurance of the back muscles. For the test, you must hold your upper body in a horizontal off the edge of a table. You will be given a bench or a stool to support your upper body until the test is started. Straps will be placed across your lower body to stabilize it to the table. You will be asked to hold this position for up to 3 minutes. You may stop the test at any time if you are unable to maintain this position or can no longer tolerate the procedure.

3) You will have the size of your muscles at the base of your spine measured (multifidus) and on your stomach (transverses abdominis) as you lie on a padded table. This technique, which uses ultrasound imaging (pictures), is frequently used to image unborn babies and is a safe procedure. You will have your stomach muscle size measured while you are relaxed, while you are bearing down in a manner similar to when you are having a bowel movement, and while you are performing a “drawing in” exercise. You will be instructed to breathe normally during these activities. You will have your back muscle measured while you are relaxed, while you are lifting one arm overhead, and while holding a 1.5, 2, or 3 lb weight (based on your body weight) in your hand when you lift your arm overhead.

4) You will have fine-wire recording electrodes placed in your low back muscles (multifidus). First, you will be shown a drawing of where the electrodes will be placed. Then, you will have the skin in this area cleaned with rubbing alcohol to remove dirt, oil and dead skin. If necessary, you will have the hair shaved in this small area, too. Betadine ointment will be applied to your skin to prevent infection. An anesthetic agent will be sprayed on your skin over the implantation site to numb the skin so that you will not feel the insertion of the needle that contains the wire electrodes. The needle that contains the fine-wire electrodes will be withdrawn, leaving behind the wire electrodes. At the conclusion of the session, the fine-wire electrodes will be removed by gently pulling on it.

5) So that the muscle responses in your major low back and abdominal muscles can be recorded, you will have the skin cleaned in certain eight locations in your
abdominal region and on your back. If necessary, you will have the hair shaved in these small areas. Your muscle responses will be measured using small plastic disks (surface electrodes) taped to your skin in these locations.

6) You will have measurements to determine how well you can maintain and control your balance while in sitting. For this, you will be assisted into a sitting position on the device that is called the “wobble chair.” After you are comfortable and secured with a seat belt on the wobble chair, your normal limits of stability will be determined by placing the wobble chair in a position of approximately 10 degrees forward and then approximately 10 degrees backward tilt while data is collected for 5 seconds. Using that information, the wobble chair settings will be systematically adjusted to create easy, moderate, and difficult levels of stability. For your safety, while you are on the wobble chair, someone will be standing nearby to assist you in case you are unable to stabilize your balance. You will be allowed to practice at each setting until you feel comfortable. Then, you will perform at least two 1 minute trials at each setting while data is recorded. You will be given rest breaks between each repetition. While on the wobble chair, the activity of your muscles will be detected from the electrodes on your abdominal and back muscles. Your body movements will be detected by special cameras that track the positions of small plastic disks placed on the mid and lower back as well as on the wobble chair.

7) You will have tests to see how your muscles in your trunk respond to small, rapid pulling forces. For these tests, you will kneel on a chair and wear a belt around your hip to secure your lower body. You will wear a harness on your upper body. You will keep your posture upright and a cable will be attached from the harness to a motor mounted on the on a frame in front of you. This cable will tend to pull you forward in the harness and you will need to maintain your normal upright posture. Your response to different levels of pull from the rope attached to the motor will be tested. Depending on your weight, the amount of these pulls will be between 20N (5 lb) and, at most, 180 N (40 lbs). At each level of pull, you will feel small, random pulling forces through the cable which will cause your upper body to move a small amount backward and forward. Each series of pulls lasts for 30 seconds and each level (light, medium, and strong) will be repeated twice. During these tests, the reflex activity from your trunk muscles will be recorded.

This research includes collaboration between Dr. Heiss and engineering researchers at Virginia Tech. The data from the wobble chair and tests of your muscle responses to the pulling forces will be processed by a research assistant at Virginia Tech, under the supervision of Shane Ross, PhD, a co-investigator on the study. All personal
identifiers will be removed and your data will be labeled according to your assigned subject number in the study.

Outpatient Physical Therapy
You will attend outpatient physical therapy at the Ohio State University Spine Center, 2050 Kenny Road, Columbus, OH. At your first visit, you will answer questions about your low back problems and your ability to work or perform your usual activities. You will answer questions about your health and past medical history. The therapist will help you complete the Patient Specific Functional Scale to identify five important activities of difficulty because of your low back pain.

The physical therapist will perform a routine examination of your low back and legs. You will be asked to rate your pain when you bend forward, backward and side-to-side and when twisting. You will perform movements of your trunk and legs to test the strength of the muscles. You will have the reflexes in your legs checked when the therapist taps the muscles with a reflex hammer. You will answer questions about whether you have any loss of feeling in your legs when the therapist touches you lightly or uses a sharp edge of a pin used for testing purposes. You will perform tests to determine if your pain is affected by movements of your legs or when the therapist presses on areas of your low back.

Once the low back examination is completed, the physical therapist will start the activities involved in the rehabilitation program for your low back, called segmental stabilization exercise or SSE training. You understand that in turn for free physical therapy, you are being asked to attend 9 more physical therapy appointments. These appointments will be scheduled at your convenience twice a week for the first two weeks (weeks 1 and 2), then once per week for four weeks (weeks 3-6), then once every 2 weeks (weeks 8 and 10). You understand that you must attend at least 7 of the 10 appointments. If you miss more than 2 consecutive visits, you will be removed from the research study.

You understand that during the time you are enrolled in the study, you should not take strong pain medication such as Demerol, morphine, or Percocete during this time. You are allowed to take mild pain medications, such as nonsteriodal anti-inflammatory drugs (examples: aspirin, ibuprofen, naprosyn) or Tylenol. If you are unsure if a medication is permitted, you understand that you can ask your physical therapist or the researchers for confirmation. You also understand that you should not seek other treatments for your low back pain such as acupuncture, massage, or chiropractic care.
At each physical therapy appointment, you will perform exercises under the supervision of the physical therapist. You understand that these exercises start at a very light level
and are progressed based on your tolerance. The exercises involve stretching the low back and leg muscles, exercising the stomach and low back muscles, practicing your daily activities using good body mechanics, and performing general exercises such as walking or swimming for fitness. When you are learning to specifically exercise your deep low back (multifidus) and abdominal muscles, the physical therapist will use the ultrasound imaging machine so that you can see if and when your muscles are working. The therapist will spend time teaching me about the causes and ways to prevent low back pain. You will learn exercises to do at home every day. You will be given an exercise sheet to use to record that you performed your exercises as directed. You will bring the exercise sheet and give them to the physical therapist at every appointment.

As your pain, strength, and flexibility improve, the therapist will progress your exercise program. You may be asked to do more repetitions of your exercises, lift weights, do new exercises and walk, bicycle or swim for longer durations or participate in aerobic exercise classes. You will practice doing activities in your work or play that are difficult for me with the coaching of the physical therapist in how to move your body and control your muscles. Your home exercises will be progressed in a similar manner to those that you do in the clinic.

Six months after finishing the study, you will be contacted by phone or email. You will be asked if you have had a recurrence of back pain in the past 6 months since finishing the study. If you have had a recurrence of pain, we will ask you if it is due to a new injury or from the same problem that you are having now. We will also ask you to rate your pain at the time we contact you using a scale from 0 to 10 (best or mildest pain to worst or most intense pain).

4. How long will I be in the study?
Your active participation in the study will be 11 weeks. You will also be interviewed by phone or email 6 months later. The following table provides a breakdown of your specific time commitment.
<table>
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<tr>
<th>Activity</th>
<th>Time Commitment</th>
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<tr>
<td>Baseline Data Collection (week 0)</td>
<td>4.0 hr</td>
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<tr>
<td>Post Treatment Data Collection (week 11)</td>
<td>4.0 hr</td>
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<tr>
<td>Physical Therapy Treatment: 10 visits x 45 min</td>
<td>7.5 hr</td>
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<tr>
<td>Home Exercise Program: 30 min/day x 70 days</td>
<td>35 hr</td>
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<tr>
<td>Phone or Email Interview at 6 months</td>
<td>.25 hr</td>
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<td><strong>Total</strong></td>
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5. **Can I stop being in the study?**

You may leave the study at any time. If you decide to stop participating in the study, there will be no penalty to you, and you will not lose any benefits to which you are otherwise entitled. Your decision will not affect your future relationship with The Ohio State University.

6. **What risks, side effects or discomforts can I expect from being in the study?**

When doing the tests in the research lab on 2 occasions, there is a risk that you may experience muscle soreness, sprain or strain, or fatigue during the activities. To minimize the chance of this happening, you will be given frequent rest breaks and will perform warm-up exercises whenever you are asked to do something strenuous with your low back muscles. You may experience some temporary discomfort with insertion of the needles to place the fine-wire electrodes for study of the multifidus, a deep muscle at the base of the low back. An anesthetic agent will be sprayed on your skin over the implantation site to numb the skin to reduce the risk of discomfort with this procedure. There is a very small risk of a local infection. The researchers will clean your skin carefully with alcohol and betadine and use sterile instruments to minimize this risk.

During the activities to measure the way your trunk muscles coordinate to maintain your upper body balance, you will be secured in a frame to prevent falling. When you pull back against the cable attached to the harness that you wear, you may experience discomfort in your back muscles. In order to minimize the risk of straining your low back, you will do warm up exertions before you give your exertion. These efforts are not large but may cause short term muscle soreness.
When you sit on the wobble chair, you may find sitting without support to be uncomfortable and tiring. This discomfort should be relieved when you can stand at the end of the test.

There is a risk of skin irritation or allergic reaction from the adhesive used to attach the small disks to the skin to record the muscle activity and movements of the body. You understand that the electrodes do not send an electrical current to your body so you will not feel an electrical current or experience an electrical shock.

In addition to the above risks, side effects or discomforts, there is a risk of aggravating your back pain as with any exercise program. There is a risk of increased pain, soreness, and fatigue in your arms, legs, and trunk during and shortly after the exercises or aerobic activities (walking, stationary bicycling, or swimming). Most patients who experience this response report that the increase in discomfort occurs during the exercises and up to 30 minutes afterwards but then it goes away. In previous research using similar exercises, most patients reported improvement in their pain within a few weeks and ability to carry out their work and daily activities.

There is a small chance that you may not experience improvement in your low back pain with the exercise program. Although rare, there is a very small risk that the exercise program could make the back pain worse in intensity and duration, but this has not been reported in other studies that used this exercise program for patients with low back pain.

7. **What benefits can I expect from being in the study?**
   You may directly benefit from the exercise program because of the improved strength and fitness of your low back muscles and the education and training that you have received in the prevention of low back injury. You may also reduce the risk of experiencing another episode of low back pain.

   The information that is gained from these experiments will improve the scientific understanding of the neuromuscular problems of those with acute, recurrent low back pain. Rehabilitation specialists can better evaluate the problem of low back pain and design more specific treatments (for example, what exercises to prescribe) for individuals with low back pain

8. **What other choices do I have if I do not take part in the study?**
   You may choose not to participate without penalty or loss of benefits to which you are otherwise entitled. You may receive physical therapy or other treatments for your low back.
9. **Will my study-related information be kept confidential?**

   Efforts will be made to keep your study-related information confidential. However, there may be circumstances where this information must be released. For example, personal information regarding your participation in this study may be disclosed if required by state law. Also, your records may be reviewed by the following groups (as applicable to the research):
   - Office for Human Research Protections or other federal, state, or international regulatory agencies;
   - U.S. Food and Drug Administration;
   - The Ohio State University Institutional Review Board or Office of Responsible Research Practices;
   - The sponsor supporting the study, their agents or study monitors; and
   - Your insurance company (if charges are billed to insurance).

   If the study involves the use of your protected health information, you may also be asked to sign a separate Health Insurance Portability and Accountability Act (HIPAA) research authorization form.

10. **What are the costs of taking part in this study?**

    As a participant in this study, there are no costs to you.

11. **Will I be paid for taking part in this study?**

    You will be paid $25.00 for each visit at the laboratory. If you are unable to complete the study for any reason, the compensation will be prorated based on the number of visits to the lab. You will be provided a voucher to cover the cost of parking when coming to the research lab.

    By law, payments to subjects are considered taxable income. If you are an OSU employee, any compensation you receive as a result of participating in the study will be made through the payroll system and applicable taxes will be deducted.

12. **What happens if I am injured because I took part in this study?**

    If you suffer an injury from participating in this study, you should notify the researcher or study doctor immediately, who will determine if you should obtain medical treatment at The Ohio State University Medical Center. The cost for this treatment will be billed to you or your medical or hospital insurance. The Ohio State University has no funds set aside for the payment of health care expenses for this study.
13. What are my rights if I take part in this study?
If you choose to participate in the study, you may discontinue participation at any time without penalty or loss of benefits. By signing this form, you do not give up any personal legal rights you may have as a participant in this study.

You will be provided with any new information that develops during the course of the research that may affect your decision whether or not to continue participation in the study.

You may refuse to participate in this study without penalty or loss of benefits to which you are otherwise entitled. An Institutional Review Board responsible for human subjects research at The Ohio State University reviewed this research project and found it to be acceptable, according to applicable state and federal regulations and University policies designed to protect the rights and welfare of participants in research.

14. Who can answer my questions about the study?
For questions, concerns, or complaints about the study you may contact Dr. Deborah Heiss at 614-292-0380 or 516 Atwell Hall, 453 West 10th Ave, Columbus, OH 43210.

For questions about your rights as a participant in this study or to discuss other study-related concerns or complaints with someone who is not part of the research team, you may contact Ms. Sandra Meadows in the Office of Responsible Research Practices at 1-800-678-6251.

If you are injured as a result of participating in this study or for questions about a study-related injury, you may contact Dr. Deborah Heiss at 614-292-0380 or 516 Atwell Hall, 453 West 10th Ave, Columbus, OH 43210.

Signing the consent form
I have read (or someone has read to me) this form and I am aware that I am being asked to participate in a research study. I have had the opportunity to ask questions and have had them answered to my satisfaction. I voluntarily agree to participate in this study.

I am not giving up any legal rights by signing this form. I will be given a copy of this form.
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**Investigator/Research Staff**

I have explained the research to the participant or his/her representative before requesting the signature(s) above. There are no blanks in this document. A copy of this form has been given to the participant or his/her representative.

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**Witness (es) - May be left blank if not required by the IRB**

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Date and time

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Appendix C: Research protocol

All potential subjects will be screened in person or by phone by the PI (Dr. Deborah Givens) or her designee to ensure that the individual meets basic eligibility criteria (e.g. age, pain status/duration, LBP history, and height/weight). Those with LBP, who meet this initial screening, will be scheduled for an examination by the two PTs participating in the research project at the OSU Spine Center, 2050 Kenny Road. The information obtained in the exam serves as the evaluation for participation in the study and for outpatient PT. Those recruited as asymptomatic controls will undergo a similar initial evaluation by a PT at the PI’s lab in 235 Atwell Hall. We will obtain participation consent and HIPAA authorization according policies and procedures of the OSU Institutional Review Board. The subject will provide basic information such as age, gender, height, weight, and occupation. A structured questionnaire with an interview will obtain a detailed history which includes: the temporal and physical nature of the LBP, including the VAS for pain intensity; prior history of LBP including precipitating events; treatments for LBP (past and current); medical history; work history; and current medication intake. Both groups will complete the MHPAQ. The LBP group will complete the VAS, Oswestry, PSFS, and FABQ.
The PT will complete a detailed physical examination using the treatment-based classification system examination procedures and forms from our previous study. In brief, this involves examination of the trunk and lower extremities for range of motion, strength, sensation, and reflexes. For the subjects with LBP, an important component of the evaluation is the pattern of the pain response (increase/decrease or change in distribution) during active trunk movements and daily activities.

Subjects who meet all of the criteria for the study will have baseline testing within 3-5 days in the PI’s Lab. Subjects with LBP who are not eligible will be scheduled for outpatient PT, in accordance with the clinic’s policies and procedures. All data collection procedures in the lab will be carried out by a trained research assistant who is blinded to the subject’s group. Each data collection session will take approximately 4 hours. For the LBP group, the procedures will be the same for the pre and post-treatment testing.
Appendix D: Segmental stabilization exercise protocol

STAGE 1

<table>
<thead>
<tr>
<th>Therapeutic Exercise</th>
<th>Stage 1 (Pain Management – max 2 weeks)</th>
<th>Patient will progress to Stage 2 when:</th>
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<tr>
<td>Stretching trunk</td>
<td>double/single knee to chest include “Pelvic Clock” in supine, sitting, standing, or ½ kneeling</td>
<td></td>
</tr>
<tr>
<td>Stretching LE</td>
<td>Gastroc After walking Hams Quads 30 secs; 3-5 reps</td>
<td>contraction of TrA for 3 reps 10 sec in neutral pelvis position with proper breathing.</td>
</tr>
<tr>
<td>Motor Control</td>
<td>Train central and lateral diaphragm breathing Describe pelvic tilt and mid-range (neutral zone). Have patient try to move through range of movements (note: low level global mm necessary for movement). “Pelvic Clock” in sitting on the edge of firm table/chair or in supine. Have pt. achieve neutral lordosis w/o use of global muscle substitution. – (this is a balancing act (proprioception) of the COM acting through the Lx. Once the perception is achieved then global mm can be used to move in and out of the neutral spine position. Until then patients may use lower limb or thorax to manipulate COM acting through the Lx spine – initially OK – but this technique cannot be progressed to functional activities.)</td>
<td>contraction of Mult for 3 reps 10 sec in neutral pelvis position with proper breathing.</td>
</tr>
<tr>
<td><strong>Functional Activities</strong></td>
<td>Patient-specific functional scale (PSFS) administered. Based on that, PT designs progression of appropriate functional activities. Very important to target low-load, high-risk activities.</td>
<td></td>
</tr>
<tr>
<td>--------------------------</td>
<td>--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td><strong>General Conditioning Exercises</strong></td>
<td>Walking up to 15 min at an RPE level of 3 (moderate) w/ no increase or detrimental change in symptoms (less than 20% increase). Control of the lumbo–pelvic movements can be integrated here.</td>
<td></td>
</tr>
<tr>
<td><strong>Therapeutic Exercise</strong></td>
<td><strong>Stage 1 (Pain Management – max 2 weeks)</strong></td>
<td>Patient will progress to Stage 2 when:</td>
</tr>
<tr>
<td><strong>Positional Exercises to decrease pain</strong></td>
<td>Positional Exercises (Fine – so long as extreme sustained positions do not cause increase in catching sensation when they return to)</td>
<td>A change (for the positive) in two of the three characteristics of pain as measured on the patient-specific functional scale:</td>
</tr>
</tbody>
</table>
### Patient Education

- **Anatomy (Active, Passive and motor control subsystems) posture**
- **Body mechanics (sitting, lifting, bed, ADLs)** – These are often high load biomechanical problems – most instability patients would need reference to low loading high risk activities previously identified as problematic; reach for phone, coffee, etc.
- **Pain management (ice, stretching, aerobic ex, relaxation, self traction, lumbar roll – in the context of supporting the neutral pelvis position)**
- **Back education booklet given**
- **Benefits of General exercise**
- **Benefits of Specific Exercises.**

### Home Program

- **Practice TrA/Mult co-contraction with breathing** (10 10 sec holds - 3x/day). Include easier position as well as more challenging position (e.g. supine, sitting; sitting, 4 pt kneel). Encourage patient to practice in a variety of positions.
- **Practice body mechanics** – this

### Analysis

- **Normal postures**
  - Side glide
  - Trunk ex/flexion
  - Self traction over a pillow
  - Supine rotation

- **Intensity (visual analogue scale)** (pain needs to decrease by 20-25%; about 2 points on VAS)
- **Behavior (e.g. increase in available trunk ROM prior to onset of pain)**
- **Location (e.g. centralized)**
- **Nature** – recorded but not used in progression

- **Verbalize/explain and demonstrate** (if applicable)
  - Patient can accurately explain/verbalize what they can/will change at work to decrease postural stresses;
  - Patient can accurately explain “what is wrong with my back” and can explain “why I am doing these particular exercises.”
  - Patient can describe low-load movement patterns that are of increased risk for them.
  - Patient can articulate the concept of retraining the motor control and muscles as compensatory mechanisms for physical changes in the spine.

- **Proficiency of home program**
  - Patient can demonstrate proficiency of home program (defined proficiency as ability to perform all home exercises, for the prescribed frequency, without more than 1 cue/corrective prompt from PT)
includes low-load postures with a neutral pelvis position with TrA/Mult isolation techniques. (e.g., reaching for the mouse, phone, gear shift, etc)
Practice pain management strategies (including reducing shared attention and fatigue during tasks of high risk)
### STAGE 2

<table>
<thead>
<tr>
<th>Therapeutic Exercise</th>
<th>Stage 2 (Beginning Impairment and Functional level)</th>
<th>Patient will progress to Stage 3 when:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stretching trunk</td>
<td>Stretching trunk in single planes</td>
<td>No increase or detrimental change in symptoms with stretches</td>
</tr>
<tr>
<td></td>
<td>supine</td>
<td>Can complete number of reps easily using correct form.</td>
</tr>
<tr>
<td></td>
<td>seated</td>
<td>Therapist ensures that those with good flexibility do not do extreme stretches (as they tend to do). Need to explain that people tend to do the activities that they are good at and these activities are the least necessary for them. A muscle stretch should be felt in the muscle belly – for example a bent knee hamstring stretch – is felt in the hamstrings not the back of the knee.</td>
</tr>
<tr>
<td></td>
<td>standing</td>
<td></td>
</tr>
<tr>
<td></td>
<td>prone</td>
<td></td>
</tr>
<tr>
<td></td>
<td>supine trunk rot.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>30 second hold, 3 reps post ex.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pelvic Clock – stretch and stability progressions.</td>
<td></td>
</tr>
<tr>
<td>Stretching LE</td>
<td>Stretching LE hamstrings –90/90, chair,long sitting</td>
<td>No increase or detrimental change in symptoms with stretches</td>
</tr>
<tr>
<td></td>
<td>iliopsoas/ rectus femoris – half kneeling, standing, foot on chair piriformis –tailor position or supine</td>
<td>Can complete number of reps easily using correct form.</td>
</tr>
<tr>
<td></td>
<td>30 second hold, 3 reps post ex.</td>
<td></td>
</tr>
<tr>
<td>Motor Control</td>
<td>Patient co-contracts while initiating controlled movements of the limbs. (ex: co-contraction in crook lying, progression of a controlled single leg slide or lateral leg drop out). In functional postures patients holds and then moves in and out of neutral pelvic position. Quality</td>
<td>Patient able to complete a previously painful movement with proper form (co-contraction and breathing) and minimal to no pain. The movement could be a component of an activity from the Stratford scale or an ADL (standing, brushing the teeth)</td>
</tr>
</tbody>
</table>
of movement important. Ex: 4 pt kneeling, flexion of 1 UE while co-contracting
Ex: prone: co-contraction with knee flexion or hip ext.
Ex: standing – weight transference – progress to single step up and down

<table>
<thead>
<tr>
<th>Functional Activities</th>
<th>ADLs (specific for the increased risk activities as reported by the patient and/or determined by PT) dressing/self care house chores work recreational Baseline level established - must use good form/control</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Patient is asked to rate how well he can hold and then move in and out of the neutral pelvis position – does he keep breathing, is he able to achieve this without a gross co-activation of the “bad” (global) muscles☺</td>
</tr>
<tr>
<td></td>
<td>Patients symptom reproduction with functional activity in minimal (less than a 20% increase) Quality and ease of movement improve Patient verbalizes interest in functional activities</td>
</tr>
<tr>
<td>Therapeutic Exercise</td>
<td><strong>Stage 2 (Beginning Impairment and Functional level)</strong> Patient will progress to Stage 3 when:</td>
</tr>
<tr>
<td>General Conditioning Exercises</td>
<td>General Conditioning Exercises – may need to consider interval training to maintain quality of movements (Treadmill) walking OR stationary bicycling (no resistance to start – patient must maintain neutral pelvis/ good form throughout. UE/LE strengthening exercises swimming aerobics Baseline level established with 10 reps using good form. For aerobic conditioning – exercise at workload that elicits an RPE of 3 (moderate) for 15 minutes.</td>
</tr>
<tr>
<td></td>
<td><strong>Treadmill</strong>: speed, grade, OR time can be increased by 20% w/o increase in Sx or detrimental BP/pulse response <strong>Bicycle</strong>: patient has increase tolerance for biking time by 20% w/o increase in Sx or detrimental BP/pulse response <strong>UE/LE strengthening</strong>: patient can complete 2 sets of 10 reps of strengthening program using the 10 RM max. <strong>Swimming</strong>: patient can complete 15 min of overhead stroke w/o increase in Sx or detrimental BP/pulse response <strong>Aerobics</strong>: patient can complete 15 min of low impact aerobics tape/class w/o increase in Sx or detrimental BP/pulse response</td>
</tr>
<tr>
<td>Positional Exercises to</td>
<td>Continued as needed for pain relief and management</td>
</tr>
<tr>
<td>Patient Education</td>
<td>Patient ed continued as needed</td>
</tr>
<tr>
<td>-------------------</td>
<td>--------------------------------</td>
</tr>
<tr>
<td><strong>Home Program</strong></td>
<td><strong>Stretching ex (trunk/LE)</strong></td>
</tr>
<tr>
<td></td>
<td>Motor control ex, given above</td>
</tr>
<tr>
<td></td>
<td>Functional activities</td>
</tr>
<tr>
<td></td>
<td>General conditioning</td>
</tr>
<tr>
<td></td>
<td>Pain management</td>
</tr>
<tr>
<td></td>
<td>Body mechanics</td>
</tr>
</tbody>
</table>
### STAGE 3

<table>
<thead>
<tr>
<th>Therapeutic Exercise</th>
<th>Stage 3 (Moderate/Advanced Impairment and Functional level)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stretching trunk</td>
<td>Stretching exercises in Multiple planes</td>
</tr>
<tr>
<td></td>
<td>30 second hold, 3 reps post ex.</td>
</tr>
<tr>
<td>Stretching LE</td>
<td>Stretching exercises continued as needed and following conditioning exercise Elvis can continue and live on.</td>
</tr>
<tr>
<td>Motor Control</td>
<td>Further refine a particular movement and bring subcomponents together. Identify 2 or 3 faulty and painful movements. Break them down into subcomponents (i.e. erasing the board). Do high reps of components while performing co-contraction. First carried out while in neutral spine and then with normal spine movement. (Must control pain and co-contraction throughout) Ex. sit to stand, walking, lifting, bending, twisting – or use activities identified on Pt Specific Functional Scale.</td>
</tr>
<tr>
<td>Functional Activities</td>
<td>Patient-specific per scale</td>
</tr>
<tr>
<td>General Conditioning Exercises</td>
<td>Treadmill: increase speed, grade as tolerated to a level that elicits an RPE of 3 (moderate) w/o increase in Sx or detrimental BP/pulse response. THR maintain for duration. Bicycle: add pedal and arm resistance; increase workload as tolerated to a level that elicits an RPE of 3 (moderate) w/o increase in Sx or detrimental BP/pulse response. THR maintain for duration. Use of real bike on flat, then hill. UE/LE strengthening: increase weight and reps as tolerated w/o increase in Sx or detrimental BP/pulse response. Add exercises that involve Multiple planes Swimming: increase workload as tolerated to a level that elicits an RPE of 3 (moderate) w/o increase in Sx or detrimental BP/pulse response. THR maintain for duration. May add different kinds of strokes (butterfly, breast) Aerobics: increase workload as tolerated to a level that elicits an RPE of 3 (moderate) w/o increase in Sx or detrimental BP/pulse response. THR maintain for duration. Add high impact, kickboxing or a full class, if appropriate</td>
</tr>
<tr>
<td><strong>Positional Exercises to decrease pain</strong></td>
<td>Continued as needed for pain relief and management</td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>--------------------------------------------------</td>
</tr>
<tr>
<td><strong>Patient Education</strong></td>
<td>Patient ed continued as needed</td>
</tr>
</tbody>
</table>
| **Home Program** | The patient works towards functional activities and tries to remember the “maintenance” levels of motor skill. Patient is aware that all new activities or previously learnt activities performed at a higher level need to be thought about and performed in a sensible way until the skill is learnt.  
Increased rep/weight/time  
Pt. carries out movement components on daily basis with pain control, gradually increase the speed/complexity of mvt – until it reflects the individuals ADLs.  
Stretching ex (trunk/LE)  
Functional activities  
General conditioning  
Pain management  
Body mechanics |