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Effects of task difficulty on lumbar spine postural control

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Abstract

Effects of Task Difficulty on
Lumbar Spine Postural Control

by Brian L. Bettendorf

The experimental purpose was to determine how task difficulty and pain history affect postural control measurements. The practical purpose was to develop a field measure of postural control. Participants were assigned to a low back pain (6 males, 6 females, M = 36.8 years, SD = 7.32, M = 169 cm, SD = 5.57, M = 64.3 kg, SD = 14.2) or control group (6 males, 6 females, M = 39.1 years, SD = 6.92, M = 171 cm, SD = 8.21, M = 67.6 kg, SD = 14.9). PATH and center of pressure trajectories were quantified in medio-lateral (MAX ± M-L, ABS AVG M-L, RMSE M-L) and antero-posterior directions (MAX ± A-P, ABS AVG A-P, RMSE A-P) using a Bertec force plate. Task difficulty significantly affected all measures except RMSE. The field measure cannot be recommended as the interaction of pain history and task difficulty was not statistically significant.

DEDICATION

This is dedicated to my family, especially Tracy. Without your persistent support, mostly patient demeanor, and occasional waning excitement, I may not have completed this thesis. Thanks for sticking with me.

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There are numerous people to thank along the way, not only for their assistance in guiding this thesis reach to fruition, but also for inspiration, guidance, and mentorship, the foremost of which is Dr. Emily Wughalter. Without the impact Dr. Wughalter had on me the first time I entered the graduate program at San Jose State University, I may not have returned to the program over three years later to complete the degree. Her passion for teaching is evidenced by a unique ability to synthesize the available body of knowledge and inspire a diverse group of students to understand and learn, a characteristic unsurpassed by any of my previous teachers. My remaining committee members Dr. V. Gregory Payne and Dr. James C. Kao provided insight critical to a rigorous review of the literature, study design, and interpretation of the data. The expertise of Dr. Payne in motor development and Dr. Kao in biomechanics complemented the emphasis on motor learning in this study. The views provided by the members of the committee enhanced the multidisciplinary content important for the thesis to attain academic credibility.

The data may have not been collected if it were not for Dr. Gene Alexander and Chris Dyrby of the Stanford Biomotion Laboratory. When the force platform at San Jose State University failed to work, Dr. Alexander granted permission for my use of the Biomotion Laboratory. Chris Dyrby deserves extra thanks for taking the time to teach me how to use the force platform and data collection software. Chris was always available for questions and gave me access to the lab during times that were both critical and convenient for me and the study participants.

Thanks to Paul Chek for facilitating my initial critical thoughts on the connection between the transversus abdominis and postural control. If it wasn't for some early discussions between he and I my interest in motor learning and motor control may have been delayed. He continues to keep me thinking outside of my comfort zones.

Finally, I must thank all the participants who were kind enough to take the time to participate in the research and all the people who helped me find qualifying participants. The participant profile for this study was rigid, but most people seem to know someone with low back pain. My long-term hope is that this study can become part of the solution to the problem of low back pain.

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CHAPTER 1

Introduction

Spinal stability is controlled by passive, active, and neural subsystems that work interdependently to maintain stability of the spine (Panjabi, 1992a, 1992b). When one or more of the subsystems fails to function optimally, the body is at greater risk for injury due to the increased demands placed upon the other subsystems (Radebold, Cholewicki, Panjabi, & Patel, 2000). Clinical assessments for measuring deficits in the neural and deep active subsystem of the lumbar spine have only recently been designed (Richardson, Jull, Hodges, & Hides, 1999).

The deep active subsystem of the lumbar spine includes the transversus abdominus, multifidus, diaphragm, and the muscles of the pelvic floor, which are under control of the neural subsystem. Hodges and Richardson (1996) showed that in response to the initiation of movement, feedforward control of transversus abdominis is delayed in people with low back pain. Measured as motor activation times of less than 50 ms, a feedforward response is the anticipatory sending of information to prepare the motor system for future motor action. Richardson and Jull (1995) discussed the importance of retraining the neural subsystem as part of a rehabilitative program following an episode of back pain. They have shown that specific motor control exercises can be implemented to retrain the multifidus and transversus abdominis to contract at the appropriate time. Without the specific exercises, atrophy and temporal delay of the multifidus and transversus abdominis reduces the stabilization capacity at one or more levels of the lumbar spine. This temporal delay is thought to be a risk factor related to injury of the

lumbar spine (Hodges & Richardson, 1996, 1997a, 1999a; Radebold et al., 2000). Exercise programs emphasizing motor control retraining have shown lower recurrences of low back pain than exercise programs without specific retraining (Hides, Jull, & Richardson, 2001; O'Sullivan, Twomey, & Allison, 1997). Early detection of the temporal delay within the deep active subsystem and specific motor control exercises may contribute to rehabilitative outcomes and decrease the recurrence of low back pain.

The transversus abdominis has been shown to respond in a feedforward manner with movements of the lower limbs (Hodges & Richardson, 1997b) and upper limbs (Hodges, Cresswell, & Thorstensson, 1999, 2001; Hodges & Richardson, 1997a, 1999a, 1999b). Shoulder flexion causes a compensatory reactive force in the spine acting backward and downward on the center of mass equal in magnitude to the arm movement (Hodges & Richardson, 1999b) and is preceded by a feedforward response in the postural muscles (Bouisset & Zattara, 1981; Hodges & Richardson, 1997a). In response to the reactive forces generated by shoulder flexion, afferent feedback from the arm and proprioceptive information resulting from trunk motion influences the postural responses of the trunk muscles (Hodges et al., 2001). Postural responses as measured by increased center of pressure trajectories are observed when the feedforward control of the trunk musculature is absent, suggesting an inability to adequately stiffen the spine (Radebold, Cholewicki, Polzhofer, & Greene, 2001; Cholewicki, Polzhofer, & Radebold, 2000).

Hodges and Richardson (1999b) measured the timing of transversus abdominis contractions in participants with and without a history of low back pain while performing standing right shoulder flexion at fast (as rapidly as possible), intermediate (self-paced),

and slow (30° per second) movement speeds. The participants with a history of low back pain failed to elicit a feedforward response in the transversus abdominis during the intermediate and fast movement speeds. The failure of the transversus abdominis to activate in a feedforward manner in either group during the slow movement speed was suggested by Hodges et al. (1999b) to support the role of the transversus abdominis in controlling reactive forces and in the sufficiency of the passive viscoelastic properties of the tissues to maintain stability under small force perturbations. Further support has been provided by Aftab, Ishac, and Winter (2002); they found that during a shoulder flexion task the initial control of center of center of mass is due to passive forces.

The transversus abdominis contributes in a non task-specific way to trunk control (Hodges et al., 1999). There is invariant timing in the anticipatory postural contraction of the transverus abdominis when limb movement reaction time is varied in a choice reaction time shoulder flexion or shoulder abduction task; this is in contrast to the superficial abdominal muscles, which show varied timing of contraction as response expectation to limb movement varied (Hodges & Richardson, 1999a). The results of Hodges and Richardson provide support that the anticipatory activity of the transversus abdominis is generated in parallel with the command for movement, meaning its activation is initiated directly by the stimulus to move and independent of the motor command to move. Hodges' (2001) research provided additional support that in people with low back pain, the feedforward control of the transversus abdominis becomes increasingly delayed as task complexity increases, secondary to alterations in motor planning.

When the feedforward activation of the transversus abdominis is delayed, a mechanical model of spinal stability indicates the possibility of the normal degrees of freedom at each spinal joint increasing with the addition of shear, a non-physiological movement (Bergmark, 1989; Panjabi 1992a). Increased activation of the global muscles promotes rigidity, which is associated with a lack of movement, whereas increased activation of the local muscles promotes stiffness, which is associated with movement (Hodges, 2001). While static rigidity can be functionally important, the ability to dynamically accelerate and decelerate the joints of the spine with the proper amount of force production, absorption, and transference along with the proper timing is a complex task to coordinate. A spine can be rigid, yet still unstable at the segmental level, as the global musculature responsible for producing rigidity have no direct connections to the lumbar spine. The mechanical model however, is only one approach. A neural model suggesting alterations in motor programming has been supported by Hodges (2001), which interacts with the biomechanical model regarding the issue of coordination of joint movement and the temporal activation of the global, but more specifically the local muscles. Additionally, a theory based on dynamical systems suggests that as coordination improves there is a release in the degrees of freedom utilized by the body (Lee, 1998). Attaining the appropriate amount of stiffness within the local lumbar muscles at the appropriate time is considered an improvement in coordination (Bergmark). As improved coordination requires less motor planning, a freezing in the number of degrees of freedom used is observed in low skilled and complex motor planning activities (Bergmark; Panjabi 1992a). All the models describing control of

human movement support the interdependence of a feedforward-feedback system during skilled movements as discussed in a review by Lee (1998). A discussion of the various systems is beyond the scope of this review, but viewing the body as a purely mechanical system is too simplistic. The neural and dynamical systems theories take into consideration the complex and unpredictable functioning ubiquitous in living systems.

Skilled movements show task-specific flexibility in attaining a goal. If one part of the system is damaged, it is able to compensate without the need to reorganize a new movement plan providing that the biomechanical disturbance (amount, direction, speed, duration of load) or neural disturbance (task complexity, attentional focus, past experiences) does not exceed the capabilities of the system to compensate (Saltzman & Kelso, 1987). In people with low back pain, the delay in feedforward control of the deep active subsystem may produce or exacerbate an existing increase of the controllable degrees of freedom in the lumbar spine. This may be such a disturbance that the system is unable to compensate for without specific retraining and may present as a change in postural control mechanisms.

Postural control is defined as controlling the body's position in space for the purposes of stability and the relationship between the body segments and the environment for a given task (Shumway-Cook & Woollacott, 2001). As described by Radebold et al., (2001), variations in postural control are related to motor control dysfunction. Postural control has been quantified using center of pressure measurements to identify impairments in standing balance (Byl & Sinnot, 1991), neuromuscular disorders

(Shumway-Cook & Woollacott, 2001), and more recently as a measure of the neural control subsystem of the lumbar spine during unstable sitting (Cholewicki et al., 2000).

Postural control measurements have been able to differentiate between individuals with or without low back pain using upright stance posturography under varying sensory conditions (Byl & Sinnot, 1991), muscle response times during a quick force release test, and center of pressure measures during unstable sitting (Radebold et al., 2001). While standing requires greater neuromuscular control, upright stance does not allow for isolation of trunk control mechanisms as the joints of the lower body strongly influence postural control. Seated measurements of postural control have been examined to remove the proprioceptive influence of the lower body joints and to isolate control strategies of the trunk and higher centers (Cholewicki et al., 2000; Forssberg & Hirschfeld, 1994). Radebold et al., (2001) collected center of pressure (CoP) trajectories during unstable sitting following the same procedures established by Cholewicki et al. (2000) to examine trunk postural control and were able to differentiate between participants with or without low back pain.

Cholewicki et al. (2000) quantified the use of unstable sitting using summary statistics and PATH of CoP trajectories as a measure of postural control of the trunk and explained its applicability in examining postural control deficits of the lumbar spine in individuals with low back pain. Radebold et al. (2001) showed that individuals with low back pain had poorer postural control as shown by greater CoP differences at the same level of unstable sitting than individuals without low back pain. By attaching a variable diameter hemisphere to the bottom of a seat Radebold et al. (2001) increased task

difficulty using four levels of seat instability. The CoP measurements were gathered at each level of instability over five 7-second trials with eyes open and with eyes closed. Closing the eyes further increased the challenge to postural control. The third level of difficulty (44 cm hemisphere) with eyes closed showed the greatest differences in postural control between participants.

While Radebold et al. (2001) used changes in the visual system during the unstable sitting test to increase task difficulty, using a simple reaction time test of repeated slow and fast shoulder flexion to increase task difficulty, instead of changes in the visual system, may improve the sensitivity of determining motor control deficits by examining postural control. These deficits may be elucidated through the addition of reactive forces by the dynamic action of shoulder flexion (Hodges et al., 1999b), afferent feedback from the shoulder during shoulder flexion, and proprioceptive information from the trunk as it stabilizes the resultant reactive forces (Hodges et al., 2001). A 44 cm hemisphere attached to a seat was used to provide unstable sitting as it showed the greatest measurable differences in postural control between participants with and without low back pain (Radebold et al., 2001). The most difficult seat level (22 cm) in the study by Radebold et al. (2001) showed the greatest difference in postural control between participants with and without a low back pain, however the differences in postural control could not be measured as only 13% of the low back pain participants finished the test with eyes closed. Since the important role of vision on postural control (Vuillerme, Nougier, & Prieur, 2001) and the decrease in postural control observed when vision is obstructed (Byl & Sinnott, 1991; Radebold et al. 2001; Vuillerme et al. 2001) have been

determined, the dynamic action of slow or fast shoulder flexion instead of altering visual status may show differences in postural control between individuals with or without a history of low back pain. This information may help to further explain the mechanisms by which motor control of the lumbar spine is performed.

The purpose of this research was to experimentally examine if the level of task difficulty interacts with pain history during unstable sitting as indicated by postural control measurements. The practical purpose of this research was to design an ecologically valid measurement that can show differences in postural control between individuals with and without a history of low back pain. Expanding upon the work of Radebold et al. (2001), the experimental design of this study was to determine how the level of task difficulty during unstable sitting and history of pain affect postural control measures. Force plates are not readily available to most healthcare workers, coaches, trainers, and therapists, supporting the need for an easy to perform and accurate motor control measurement of the lumbar spine. In this study, an attempt was made to show that the level of task difficulty attained during the unstable sitting trials can be used as a measure of normal motor control of the lumbar spine. Using task difficulty to show differences in postural control requires only basic understanding of complex anatomy and integrated function of the lumbar spine and the central nervous system. Task difficulty has the potential to show the need for more thorough clinical testing, such as an evaluation by a healthcare worker trained at evaluating neuromuscular dysfunctions.

Problem Statement

The experimental design of this study was to determine:

1. How level of task difficulty during unstable sitting and pain history affect postural control measurements.

Hypotheses

The following null hypotheses were made for the experimental purpose of the study:

1. Task difficulty and history of back pain will not interact to affect CoP measures.
2. Task difficulty will not affect CoP measures.
3. History of back pain will not affect CoP measures.

Limitations

The study was limited to:

1. Unknown or unaccounted for variables to include past perceptual-motor skill experiences, motivation, or the influence of persisting pain on information processing may influence postural control.
2. Research in motor control regarding information processing and dynamic systems models is still unclear as to how feedforward and feedback mechanisms are organized (Lee, 1998; Shumway-Cook & Woollacott, 2001*)*

Delimitations

The study was delimited to:

1. Participants were selected by gender, weight, torso length, vestibular dysfunction, prior medical history, and between the ages of 30 and 50 years. Participants were divided equally by gender into males and female. Body weight and torso length were controlled for as Cholewicki et al. (2000) showed a correlation to postural control during unstable sitting. The important role of the vestibular system in postural control prevented individuals with vestibular dysfunction from participating. Medical history was reviewed to ensure participants met the criteria for participation and did not have any contraindicated medical conditions. An age range of 30 – 50 years was established to control for any potential variations is postural control secondary to changes in motor development across the lifespan, which may confound the reliability of the data. It was assumed that motor development would be similar within the specified age range. The participant information sheet (Appendix A) was used to qualify each participant.
2. In this study the lumbar spine was examined in isolation from the lower body. Since sensory information from the lower body is nearly eliminated during an unstable sitting test, the results of this study may not have generalizability to tasks that involve upright stance.
3. Low back pain participants were selected from those who have had persisting or periodic pain for longer than 6 months. The low back pain participants were free from neurological deficits, structural deformities, fractures, spinal stenosis, disc herniation, genetic spinal disorders, or previous spinal surgery, and screened by an

orthopedic surgeon or physiatrist within 3 months prior to their participation in this study as long as their symptoms had not changed since the visit.

4. Low back pain participants did not receive treatment and they were not involved in an exercise program that had changed in the type of exercise performed over the 3 months prior to their participation in the study.
5. Healthy control participants had no history of any neuromuscular or postural disorder, and never experienced low back pain lasting longer than three consecutive days.
6. None of the participants had any visual or vestibular disorders, and no type of injury that caused shoulder or neck pain during shoulder flexion of the dominant arm. Rapid shoulder flexion causes anticipatory neck muscle activity (Gurfinkel, Lipshits, & Lestienne, 1988), which may affect CoP measurements if pain inhibition of the neck musculature occurs.

Assumptions

1. All participants would be able to follow directions for correct testing.
2. Loss of balance as defined by grabbing onto the handrail during the test, or allowing the edge of the unstable platform to make contact with the force plate, is related to a loss of postural control.
3. Using an unstable surface is a valid and reliable method to increase dependence upon the spinal reflex pathways in the context of examining postural control (Shumway-Cook & Woollacott, 2001).

4. Shoulder flexion is a valid and reliable method to increase dependence upon spinal reflex and feedforward control pathways in the context of examining postural control (Hodges et al., 2001; Hodges & Richardson, 1999a, 1999b).

Definition of Terms

The following definitions were used to conceptualize and operationally define the study:

Postural control. Controlling the body's position in space for the dual purposes of stability and orientation (Shumway-Cook & Woollacott, 2001). The ability to remain on the unstable platform without grabbing onto the handrails or allowing an edge of the unstable platform to contact the support base, while the eyes remain fixed at a target placed at eye level and shoulder flexion is perfomed.

Postural Stability (Balance). Postural stability is the ability to maintain the center of mass within the limits of the base of support (Shumway-Cook & Woollacott, 2001). For the purpose of this study, balance was indicated by the ability to prevent the following from occurring once the testing had begun: grab onto the handrails, allow an edge of an unstable platform to make contact with the force plate, or use either arm to regain balance.

Center of Pressure. Center of pressure is the location of the vertical ground reaction force vector from a force platform. It is equal and opposite to a weighted average of all downward forces acting on a force plate (Winter, 1990). The maximum, minimum, absolute average, and the root mean square error of the distance traveled in antero-posterior and medio-lateral directions were analyzed, and the average distance

traveled per unit of time (PATH) was be determined by averaging the total forces measured in the antero-posterior and medio-lateral directions.

Healthy Participants. Healthy participants had no history of any neuromuscular, postural, visual, or vestibular disorder, and reported having never experienced back pain lasting longer than 3 consecutive days (Radebold et al., 2001). Also seen referred to in this study as participants with no history of low back pain, all participants must have met the criteria stated previously.

Low Back Pain. Persisting or periodic pain lasting longer than 6 months has been defined as chronic low back pain (Radebold et al., 2001). Participants were excluded if they ever had chronic, unremitting pain, neurological symptoms, pain extending beyond the gluteal fold, fractures, previous abdominal or spinal surgery, or recent pregnancy. For this study, participants must have had chronic low back pain meeting the previously stated criteria and not be experiencing pain at the time of the study.

Importance of the Study

Acute low back pain resolves in 2-4 weeks for the majority or people, yet 60-80% of them have a recurrence within the first year (Hides, Richardson, & Jull, 1996). There are many treatment strategies and theories on which treatment works best, yet the traditional model of strengthening the abdominal muscles shows little clinical benefit (Helewa, Goldsmith, Lee, Smythe, & Forwell, 1999). Recent approaches (Hides et al., 2001; O'Sullivan et al., 1997) utilized in orthopedic rehabilitation are connected to strong scientific basis and clinical success both acute and long-term. Unlike neurorehabilitation programs that work with people who may have had a cerebro-vascular accident or an

injury that presents with upper motor-neuron dysfunction, orthopedic rehabilitation has not typically incorporated specific motor control exercises (Richardson & Jull, 1995).

According to Taub, Uswatte, and Elbert (2002), the recent advances in neurorehabilitation are resulting in a paradigm shift in treatment strategies within physical rehabilitation programs. The motor control approach to treatment considers the temporal importance of the local musculature instead of what has been traditionally a biomechanical approach, emphasizing retraining a combination of flexibility, strength, and endurance of the global musculature (Richardson & Jull, 1995). Taub (2002) discussed how the temporal activation of the muscular system is skill dependent, which reinforces the importance of content and context specificity in rehabilitation program

With the existing knowledge base on the importance of motor control and how to retrain motor control dysfunction of the lumbar spine, an easy, reliable, and sensitive indicator that can differentiate between functional and dysfunctional motor control has not been developed. Following an approach similar to Radebold et al. (2001) the effect of task difficulty on postural control was examined. Instead of increasing task difficulty by altering vision, slow and fast shoulder flexion was used to create a dynamic and challenging condition. If significant differences in postural control between individuals with and without a history of low back pain can be shown during unstable sitting, a method for evaluating motor control deficits of the lumbar spine may be available for clinical and field use. This could potentially decrease injuries through the early detection of motor control deficits, as an injury prevention tool, a decrease in the recurrence of low back pain, and as a guide in rehabilitation programs.

Field measures are important for practitioners in order to have easy, valid, and reliable measures to guide decision making for treatment or training plans. A field measure needs to be easy so many people can be tested with a minimum amount of instruction, valid so it measures what it is supposed to measure, and reliable for repeatability of the measure. While field measures may lack specificity in quantifying a measure, their ability to quickly assess an individual has the potential to save both time and money.

CHAPTER 2

Review of Literature

This research experimentally examined if the level of task difficulty interacts with pain history during unstable sitting as indicated by postural control measurements. The practical purpose of this research was to design a field measure to show differences in postural control between individuals with and without a history of low back pain. This review of literature is divided according to the following subheadings: overview of functional anatomy and biomechanics; motor control and rehabilitation in low back pain; postural control and low back pain. A summary is provided at the end of the chapter.

Overview of Functional Anatomy and Biomechanics

Panjabi (1992a, 1992b) introduced a model for spinal stability in terms of three subsystems. The passive, active, and neural subsystems work interdependently to maintain stability of the spine. The passive system consists of osseous, articular, and ligamentous structures in their ability to control motion of the spine, both at the end-ranges and within the mid-range of movements (Bergmark, 1989, Panjabi, 1992a). The active system is made up of the muscles in the form of the actin and myosin cross-bridges serving a mechanical role in stability (Bergmark, 1989; Panjabi, 1992a). And the neural control system consists of the sensory feedback, spinal input, and cortical control (Bergmark, 1989; Panjabi, 1992a). When one or more of the subsystems is not functioning optimally, Panjabi (1992a, 1992b) stated that the other subsystems must compensate otherwise instability will occur. The excessive motion of instability is associated with injury, degenerative disc disease, and muscle weakness (Panjabi, 1992b).

The concept of stability has also been called the neutral zone (Panjabi, 1992a). When movement occurs outside of the neutral zone, stretch sensitive pain receptors may be activated and the state of clinical instability is the result (Panjabi, 1992a). There are six joints making up the low back, each joint having six degrees of freedom by virtue of three rotational planes and three translational axes, for a total of 36 degrees of freedom (Bergmark, 1989; Panjabi, 1992a). When instability occurs, the component of shear is added, increasing the movement possibilities at the joint (Bergmark, 1989; Panjabi, 1992a). The requirements of the active and neural system play an increasingly important role in spinal stability when the passive system can no longer maintain a neutral zone (Bergmark, 1989; Panjabi, 1992a).

The active subsystem can compensate for instability by increasing its stiffness, thereby decreasing the size of the neutral zone (Panjabi, 1992b). By decreasing the neutral zone, or by eliminating shear, repeated micro-trauma to the spine is minimized along with the demands on the neural system. The active subsystem can be thought of in two discrete layers, a global stabilizing system and a segmental stabilizing system (Bergmark, 1989; Panjabi, 1992a). The global system consists primarily of the rectus abdominis, external oblique, anterior fibers of the internal oblique, lateral fibers of the quadratus lumborum, and the iliocostalis and longissimus (Bergmark, 1989). These muscles consist of primarily Type II phasic motor units and are responsible for gross movements of the spine (Bergmark, 1989). This global system of superficial spinal muscles is also used to transfer load between the pelvis and the thorax, and to balance

external loads imposed upon the body to spare the segmental system and the load upon the spine (Bergmark, 1989).

The segmental stabilizing system of muscles has origins or attachments directly or indirectly at the lumbar spine. The segmental system controls spinal stiffness, individual joint motion, and the position of the lumbar spine (Bergmark, 1989). The Type I tonic motor unit stabilizer system consists of the multifidus, transversus abdominis, posterior fibers of the internal oblique, and the medial fibers of the quadratus lumborum (Richardson et al., 1999). The multifidus, because of its fiber orientation, is at the best mechanical advantage for resisting shear forces (Richardson et al., 1999). Additionally, the fibers of the transversus abdominis course medially and inferiorly, blending with fibers of the internal oblique and the fascia of the abdominal wall, creating a complement to the multifidus in its ability to resist anterior shear forces at L4-5 and L5-S1 (Richardson et al., 1999).

As structure dictates function, the normal lordosis of the lumbar spine creates the greatest shear forces at the L4-5 and L5-S1 vertebra. When the transversus abdominis contracts it creates a hollowing of the abdominal wall, increasing intra-abdominal pressure and tension through the thoracolumbar fascia (Richardson et al., 1999). Intra-abdominal pressure adds to the stiffness of the spine via the contraction of the transversus abdominis, but only if the pelvic floor from below and the diaphragm from above are functioning properly (Hodges, Butler, McKenzie, & Gandevia, 1997; Hodges, Gandevia, & Richardson, 1997). In addition, as the multifidus contract they increase passive resistance to movement by expanding within their fascial sheath that blends into the

thoracolumbar fascia (Richardson et al., 1999). The transversus abdominis and multifidus contract together to create active stiffness of the spine, while tensioning of the thoracolumbar fascia increases passive tension. Working together under control by the neural system, the active and passive systems influence the intra-abdominal pressure mechanism to ensure a stiff spine (Bergmark, 1989).

McGill (2001) has discussed the importance of spinal stabilization in the prevention of injury and in rehabilitation, reinforcing the concept by describing *in vitro* research of the vertebral column that showed buckling of the spine with only 20 pounds of load when joint rotation was induced. Chiang and Potvin (2001) and Huang, Andersson, and Thorstensson (2001) have shown the important role of the abdominal muscles, rather than the dorsal muscles in maintaining stability of the spine during lateral bending with and without load. The increased activation of the transversus abdominis with increased lateral flexion and load further supports its role in spinal stability through its influence on intra-abdominal pressure.

The spine by itself is inherently unstable. The control of spinal stability is highly dependent upon the ability of the segmental system to control joint motion while the global system dissipates external loads and transfers internal forces (Richardson et al., 1999). Understanding the anatomy and biomechanics of the lumbar spine is important to understand the integrated influence of central commands upon muscle control.

Motor Control and Rehabilitation in Low Back Pain

Hides, Stokes, Saide, Jull and Cooper (1994) examined the effect of low back pain on the size of the lumbar multifidus. They studied 26 participants with unilateral

low back pain symptoms and 52 healthy participants were examined. The cross-sectional area of the multifidus at each level of the lumbar spine was viewed by real-time ultrasound and compared to a manual evaluation of segmental instability. There was evident wasting of the multifidus on the ipsilateral side that corresponded to the vertebral level identified as being unstable in the participants with low back pain. The control participants showed no differences. The average between-side difference in low back pain participants was 31 +/- 8% (14%-46%), and in the control group was 3 +/- 4% (0-17%). There was not a correlation between the amount of wasting and the severity of the symptoms.

Hides et al. (1994) have suggested that rapid multifidus wasting with low back pain was due to inhibition from perceived pain via a long loop reflex pathway that has a protective role to prevent movement and subsequently caused a negative metabolic effect and instability. Disuse atrophy was ruled out as a causative factor since the wasting of the multifidus was localized instead of a reduction of muscle size throughout the length of the muscle and over different levels. Results indicated that multifidus recovery is not automatic after resolution of low back pain (Hides et al., 1996). These researchers proposed that the most likely mechanism for a decrease in muscle size was through reflex inhibition. Reflex inhibition occurs through activation of the nocioceptive pathway or through non-painful, but inflamed tissue. Nocioceptors are pain receptors within the joint capsule and are usually silent but respond to stretch following inflammation and hamper alpha motor-neuron activity at the anterior horn of the spinal cord (Arendt-Nielsen, Graven-Nielsen, Svarrer, & Svensson, 1995). In the absence of pain, reflex inhibition

may be due to the influence of the gamma motor system in inflamed joints (Arendt-Nielsen et al., 1995).

The transversus abdominis is controlled independently of the motor command for limb movement (Hodges & Richardson, 1999a). A rapid movement of the arm or lower limb has been shown in non-low back pain participants to be preceded by activation of the trunk muscles, with the transversus abdominis being the earliest and its contraction being non-direction specific (Hodges & Richardson, 1996, 1997a, 1997b, 1999a). In participants with low back pain the contraction of the transversus abdominis shows a significant delay and its onset varies with the direction of force (Hodges & Richardson, 1996, 1999b).

Hodges and Richardson (1997a) had participants perform rapid shoulder flexion, abduction, and extension in response to a visual stimulus while electromyographic activity of the transversus abdominus, multifidus, internal and external oblique, and the deltoid was gathered. Activity of transversus abdominis in the low back pain participants was delayed until after activity of the deltoid, compared to the tranversus abdominus of the participants without low back pain which showed activity prior to the deltoid, independent of the direction of limb movement.

Hodges and Richardson (1999a) examined the effects of low back pain on muscle recruitment patterns during upper limb movement at slow (30° per second), intermediate (self-directed), or fast (fast as possible) speeds. Neither the healthy or low back pain individuals showed a feedforward contraction of the transversus abdominis during the slow shoulder flexion; however, the transversus abdominis contracted prior to movement

of the upper limb during the intermediate and fast shoulder flexion for the healthy but not the low back pain individuals. Hodges and Richardson (1999a) suspected that the slow shoulder flexion did not create enough challenge to the stability of the spine for the central command to warrant a feedforward activation of the transversus abdominis. Proprioceptive input to the spine from the upper limb was responsible for the increased activation of the transversus abdominis and coincided with observed movements of the trunk. This makes sense when considering the primary role of the transversus abdominis is to increase intra-abdominal pressure and tensioning of the thoracolumbar fascia to increase the stiffness of the spine, and opposing instead of creating reactive forces.

Hodges et al. (2001) found that with the addition of a load to the upper limb during movement a short-latency (50 ms) response of the erector spinae and transversus abdominis occurs. These findings support the notion that while preparatory activation of the transversus abdominis is not reflexively mediated and must be preprogrammed by the central nervous system, complex postural responses occur due to the dynamic environment to which the spine is exposed. During a shoulder flexion task the initial control of center of center of mass is due to passive forces (Aftab et al., 2002). Active control of postural center of mass occurs at about 200 ms following the acceleration of the arms. Anticipatory muscle activity seen during voluntary movement is not to control center of mass but segmental stability. The interplay between active and passive control is seen in the timing of change in hip moment about 30 ms after the onset of shoulder moment to minimize the effects of passive control of center of mass while ensuring trunk stabilization.

Dynamics system theory suggests that as coordination improves there is a release in the degrees of freedom utilized by the body (Lee, 1989). Attaining the appropriate amount of stiffness within the local lumbar muscles at the appropriate time is considered an improvement in coordination (Bergmark, 1989). As improved coordination requires less motor planning, a freezing in the number of degrees of freedom used is observed in low skilled and complex motor planning activities (Bergmark; Panjabi 1992a). Research examining the control of human movement supports the interdependence of a feedforward-feedback system during skilled movements (Lee, 1998). Skilled movements show task-specific flexibility in attaining a goal. If one part of the system is damaged it is able to compensate without the need to reorganize a new movement plan providing that the biomechanical disturbance (amount, direction, speed, duration of load) or neural disturbance (task complexity, attentional focus, past experiences) does not exceed the capabilities of the system to compensate (Saltzman & Kelso, 1987). The delay in feedforward control and a subsequent increase of the controllable degrees of freedom in low back pain may be such a disturbance that the system is unable to compensate for without specific retraining.

Deficits in lumbar proprioception have been found in individuals with low back pain (Gill & Callaghan, 1998), which may delay the timing of muscle activity to counteract the reactive forces generated by shoulder flexion, especially as the speed of shoulder flexion increases. Vibration induced muscle spindle stimulus (Brumagne, Lysens, Swinnen, & Verschuren, 1999) and lumbar fatigue (Taimela, Kankaanpaa, & Luoto, 1999) have been able to provide support for the important role of the lumbar

multifidus muscle spindle, as opposed to the joint capsule, in accurate position sense of the lumbar spine.

Contraction of the diaphragm during rapid shoulder flexion, independent of the phase of respiration, occurs 20 ms prior to the onset of the prime mover (Hodges et al., 1997). The anticipatory contraction of the diaphragm is coincided with that of the transversus abdominis and supports the intricate role of the diaphragm in spinal stability through an increase in intra-abdominal pressure. The transversus abdominis shows an earlier activation time during respiratory exhalation, compared to respiratory inhalation (Hodges et al., 1997). While the reasons for earlier activation are not known, Hodges et al. (1997) suspected it is due to dynamic modification of intra-abdominal pressure to maintain spinal stability.

Rehabilitation of the low back musculature has traditionally been thought of in biomechanical terms, emphasizing functional qualities like flexibility, strength, and endurance of the global muscles (Helewa et al., 1999; Richardson et al., 1999). To work with the body in a purely mechanical manner ignores the importance of the motor control system. Training individuals in sports and occupations that require the lifting of heavy loads or consisting of fast, ballistic movements should consider the effects of motor control on muscle function and fatigue (Ng & Richardson, 1990; Richardson & Bullock, 1986; Richardson and Jull, 1995; St. Clair Gibson, Lambert, & Noakes, 2001). High-speed repetitive jump training improves peak torque of the gastrocnemius, but decreases the strength of the soleus (Ng & Richardson, 1990). This is in agreement with the findings of Richardson and Bullock (1986) who showed that fast alternating flexion-

extension exercise of the knee joint facilitated improvements in the phasic musculature and inhibited the tonic musculature of the knee. Fatigue has mostly been explained by peripheral mechanisms (St.Clair-Gibson et al., 2001). Motor cortex and efferent pathways appear to limit activity output so that maximal muscle capacity is never utilized to ensure that no body system is stressed beyond its safety capacity (St. Clair Gibson et al., 2001).

The use of labile surfaces to increase sensorimotor stimulation has been used to unconsciously train the coordinated activation of previously inhibited muscles (Bullock-Saxton, Janda, & Bullock, 1993). Miller and Medeiros (1987) found that using multi-sensory kinesthetic cueing for the lower abdominal muscles resulted in greater EMG activity of the internal obliques and transversus abdominis during a slow eccentric phase of a curl-up exercise with the feet unsupported. Vera-Garcia, Grenier, and McGill (2000) examined EMG activity of the abdominal muscles during curl-ups on both stable and labile surfaces. Abdominal muscle activity increased in proportion to the greater demand for stability, with the external obliques showing the greatest increase. Vera-Garcia et al. (2000) suggested that utilizing labile surfaces increases the demands on the motor-control system, which may be desirable.

Low back extensor exercises are often utilized in the rehabilitation or prevention of low back pain. The goal is typically to improve the functional characteristics such as strength, endurance, and flexibility of the tissues while limiting shearing and compressive forces through the spine (McGill, 2001). The facet joints limit passive extension of the spine, whereas the annulus fibrosis or posterior longitudinal ligament resists passive

flexion. Richardson (1995) discussed the importance of considering motor control rehabilitation prior to improving the functional characteristics of the muscles.

Hides et al. (1996) investigated recovery of the multifidus following a first episode of low back pain. They recruited 41 participants with their first episode of low back pain occurring within three weeks of participation in the study were randomly assigned to a control or treatment group. The treatment group performed specific exercise therapy aimed at improving the control of the transversus abdominis with co-contraction of the multifidus. After four weeks, the specific exercise therapy group showed more complete multifidus recovery than the control group. The change from week 4 to week 10 was negligible. All of the participants had resolution of their pain, and there were no changes between groups for the outcome measures of pain, disability, or range of motion. Hides et al. (1996) have shown that multifidus recovery is not automatic after an initial bout of low back pain, which is a factor that contributes to the high recurrence of low back pain.

O'Sullivan et al. (1997) examined the influence of specific exercises on participants with chronic low back pain due to spondylolysis or spondylolisthesis. Participants were randomly placed into a control group or a specific exercise group. The control group went through 10 weeks of treatment as directed by their medical practitioner. The specific exercise group underwent 10 weeks of treatment training them to specifically contract the deep abdominal muscles with co-activation of the multifidus independent of the global muscles. The exercises were performed with slow, precise, low levels of maximal voluntary contraction. The activation of the transversus abdominis and

multifidus was finally progressed into upright functional tasks. The specific exercise group showed a decrease in pain and disability that was maintained at a 30-month follow-up, and the control group showed no change.

Back extensor strength training alone increases postural control efforts while balance skill training increases postural stability (Kollmitzer, Erbenbichler, Sabo, Kerchan, & Bochdansky, 2000). Kollmitzer et al. (2000) evaluated 26 healthy individuals after 1 month of either back extensor training or balance skill training, at which time they switched groups for an additional month of training. The participants were randomly assigned to a group and were tested by posturography, balance skill, and isometric maximal voluntary contraction of the back extensors prior to training, following the first month of training, and after the second month of training. Significant improvements were observed in both groups for all measures at the end of the training, but it was the balance skill-training group who showed a reduction in postural sway compared to the strength training group. While improving endurance of the low back muscles is important during the rehabilitation of low back pain (Allan & Waddell, 1989), the addition of sensorimotor skill training appears to show the need for an integrated approach to the treatment of low back pain and further research in the realm of motor control evaluation and rehabilitation.

Lumbar flexion sustained for 20 minutes induces changes in the viscoelastic structures of the lower back resulting in a 68% loss in tension of the lumbar multifidus (Jackson, Slomonow, Zhou, Baratta, & Harris, (2001). A 36% recovery of the tension occurred within the first 10 minutes of rest, but even after 7 hours the multifidus had only

recovered to 79% of its original value (Jackson, et al.). Reaction time to a load has also been shown to increase in the presence of lumbar fatigue, expectation, and vibration (Wilder, Aleksiev, Magnusson, Pope, Spratt, & Goel, 1996). In sports or activities that require sustained flexion or repetitive vibration, such as cycling, rowing, sitting on the bench waiting to play basketball, driving, and using a jackhammer, a reduction in the tension capabilities may leave the spine vulnerable to injury.

In the present study the complex and interdependent nature of lumbar spine control mechanisms were examined. Independent of the strength or endurance capacity of the trunk muscles, if there is a timing error in the contraction of the transversus abdominis or in the co-contraction of the transversus abdominis and multifidus, stiffness of the spine is reduced. Decreased stiffness may potentially increase risk for re-injury by leaving the spine vulnerable during the time it takes for the deep abdominal wall to contract. This study provides an impetus for continuing to examine what influences motor control, compensations in the presence of motor control deficits, and methods of restoring adequate motor control.

Postural Control and Low Back Pain

Reaction time and postural control are reduced in people with low back pain (Luoto, Taimela, Hurri, Aalto, Pyykko, & Alaranta, 1996). A reduction in reaction time may contribute to the development of low back pain. Postural control is partly maintained by feedback mechanisms. The receptors for feedback are eyes, ears, Golgi tendon organs, muscle spindles, joint receptors, and touch receptors. The information from the receptors is combined in the central nervous system to analyze the postural state.

The results of Luoto, Aalto, Taimela, Hurri, Pyykko, and Alaranta (1998) based upon one-footed and externally disturbed two-footed postural control between healthy and low back pain participants concluded that noncortical reasons for impairment appear to be more important than cortical reasons, which are in contrast with Hodges and Richardson (1996) who believed that activation of the transversus abdominis has less to do with excitability at the cord level and more to do with the processing strategies of the brain. In a follow-up study Luoto, Taimela, Hurri, and Alaranta (1999) showed that chronic low back pain impairs short-term memory, which decreases reaction time. While the measurement methods were indirect, the study did expose the deleterious effect of chronic low back pain on information processing, which is a cortical task.

To get more specific in their testing of the transversus abdominis, Hodges and Richardson (1997) examined the influence of a weight shift to single limb and contralateral hip movement on onset of trunk muscle contraction. Since activity of the transversus abdominis and multifidus occurred less than 50 ms after electromyographic (EMG) activity of the prime movers, it was determined that the transversus abdominis and multifidus cannot be reflexively mediated and must be under control of the central nervous system in a feedforward manner. In the presence of low back pain the contraction of the transversus abdominis becomes phasic.

Radebold et al. (2000) exerted isometric contractions in trunk flexion, extension, and lateral bending to seated participants. Compared to healthy participants, those with low back pain demonstrated longer reaction times to the antagonist muscle switching off and the agonist muscle switching on. The lack of a fast reflex may be due to damaged

proprioceptors or as a compensation strategy to stabilize the spine (Radebold et al., 2000).

To examine if the delay in transversus abdominis activation in people with low back was due to changes in motor planning or in delayed transmission of the descending motor command Hodges (2001) had participants perform a choice reaction time rapid shoulder flexion or abduction. Electromyography of the deltoid, superficial abdominal muscles, and the transversus abdominis was measured. The control group exhibited increased reaction times of the deltoid and superficial abdominal muscles, but not of the transversus abdominis as task difficulty increased. The reaction time of the tansversus abdominis along with the deltoid and superficial abdominal muscles increased in the participants with low back pain as task difficulty increased, supporting the notion that the delay is due to changes in motor planning. The changes in motor planning are suspected to be caused by fear avoidance or attentional focus (Hodges, 2001).

Many of the studies utilized surface EMG as their measurement of trunk musculature activation (Luoto et al., 1998; Luoto et al., 1996; Radebold et al., 2000). Based upon prior research (Hides et al.; 1996; Hides et al., 1994; Hodges & Richardson, 1997) the deep abdominal wall muscles that cannot be measured by surface EMG appear to be more important than the global system in protecting the spine. The global muscles histologically are not made for endurance. In the presence of spinal dysfunction the global muscles appear to substitute over the impaired segmental system, which is an altered pattern of motor control between trunk synergists (O'Sullivan et al., 1997). Radebold et al. (2000) suggested that longer reaction times to switching on and off may

be a compensation strategy, but that increased information processing demands are suspect (Hodges, 2001).

Even though testing balance, as Luoto et al. (1996, 1998) have proposed, shows usefulness in examining differences between healthy and low back pain individuals, and in judging the effectiveness of a treatment program, the results must be judiciously applied. As shown by Hides et al. (1996) the multifidus does not automatically recover following back pain. Even in participants who no longer had pain, multifidus recovery did not occur. The multifidus appear to be influenced be reflex inhibition (Hides et al., 1996), whereas the transversus abdominis appears to be influenced by a long loop pain inhibitory mechanism (Richardson et al., 1999). Although the importance of proprioception as explained by Luoto et al. (1996) cannot be overlooked, the first place to begin for rehabilitation and prevention of recurrence of low back pain may be in recovery of the multifidus and transversus abdominis as proposed by Richardson et al. (1999).

Standing postural control is altered in individuals with low back pain (Byl & Sinnott, 1991). Body sway was analyzed under various sensory conditions using force plate stabilometry. The test conditions were designed to test different sensory condition by combining eyes open or eyes closed (visual information – brain stem reflex pathways), stable or unstable surface with one or two-footed stance (somatosensory information – spinal reflex pathways), and head still or moving (vestibular information – brain stem reflex pathway). Body sway increased 6-700% depending upon the test condition, and the low back pain group showed significantly greater body sway than the healthy control group during the one-footed and eyes closed test conditions.

Forssberg and Hirschfeld (1994) studied postural control in sitting with surface EMG. A seated posture was chosen to eliminate the influence of the joints of the lower body and to isolate control strategies of the trunk and higher control centers. A moveable platform induced a perturbation to the participant and muscle activation patterns were measured, which does not allow quantification of the deep abdominal muscles, including the transversus abdominis. The study by Forssberg and Hirschfield provided support that control of posture is influenced by a multi-sensory input from the hip, neck, and head, including visual, vestibular, and somatosensory signals. The participants were tested in a sitting position with their legs extended in front of them. Differences in hip flexion mobility may have positioned the lumbar spine in a flexed position, which would alter the prorioceptive afferent feedback influencing postural control.

Postural control has also been quantified using CoP measurements during unstable sitting with eyes-open or eyes-closed (Cholewicki et al., 2000) and has been shown to differentiate between individuals with or without low back pain (Radebold et al., 2001). The studies by Cholewicki et al. (2000) and Radebold et al. (2001) are unique in their design in that they remove the dominant influence the lower body has on postural control by sitting the participants on a specially designed chair with footrests. Creating an unstable platform by attaching hemispheres of varying diameters to the bottom of the chair, Cholewicki et al. (2000) were able to quantify postural control of the lumbar spine. Their idea, which is plausible, is that isolating the lumbar spine from the lower body allowed them to more directly assess motor control of the lumbar spine. They also performed a random walk analysis, which is described in detail by Collins and De Luca

(1993). A random walk analysis takes into account the maximum and the mean CoP trajectories, and applies an equation to determine the temporal ordering of a series of CoP coordinates (Collins & De Luca, 1993). Plotting the temporal ordering of the coordinates was thought to allow them to observe differences between open-loop and closed-loop mechanisms of postural control. While the method appears promising, reproducibility of the measurements has not been established.

Radebold et al. (2001) were able to show that individuals with chronic low back pain, when compared to healthy controls, had significantly greater center of pressure measurements during unstable sitting using a 44 cm hemisphere. All the healthy control participants finished the most difficult seat instability level (22 cm) with eyes open, and 71% finished with eyes closed. Only 69% of the low back pain participants finished the most difficult seat level with eyes open and only 13% with eyes closed, which Radebold et al. (2001) considered due to the low back pain participants stronger dependence on visual feedback. The ability of the visual system to compensate for challenges in postural control during lower limb fatigue or in the presence of proprioceptive deficits underscores its valuable role in postural control (Vuillerme et al., 2001). Removing vision during a task has a destabilizing effect that will be amplified by other sensory deficits (Vuillerme et al.).

Most healthcare workers, coaches, trainers, and therapists do not have immediate access to a force plate to perform center of pressure measurements. Because of this limitation, an easy to perform test needs to be developed. A pressure biofeedback unit has shown to quantify motor control deficits of the transversus abdominis and

differentiate between individuals with or without low back pain in 80% of the trials (Richardson et al., 1999). Subsequently, Cairns, Harrison, and Wright, (2000) have shown good intra-tester reliability using the pressure biofeedback unit, but they were only able to differentiate between healthy and low back pain individuals in 68% of the trials. A major limitation of the pressure biofeedback unit is that is requires a high level of clinical skill that renders it inadequate for use by many health care workers, trainers, and coaches on an individual or group-testing basis.

Using measurements of postural control and surface EMG to show deficiencies in motor control is a challenging task. Although direct measurement of the transversus abdominis with indwelling electrodes appears to be the most valid way to test for its neural response (Hodges & Richardson, 1996), the method is not practical in most clinical situations. Cairns et al. (2000) and Richardson et al. (1999) have shown the pressure biofeedback to be a reliable quantification of motor control deficits, but the skill level of the clinician performing the test limits its potential. Cholewicki et al. (2000) and Radebold et al. (2001) have shown that measurements of postural control during unstable sitting can help isolate motor control deficits of the lumbar spine.

Summary

The control of spinal stability is highly dependent upon the ability of the segmental system to control joint motion while the global system dissipates external loads and transfers internal forces (Richardson et al., 1999). The segmental system is comprised primarily of the transversus abdominis and the multifidus (Bergmark, 1989). The transversus abdominus acts in a feedforward manner and has been shown to respond

prior to movement of the upper and lower limbs, independent of the direction of movement (Hodges and Richardson, 1996, 1997a, 1997b, 1999a). Reaction time and postural control are reduced in people with low back pain (Luoto, Taimela, Hurri, Aalto, Pyykko, & Alaranta, 1996). A reduction in reaction time may contribute to the development of low back pain. Independent of the strength or endurance capacity of the trunk muscles, if there is a timing error in the contraction of the transversus abdominis or in the co-contraction of the transversus abdominis and multifidus, stiffness of the spine is reduced. Decreased stiffness may potentially increase risk for re-injury by leaving the spine vulnerable during the time it takes for the deep abdominal wall to contract. Rehabilitation programs that emphasize motor control retraining of the transversus abdominis and multifidus for the treatment of low back pain continuing to show lower levels of recurrence compared to standard treatment (O'Sullivan, et al., 1997; Hides et al., 2001),

This study provides an impetus for continuing to examine what influences motor control, compensations in the presence of motor control deficits, and methods of restoring adequate motor control. Using measurements of postural control and surface EMG to show deficiencies in motor control is a challenging task. Although direct measurement of the transversus abdominis with indwelling electrodes appears to be the most valid way to test for its neural response (Hodges & Richardson, 1996), the method is not practical in most clinical situations. Cairns et al. (2000) and Richardson et al. (1999) have shown the pressure biofeedback to be a reliable quantification of motor control deficits, but the skill level of the clinician performing the test limits its potential. Cholewicki et al. (2000) and

Radebold et al. (2001) have shown that measurements of postural control during unstable sitting can help isolate motor control deficits of the lumbar spine.

Allan and Waddell (1989) stated that back pain is not a new health problem, but the perceptions and management of back pain have created a disability mentality in individuals with back pain. Prior studies of postural control during unstable sitting (Cholewicki et al., 2000; Radebold et al., 2001) have shown that people with low back pain have diminished postural control. Continued examination into the motor control strategies of the lumbar spine will benefit practitioners in the treatment of low back pain.

Since most activities are dynamic in nature, examining the influence of shoulder flexion - a dynamic activity, on postural control is important. By examining the influence of somatosensory information, the afferent and proprioceptive feedback of shoulder flexion, and task difficulty through slow and fast flexion (Hodges and Richardson, 1996, 1997a, 1997b, 1999a), a more sensitive measurement of motor control deficits of the lumbar spine may be elucidated. If the level of task difficulty significantly interacts with pain history, the test may be applicable to individuals who do not have access to a force plate to assess motor control of the lumbar spine. The ability to assess motor control of the lumbar spine without a force plate can show the need for more sensitive testing, motor control retraining to reduce the risk of injury or recurrence of low back pain, or baseline values for return to work or sport.

CHAPTER 3

Methods

This research experimentally examined if the level of task difficulty interacts with pain history during unstable sitting as indicated by postural control measurements. The practical purpose of this research was to design a field measure to show differences in postural control between individuals with and without a history of low back pain. In this chapter the methodology and statistical analysis used in the study are discussed. This chapter is divided according to the following subheadings: participants, apparatus, procedures, design, and analysis of data.

Participants

Participants in the study were matched within and between groups by sex, age, weight, and torso length. The age range was set at between 30 and 50 years. Cholewicki et al. (2000) showed low correlation of age (37 ± 10 years) on PATH. Torso length was measured from the ninth thoracic spinous process to the fifth lumbar spinous process (T9-L5). Cholewicki et al. (2000) found a positive and moderate correlation for T9-L5 distance and weight on PATH.

A total of 24 participants were assigned to either a history of low back pain group (6 males, 6 females, M = 36.8 years, SD = 7.32, M = 169 cm, SD = 5.57, M = 64.3 kg, SD = 14.2, M = 22.0 cm, SD = 2.37) or control group (6 males, 6 females, M = 39.1 years, SD = 6.92, M = 171 cm, SD = 8.21, M = 67.6 kg, SD = 14.9, M = 22.1 cm, SD = 3.31). (see Table 14). Previous studies of postural control during unstable sitting used 11 participants (Cholewicki et al., 2000) and 30 participants (16 low back pain group and 14

control group) (Radebold et al., 2001). Participants in the history of low back pain group were recruited from local orthopedic medical centers, physical therapy clinics, and by convenience sampling, while participants in the no history of low back pain group were recruited by convenience sampling from the local community. The physicians at a local orthopedic center and the physical therapists at a local clinic known by the researcher were told about the study and asked to refer any patients who fit the participant profile. The physicians of any participants referred by the physical therapists were contacted to ensure that the participants met the criteria to participate in the study.

To be included in the study low back pain participants must have had persisting or periodic pain lasting longer than six months, must have been pain free at the time of testing, and not taking any pain relieving medication. The participants must have been medically screened within three months prior to the study to exclude any symptoms with a musculoskeletal etiology. Participants were excluded if they had chronic, unremitting pain, neurological symptoms, pain extending beyond the gluteal fold, fractures, previous abdominal or spinal surgery, or recent pregnancy. Low back pain participants could not be in treatment or performing exercises that emphasized retraining of motor control deficits at the time of the study. If participants were receiving treatment, their rehabilitative program was reviewed with the treating practitioner to determine if motor control retraining exercises, as described by Richardson et al., (1999) were being performed. This was to ensure that any retraining effect of the motor control exercises on the temporal activation of the local musculature has not been enhanced as this could potentially confound the data.

Healthy control participants must have had no history of any neuromuscular, postural, visual, or vestibular disorder, and had never experienced back pain lasting longer than three consecutive days and for which they sought medical intervention. Any pain lasting three days or less was probably due to muscular trauma and would not have any lasting effect upon motor control (Radebold et al., 2001). None of the participants could experience pain in the shoulder or neck during shoulder flexion of the dominant arm.

Apparatus

A special seat equipped with leg and foot supports (Appendix B) prevented lower body movement. The foot support was adjustable to create a 90-degree hip and knee angle for all participants. An aluminum hemisphere 44 cm in diameter was attached to the bottom of the seat, which was placed on an aluminum crate 36 inches high. The top and bottom of the crate were 15.5 inches by 23.5 inches and attached at the corners by four 1-inch square aluminum tubes. The top of the crate had arm supports to provide stability between trials and to allow the participants to stabilize themselves if the felt they were going to fall. The unstable platform sat on top of the aluminum crate, which in turn sat upon a Bertec force plate (Columbus, Ohio, Model 4060-10), which was placed in a solid and stable floor. Postural sway was evaluated using force plate measurements of center of pressure displacements. As used by Radebold et al. (2001), center of pressure coordinates were recorded at 1600 Hz and low-pass filtered at 10 Hz using a fourth order Butterworth digital filter to eliminate noise.

Procedures

Prior to collection of data, the study was approved by the San Jose State University Human Subjects Institutional Review Board for the use of human participants (Appendix C). The purpose and procedures of the study were explained to each participant. Prior to enrollment as participants, the volunteers were able to ask questions about the study. All participants read and signed a written agreement to participate in research form approved by the university before beginning the study (Appendix D). Each participant in the study was assigned a code, for purposes of confidential recording. The primary researcher was the only person with knowledge of participant names and numbers. Participants were asked to wear shorts. Males were asked to wear tank tops or be topless; and females were asked to wear t-shirts, tank tops, or sport bras. The participants had their choice, but clothing could not restrict freedom of arm movement nor be too large whereby it may get caught in the equipment or influence shoulder flexion. No shoes or socks were allowed.

The procedures and the data collected were similar to those of Radebold et al. (2001). The test consisted of trying to maintain balance while sitting on an unstable platform during slow and fast shoulder flexion trials. Prior to testing, the examiner completed a participant information sheet to obtain information on sex, age, height, weight, T9-L5 distance, arm dominance, low back pain history, and corrective lens use (Appendix A). Arm dominance was determined by a question on the participant information sheet asking participants which arm would be used to throw a ball. If the participant used corrective lenses for anything other than reading they must have been

worn while performing the test. Prior to testing, the force plate was calibrated. The same procedures were followed for each participant.

After reading and signing the completed participant information sheet, each participant was assigned a number. To maintain consistent and accurate instructions, an audiotape was made (Appendix E) to familiarize participants with the testing procedure and to inform them by voice and tone as to the trial they would be performing, when to begin, and when to rest. The number of trials and the amount of rest time was made clear to the participants. Once the testing began if they grabbed onto the handrails, allowed an edge of the unstable platform to make contact with the force plate, or used either of their arms to regain balance, the trial would be stopped. Participants were allowed one re-trial at each level of difficulty before the testing was finished and a "not passed" was recorded for the given trial. A "not passed" indicated that the participant was not tested on any greater task difficulty, and would be scored a zero. A passing score following a trial was scored as a one.

Prior to sitting on the unstable platform, participants were shown and practiced 10 repetitions of slow and fast shoulder flexion defined by Hodges and Richardson (1999) as 30° per second for slow shoulder flexion and as fast as possible for fast shoulder flexion. Shoulder flexion began with the arm at the side and was raised in the saggital plane 90°. Participants were told to look at a visual cue aligned to eye level on a wall at the same distance for each participant. Participants sat in the center of the unstable platform and the investigator adjusted the footplate to have a 90-degree angle at the hip and the knee. A trial consisted of slow or fast shoulder flexion performed for 10 seconds. Each

participant performed three 10-second trials at each speed of shoulder flexion with 30 seconds of recovery between trials. One 10-second practice was allowed prior to each trial. Participants were asked to hold onto the support rails between trials to limit any additional learning and to prevent fatigue. The sequence of trials was counterbalanced such that six participants from each group performed slow shoulder flexion followed by fast shoulder flexion, while six participants from each group performed fast shoulder flexion followed by slow shoulder flexion.

Design

The three independent variables in the study were pain history, task difficulty, and trial. Pain history and task difficulty were measured as dichotomous variables, and trial had three levels. The pain history variable consisted of a history of low back pain and a no history of low back pain group. The task difficulty variable consisted of slow or fast shoulder flexion as defined by Hodges and Richardson (1999). The trial variable represents the three trials performed for each of the task difficulty variables.

The dependent variables consisted of a nominal measure of balance and a ratio measure of the center of pressure. The nominal measure of passing or not passing was used to quantify the level of difficulty attained by participants prior to losing balance. The center of pressure trajectories, which are ratio scores, were quantified with summary statistics. Center of pressure trajectories are expressed as the anterior-posterior and medial-lateral distances from center at a given instant, with the positive or negative values associated with the direction of sway from the reference point. Summary statistics included a positive and negative maximum (MAX), absolute average (ABS AVG), and

the root mean square error (RMSE) displacement of the center of pressure. The greatest sway distance achieved without losing balance was expressed as the maximum (MAX) positive and negative measure. The absolute average (ABS AVG) is the arithmetic mean of the absolute value of the center of pressure trajectories. This indicates the average size of the center of pressure trajectories without regard to sign, allowing interpretation of the average sway independent of direction. The root mean square error (RMSE) is the average sway. Center of pressure measures were collected in a medio-lateral (MAX + M-L, MAX - M-L, ABS AVG M-L, RMSE M-L) and antero-posterior directions (MAX + A-P, MAX – A-P, ABS AVG A-P, RMSE A-P) and a total center of pressure path length traveled per second (PATH). PATH is useful for describing the directional and distance sway tendency. Center of pressure therefore consisted of nine ratio scores.

Analysis of Data

The Statistical Package for the Social Sciences Standard Version 11.0.1 for Windows (SPSS, Inc., 2001) was used for all statistical analyses. Relevant descriptive information on participants is reported in Table 1. Parametric tests were used for the dependent variables MAX + M-L, MAX - M-L, ABS AVG M-L, RMSE M-L, MAX + A-P, MAX – A-P, ABS AVG A-P, RMSE A-P, and PATH, they are continuous scores. A 2 x 2 x 3 (History of Pain) x (Task Difficulty) x (Trials) ANOVA with repeated measures on the last two factors was used to examine the interaction and main effects of the independent variables on each of the center of pressure scores. Effect size statistics were calculated whenever a statistically significant effect was uncovered. The effect size

statistic used for the analysis of variance is Eta (η). All tests were two-tailed, with the critical value set at $p = .05$ for each of the dependent measures.

Table 1

Descriptive Statistics

Source	*N*	Range	*M*	*SD*
		Pain History Group		
Age (in years)	12	30.0 - 50.0	36.8	7.32
Height (in cm.)	12	160 - 172	169	5.57
Weight (in kg.)	12	50.0 - 79.6	64.3	14.2
T9-L5 Distance (in cm.)	12	18.0 - 25.0	22.0	2.37
		No Pain History Group		
Age (in years)	12	30.0 - 50.0	39.1	6.92
Height (in cm.)	12	160 - 183	171	8.21
Weight (in kg.)	12	51.8 - 88.6	67.6	14.9
T9-L5 Distance (in cm.)	12	16.0 - 27.0	22.1	3.31

Summary

This research experimentally examined if the level of task difficulty would interact with pain history during unstable sitting as indicated by postural control measurements. The practical purpose of this research was to design a field measure to show differences in postural control between individuals with and without a history of low back pain. Male and female participants were equally divided between two groups. The two groups were history of low back pain and no history of low back pain. Each participant performed three 10-second trials of shoulder flexion of the dominant arm at both slow and fast shoulder flexion speeds, which was preceded by one practice trial at each arm speed. The dependent measures center of pressure and balance were collected for each of the three trials. At the end of the testing, data were analyzed to determine the effects of history of low back pain and task difficulty on center of pressure and balance.

CHAPTER 4

Results

This research experimentally examined if the level of task difficulty would interact with pain history during unstable sitting as indicated by postural control measurements. The practical purpose of this research was to design a field measure to show differences in postural control between individuals with and without a history of low back pain. A total of 24 participants were assigned to either a history of low back pain group (6 males, 6 females, $M = 36.8$ years, $SD = 7.32$, $M = 169$ cm, SD = 5.57, $M = 64.3$ kg, $SD = 14.2$, $M = 22.0$ cm T9-L5, $SD = 2.38$) or control group (6 males, 6 females, $M = 39.1$ years, $SD = 6.92$, $M = 171$ cm, $SD = 8.21$, $M = 67.6$ kg, $SD = 14.9$, $M = 22.1$ cm T9-L5, $SD = 3.05$). Differences in center of pressure summary statistics and PATH between participants with a history of low back pain and those without a history of low back pain were analyzed with a 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) with repeated measures on the last two factors. The effect size, or strength of the finding was determined by Eta (η). This chapter includes a section on: results of study and summary.

Results of Study

Task difficulty was counterbalanced across participants. A one-way ANOVA was conducted on the order of task difficulty to determine the potential effects of learning and fatigue on the dependent variables. No statistically significant effects were revealed across the dependent variables (see Table 2).

Table 2

Summary of Analysis of Variance of Counterbalancing Across Task Difficulty

Source	Summary Statistics
RMSE A-P slow	$F(1,21) = .018, p = .894$
RMSE A-P fast	$F(1,21) = .112, p = .746$
ABS AVG A-P slow	$F(1,21) = .108, p = .723$
ABS AVG A-P fast	$F(1,21) = 1.22, p = .894$
MAX +A-P slow	$F(1,21) = .355, p = .741$
MAX +A-P fast	$F(1,21) = 3.15, p = .281$
MAX –A-P slow	$F(1,21) = .722, p = .502$
MAX –A-P fast	$F(1,21) = 1.02, p = .197$
RMSE M-L slow	$F(1,21) = .129, p = .557$
RMSE M-L fast	$F(1,21) = .468, p = .371$
ABS AVG M-L slow	$F(1,21) = .018, p = .405$
ABS AVG M-L fast	$F(1,21) = 1.78, p = .088$
MAX +M-L slow	$F(1,21) = .834, p = .090$
MAX +M-L fast	$F(1,21) = 2.27, p = .147$
MAX –M-L slow	$F(1,21) = 3.22, p = .325$
MAX –M-L fast	$F(1,21) = 2.78, p = .110$

The results of this study led to the acceptance of the null hypothesis that task difficulty as determined by the speed of shoulder flexion and history of pain will not interact to significantly affect center of pressure measurements (see Appendix F). The main effect of history of pain and the interaction of history of pain and task difficulty did not demonstrate significance across MAX +A-P, MAX +M-L, MAX –A-P, MAX –M-L, ABS AVG A-P, ABS AVG M-L, RMSE A-P, RMSE M-L, and PATH (see Appendix F). The null hypothesis that history of back pain would affect center of pressure measures was accepted.

The null hypothesis that task difficulty would not affect center of pressure measures was rejected. Summary statistics of MAX +A-P, MAX +M-L, MAX –A-P, MAX –M-L, ABS AVG A-P, ABS AVG M-L, and PATH increased significantly (see Table 2) due to an increase in task difficulty, except RMSE A-P and RMSE M-L. The means and standard deviations across task difficulty for MAX +A-P, MAX +M-L, MAX –A-P, MAX –M-L, ABS AVG A-P, ABS AVG M-L, RMSE A-P, RMSE M-L, and PATH are provided in Table 3. The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) with repeated measure on the last two factors for MAX +A-P, MAX +M-L, MAX –A-P, MAX –M-L, ABS AVG A-P, ABS AVG M-L, RMSE A-P, RMSE M-L, and PATH are provided in Appendix F.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for MAX +A-P with repeated measures on the last two factors are presented in Appendix F, Table F1. The analysis revealed a statistically significant effect for task difficulty, $F(1, 17) = 71.2, p < .001$. Approximately 90% of the variance in the MAX +A-P can be

explained by knowing task difficulty. The maximal anterior distance traveled by CoP was greater with fast shoulder flexion (M = .497 mm, SD = .046 mm), than slower shoulder flexion (M = .090 mm, SD = .018 mm). No other statistically significant interactions were revealed in this analysis.

Table 3

Mean and Standard Deviations of Task Difficulty for the Dependent Measures

	Slow		Fast	
Dependent Measure	*M*	*SD*	*M*	*SD*
MAX +A-P	.089	.018	.497	.046
MAX +M-L	.084	.014	.645	.067
MAX –A-P	-.043	.024	-.458	.015
MAX –M-L	-.095	.015	-.598	.061
ABS AVG A-P	.053	.015	.164	.014
ABS AVG M-L	.034	.005	.239	.032
RMSE A-P	.025	.018	.023	.018
RMSE M-L	-.009	.007	.017	.025
PATH	35.879	.679	71.861	6.126

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for MAX +M-L with repeated measures on the last two factors are presented in Appendix F, Table F2. The analysis revealed a statistically significant effect for task difficulty, $F(1, 17) = 63.9, p < .001$. Approximately 89% of the variance in MAX +M-L can be eplained by knowing task difficulty. The maximal medial distance traveled by CoP was greater with fast shoulder flexion ($M = .645$ mm, $SD = .067$ mm), than slower shoulder flexion ($M = .084$ mm, $SD = .014$ mm). No other statistically significant interactions were revealed in this analysis.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for MAX -A-P with repeated measures on the last two factors are presented in Appendix F, Table F3. The analysis revealed a statistically significant effect for task difficulty, $F(1, 17) = 82.3, p < .001$. Approximately 91% of the variance in MAX -A-P can be explained by knowing task difficulty. The maximal posterior distance traveled by CoP was greater with fast shoulder flexion ($M = -.458$ mm, $SD = .015$ mm), than slower shoulder flexion ($M = -.043$, $SD = .024$). No other statistically significant interactions were revealed in this analysis.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for MAX -M-L with repeated measures on the last two factors are presented in Appendix F, Table F4. The analysis of revealed a statistically significant effect for task difficulty, $F(1, 17) = 86.4, p < .001$. Approximately 91% of the variance in MAX -M-L can be explained by knowing task difficulty. The maximal lateral distance traveled by CoP was

greater with fast shoulder flexion (M = -.598 mm, SD = .061 mm), than slow shoulder flexion (M = -.095 mm, SD = .015 mm).

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for ABS AVG A-P with repeated measures on the last two factors are presented in Appendix F, Table F5. The analysis revealed a statistically significant effect for task difficulty, $F(1, 17) = 34.5$, $p < .001$. Approximately 82% of the variance in ABS AVG A-P can be explained by knowing task difficulty. The absolute average antero-posterior distance traveled by CoP was greater with fast shoulder flexion (M = .164 mm, SD = .014 mm), than slow shoulder flexion (M = .053 mm, SD = .015 mm).

The analysis for ABS AVG A-P also revealed a statistically significant effect for the interaction of history of pain and trials, $F(2, 34) = 3.39$, $p < .045$. Approximately 40% of the variance in ABS AVG A-P can be explained by knowing history of pain and trial. Differences in absolute average antero-posterior distance traveled by CoP increased across trial 1 (M = .109 mm, SD = .017 mm), trial 2 (M = .090 mm, SD = .016 mm), and trial 3 (M = .090 m, SD = .017 mm) with a history of back pain, and decreased across trial 1 (M = .115 mm, SD = .017 mm), trial 2 (M = .122 mm, SD = .015 mm), and trial 3 (M = .125 mm SD = .016 mm) without a history of back pain (see Figure 1). No other statistically significant interactions were revealed in this analysis.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for ABS AVG M-L with repeated measures on the last two factors are presented in Appendix F, Table F6. The analysis revealed a statistically significant effect for task difficulty, $F(1, 17) = 38.6$, $p < .001$. Approximately 83% of the variance in ABS AVG

M-L can be explained by knowing task difficulty. The absolute average medio-lateral distance traveled by CoP was greater with fast shoulder flexion (M = .239 mm, SD = .032 mm), than slow shoulder flexion (M = .034 mm, SD = .005 mm). No other statistically significant interactions were revealed in this analysis.

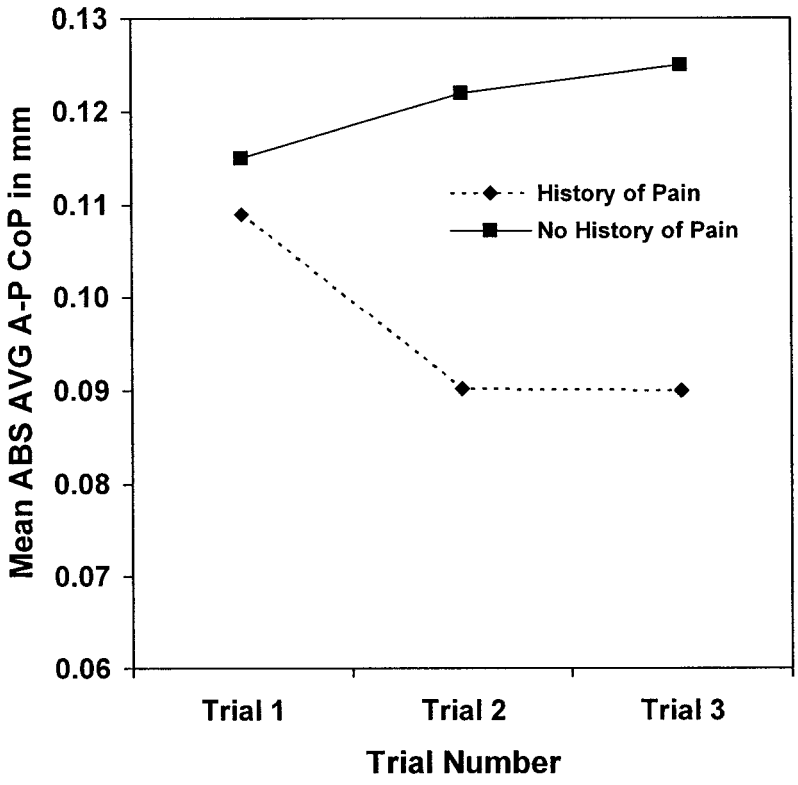


Figure 1. Mean ABS AVG A-P CoP trajectories for the interaction between trials and history of low back pain.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for RMSE A-P with repeated measures on the last two factors are presented in Appendix F, Table F7. The analysis revealed a statistically significant effect for trials, $F(2, 34) = 3.90, p < .05$. Approximately 43% of the variance in RMSE A-P can be explained by knowing the trial of performance. A significant interaction was revealed over trials between trial 1 and trial 2, $F(1,17) = 7.65, p < .05$. The average antero-posterior distance traveled by CoP was greater for trial 3 ($M = .0242$ mm, $SD = .018$ mm), than trial 2 ($M = .0233$ mm, $SD = .018$ mm), but not greater than trial 1 ($M = .0252$ mm, $SD = .018$ mm). Inspection of the data reveals that participants had a greater RMSE A-P CoP difference across trial 1 and 2 than they did across trial 2 and 3.

The analysis also revealed a statistically significant effect for the interaction of task difficulty and trials, $F(2, 34) = 3.70, p < .05$. Approximately 42% of the variance in RMSE A-P can be explained by knowing task difficulty and trials. A significant interaction was revealed for the interaction of task difficulty and trials over slow and fast shoulder flexion, $F(1,17) = 6.38, p < .05$. The average antero-posterior distance traveled by CoP was less with fast shoulder flexion across trial 1 ($M = .026$ mm, $SD = .018$ mm), trial 2 ($M = .021$ mm, $SD = .018$ mm), and trial 3 ($M = .023$ mm, $SD = .018$ mm), than slow shoulder flexion across trial 1 ($M = .025$ mm, $SD = .018$ mm), trial 2 ($M = .025$ mm, $SD = .018$ mm), and trial 3 ($M = .025$ mm, $SD = .18$ mm). Close inspection of Figure 2 reveals that participants had a lower RMSE A-P CoP during fast task difficulty from trial one to trial two. Also, participants had less RMSE CoP during trial two for the fast task

than the slow task. No other statistically significant interactions were revealed in this analysis.

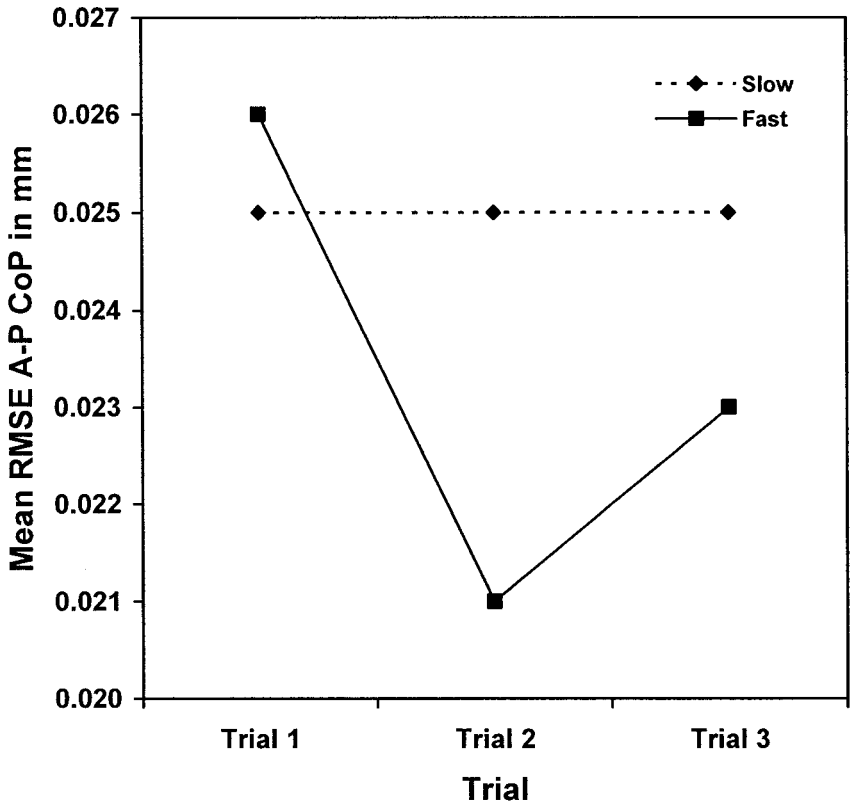


Figure 2. Mean RMSE A-P CoP trajectories for the interaction of trials and task difficulty.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for RMSE M-L with repeated measures on the last two factors are presented in Appendix F, Table F8. None of the variance in the average medio-lateral distance traveled by CoP could be explained by knowing the main effects or the interaction for history of pain, task difficulty, or trials. The analysis revealed no statistically significant effects.

The results of the 2 x 2 x 3 ANOVA (History of Pain x Task Difficulty x Trials) for PATH with repeated measures on the last two factors are presented in Appendix F, Table F9. The analysis revealed a statistically significant effect of task difficulty, $F(1, 18) = 32.4$, $p < .001$. Approximately 80% of the variance in PATH can be explained by knowing task difficulty. The total path length traveled by CoP per second was greater for fast shoulder flexion ($M = 71.86$ mm, $SD = 6.13$ mm), than slow shoulder flexion ($M = 35.98$ mm, $SD = .68$ mm).

The supplementary analysis that the ability to maintain balance during unstable sitting will not be related to a history of low back pain was unable to be tested. A failure in the equipment or methods design did not maximize the difficulty of the postural control task. Only one low back pain participant was unable to complete the test, therefore there was not enough variability in the data to perform an analysis.

Summary

The null hypothesis that task difficulty would not significantly affect center of pressure measures was rejected. Task difficulty was a significant main effect across all measures except RMSE A-P and RMSE M-L. The null hypothesis that history of pain would not significantly affect center of pressure was accepted. There was no main effect

of pain across any measure. The null hypothesis that task difficulty and history of low back pain would not interact to affect center of pressure was accepted. There were no significant interactions of task difficulty and history of pain across all measures. The main effect of trials and the interaction of task difficulty and trials exhibited a significant effect for RMSE A-P. The interaction of pain and trials exhibited a significant interaction for ABS AVG A-P. Supplementary analysis that the ability to maintain balance during unstable sitting would not be related to a history of low back pain could not be tested because of the lack of variability provided by the data.

CHAPTER 5

Discussion and Recommendations

This research experimentally examined if the level of task difficulty would interact with pain history during unstable sitting as indicated by postural control measurements. The practical purpose of this research was to design a field measure to show differences in postural control between individuals with and without a history of low back pain. In this chapter the significant findings will be juxtaposed with those from the literature. This chapter is divided into the following subcategories: discussion and recommendations, with the discussion section further divided by the main effects and interactions of the independent variables.

Discussion

Main Effects: Task Difficulty, Pain History, and Trials.

Postural responses of the trunk muscles are influenced by afferent feedback from the arm during shoulder flexion and by proprioceptive information resulting from trunk motion in response to the reactive forces generated by shoulder flexion (Hodges et al., 2001). Except for RMSE A-P and RMSE M-L, the effect of task difficulty was significant for the remaining dependent measures: MAX +A-P, MAX +M-L, MAX –A-P, MAX –M-L, ABS AVG A-P, ABS AVG M-L, and PATH.

Task difficulty as a significant factor across MAX +A-P, MAX +M-L, MAX –A-P, MAX –M-L, ABS AVG A-P, ABS AVG M-L center of pressure measures and PATH is in agreement with Hodges et al. (1999b) and Cholewicki et al. (2001). This supports the notion that during unstable sitting on a 44 cm hemisphere task difficulty was

increased from slow shoulder flexion to fast shoulder flexion. Contrary to the downward and backward reactive force generated by shoulder flexion as observed by Hodges and Richardson (1999), MAX +A-P, MAX –A-P, and RMSE A-P did not show a backward reactive force, which would have been observed as a negative bias. This may be due to differences in study design, as Hodges and Richardson examined the forces in standing and this study examined the forces in sitting.

In this study there was no significant main effect for history of pain, but RMSE A-P showed a significant main effect for trials. This is somewhat in contrast to Mientjes (1999) who showed root mean square distance traveled in the medio-lateral direction of center of pressure measures was able to distinguish between chronic low back pain participants from a healthy population during upright standing. The participants in Mientjes study were standing under conditions of visual obstruction. Standing may not isolate deficiencies in lumbar spine postural control (Cholewicki et al., 2000). Anticipation, in the form a feedforward response, is used to pretune senory and motor systems and to scale the amplitude of postural adjustments. In the absence of vision the ability to anticipate, or predict, so as to begin unconsciously planning the next motor response is diminished. Vuillerme et al. (2001) supported the contention that vision is the strongest feedback mechanism, and is able to adjust and maintain postural control in the presence lower limb fatigue; which reinforces the negative effect obstructing vision has on postural control.

While greater differences were observed between trial one and trial two than between trial two and trial three in this study, only 43% of the variance can be explained

by RMSE A-P. To examine the effect of learning, three trials at each level of task difficulty were performed. A lack of variability within the data fails to support any differences in CoP measures across trials being due to a learning effect. The effect of lumbar fatigue may have had an effect opposite to that of the potential learning, thereby negating any significant effect of learning. While there were subjective claims of fatigue as the number of trials progressed, fatigue as a factor was not studied. Since Cholewicki et al. (2001) did not show any affect of learning across trials, lumbar fatigue may be a factor to consider. A longer recovery time between trials, a reduced number of trials, and a longer trial time may reduce the perception or effects fatigue. A single trial over 30 seconds may control for both issues. A single trial will decrease any potential learning effect and testing for 30 seconds may show differences in postural control over time. The 30 seconds of data could then be divided into 10 second time frames to examine CoP changes over specified time intervals due variables such as fatigue or learning.

Interaction Effects: Task Difficulty by Pain History by Trials

In this study there was no significant interaction of history of pain with task difficulty or trials. There was a significant effect for the interaction of history of pain and trials, and task difficulty and trials. The interaction of task difficulty and trials for RMSE A-P, and the interaction of trials and pain for the ABS AVG A-P center of pressure, may be related to the lumbar fatigue during repeated shoulder flexion trials. As the muscular system of the participants fatigued it is possible that the natural response by the body would be to increase sway to receive greater feedback from the proprioceptive and vestibular system. This finding provides support for the suggestion by Panjabi (1992a,

1992b) and Saltzman and Kelso (1987) of a built in redundancy within the postural control system and by Taimela et al. (1999) that lumbar fatigue causes deficits in position sense.

Since only 43% of the variance in ABS AVG A-P can be explained by knowing trials and pain, and the significant interaction of trials and pain is only observed in ABS AVG A-P, the data may not be meaningful. It is possible that a significant effect is observed because of the nature of the analysis. By taking the absolute value of the CoP measures, all values are positive. The anterior bias observed in MAX +A-P, MAX –A-P, and RMSE A-P allows the suggestion that the lower CoP displacement across trials and task difficulty is due to a lower negative displacement during fast shoulder flexion. It was observed earlier that fast shoulder flexion produces greater sway than slow shoulder flexion. The slow shoulder flexion would produce less negative trajectories than the fast shoulder flexion, therefore the mean of the absolute value of the CoP displacements would appear more positive.

The body is constantly exposed to external or internal perturbations. The objective of the control system is to maintain the body's center of mass within the stability limit and to provide a stable platform for the head with a minimum of control effort (Kuo, 1995). When an individual attempts to stand or sit absolutely still, the body continuously sways within stability limits. How the control system works in this situation is debated, but one view is that signals from the sensory systems are continuously utilized to regulate the body position, which is considered closed loop control (Kuo, 1995). Another possibility is that sway within a hypothetical dead zone is

left unchecked by the control system until is exceeds a certain threshold, which is considered open loop control, after which feedback control (closed loop) is adopted (Collins & DeLuca, 1993; Collins, DeLuca, Burrows, & Lipsits, 1995). This open loop/closed loop mechanism allows a certain amount of variability in the control system, which may simplify the control task when the body is not in jeopardy of falling. The idea of an increase in center of pressure measures not being an absolute indicator of poorer postural control, or an individualized threshold for correcting sway, makes it difficult to interpret much of the research on postural control as greater center of pressure measures have traditionally been treated as indicators of poorer postural control.

It is possible that in the presence of lumbar fatigue, the participants increased spinal flexion to lower the center of gravity, thereby facilitating proprioceptive feedback from the lumbar spine (Hodges, 2001) and improving balance capabilities. Spinal flexion is coupled with an increase in forward head posture to maintain a horizontal reference of the visual system. A greater percentage of the body's mass may be held anterior to the center of pressure, especially as the shoulder moves from 0 - 90 degrees of shoulder flexion, which is supported by the positive mean center of pressure values for MAX +A-P slow (.089 mm) and MAX +A-P fast (.497 mm), and RMSE A-P slow (.025 mm) and RMSE A-P fast (.023 mm). When comparing the range of center of pressure measures between MAX +A-P (.088 mm – .526 mm) and MAX –A-P (-.019 mm – -.518 mm) across trials a slight anterior bias is observed (see Figure 3).

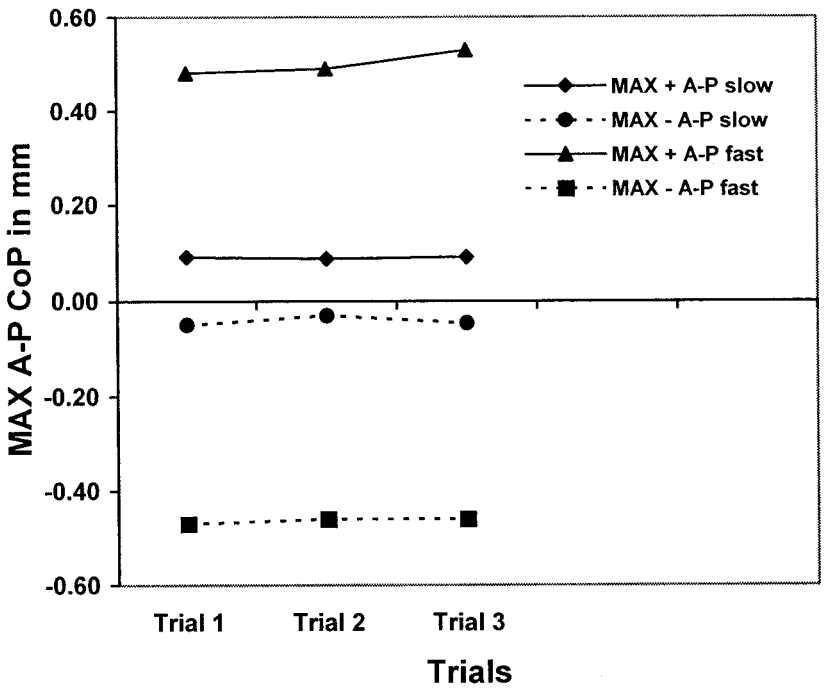


Figure 3. MAX A-P CoP trajectories for the interaction between trials and task difficulty.

This research has shown that increasing task difficulty through changes in the speed of shoulder flexion during unstable sitting can be used to increase postural control demands. While the problem of examining postural control in the absence of a force plate has not been found, the knowledge base required for the transition from static examination of postural control to dynamic examination of postural control has been increased. Task difficulty may be used to increase postural control requirements during

unstable sitting, but has not yet been able to differentiate between healthy individuals and those who have poor postural control secondary to a history of low back pain or other postural control disorders.

Recommendations

Skilled movements show task-specific flexibility in attaining a goal. If one part of the system is damaged, the system is able to compensate without the need to reorganize a new movement plan providing that the biomechanical disturbance (amount, direction, speed, duration of load) or neural disturbance (task complexity, attentional focus, past experiences) does not exceed the capabilities of the system to compensate (Saltzman & Kelso, 1987). The results of Hodges et al. (1999a) provided support that the anticipatory activity of the transversus abdominis is generated in parallel with the command for movement, meaning its activation is initiated directly by the stimulus to move and independent of the motor command to move. Further research provides additional support that in people with low back pain, the feedforward control of the transversus abdominis becomes increasingly delayed as task complexity increases, secondary to alterations in motor planning (Hodges, 2001). The change in motor planning is suspected to be caused by fear avoidance or attentional focus (Hodges, 2001).

Pain history as a main effect or the interaction of pain history and task difficulty failed to show a significant effect. Without a significant interaction between pain history and task difficulty, using unstable sitting with slow and fast shoulder flexion, a field measure of postural control deficits to predict injury or the recurrence of an injury or to guide a rehabilitation program cannot be justified. The delay in timing of transversus

abdominis contractions and the failure of the timing to return under traditional rehabilitation programs (Hodges and Richardson, 1996, 1997a, 1997b, 1999a) coupled with the long-term atrophy observed in individuals with a history of low back pain (Hides et al., 1996), underscore the need for clinical and field methods of testing that are easy to perform and reliable. Until an easy and reliable clinical or field test is designed, the addition of activities that are task specific and also challenge individually or as a group the subsystems underlying postural control should be included as part of rehabilitation programs and fitness programs of apparently healthy individuals.

Rankin, Woollacott, Shumway-Cook, and Brown (2000) used a dual-task paradigm involving standing platform perturbations as the primary task and math task involving subtraction by threes as the secondary task. Postural muscle activity as measured by electromyography was delayed in the math task group suggesting less attentional processing capacity was available for balance. Most dual-task studies employ verbal responses for the secondary task. Dault, Yardley, and Frank (2003) compared a verbal response mental task to a silent response mental task. They showed that the motor task of articulation produced increased postural sway frequency and sway path. The changes in sway that accompany a secondary task are not wholly attributable to attentional load, but appear to be partly due to the motor requirements of the task. This provides further support for the use of shoulder flexion as a way to increase task difficulty during a postural control task. Additionally, anticipation of pain and fear-avoidance beliefs about physical activity, independent of the sensory perception of pain are predictors of physical performance during a postural control task (Al-Obaidi, Nelson,

Al-Awadhi, & Al-Shuwaie, 2000), and anxiety regarding the possibility of falling increases the attentional demands for locomotion (Gage, Sleik, Polych, McKenzie, & Brown, 2003). This underscores the need to examine psychological variables in addition to physical variables when testing postural control.

Further research may examine the role of attentional demand on postural control using a similar study design. By requiring participants to pay less attention to the task of balancing while sitting by having them perform a novel cognitive task in addition to a motor task, the challenge to the postural control system will be amplified and may further simulate the complex demands of sport or daily life. At this time creating a truly functional test of postural control is unrealistic however, a postural control test combined with validated questionnaires about the anticipation of pain, fear avoidance beliefs, and anxiety regarding the fear of falling, may interact to increase the meaningfulness of postural control measures and be able to show differences between individuals with and without a history of low back pain.

The reliability of estimates of center of pressure measures on an individual level are limited (Newell, van Emmerik, Lee, & Sprague, 1993). Reliably determining whether an individual belongs to a certain diagnostic group or not by using center of pressure measures is difficult. This is due to the relatively large intra-subject variability and inter-subject variability (Samson & Crow, 1996; Figura, Cama, Capranica, Guidetti, & Pulejo 1991). The task for researchers and clinicians appears to be to find adequate methods of acquiring relevant information so that clinically useful measures of postural control and balance can be developed. The addition of a dual task paradigm to the

unstable sitting test with slow or fast shoulder flexion may challenge the feedforward capabilities of the postural control system enough to show the effect of task difficulty and history of pain on center of pressure trajectories.

Summary

This research did not show how the level of task difficulty and pain history interact to affect postural control measurements, nor can it be used as a field test to show differences in postural control between individual with or without low back pain in the absence of a force plate. Task difficulty was the consistent significant effect across center of pressure measures, supporting the hypothesis that fast shoulder flexion was a more difficult motor control task than slow shoulder flexion. The interaction of history of pain and trials significantly affected ABS AVG A-P, but the effect size indicates the strength of the relationship at 40% is weak. The significant main effect of trials and the interaction of task difficulty and trials for RMSE A-P describe a low strength for the variance in CoP they account for, 43% and 42%, respectively. The significant differences in ABS AVG A-P and RMSE A-P across trials may be related to fatigue, but this cannot be elucidated from the current study.

Future research needs to continue to examine the complexity of human postural control mechanisms. The task-specific skills required for goal attainment underscore the difficulty in designing a simple, valid, and reliable test to measure postural control. A variety of tests examining anxiety, fear avoidance beliefs, under a dual-task and static-dynamic paradigm using both broad based measures of postural control such as center of pressure measures and task-specific examination of postural control may be required to

fully understand the built-in redundancy of the postural control system and differentiate measurements of postural control between individuals with a history of back pain and those without a history of low back pain. It is recommended that until an easy to perform, valid, and reliable field measure or clinical examination of postural control is developed, the addition of activities that are task specific and also challenge individually or as a group the subsystems underlying postural control should be included as part of rehabilitation programs and fitness programs of all individuals.

REFERENCES

Aftab, E.P., Ishae, M.G., & Winter, D.A. (2002). Anticipatory control of center of mass and joint stability during voluntary arm movement from a standing posture: interplay between active and passive control. *Experimental Brain Reasearch, 143*, 318-327.

Allan, D.B., & Waddell, G. (1989) An historical perspective on low back pain and disability. *Acta Orthopaedica Scandinavica Supplementum, 234*(60), 1-23.

Al-Obaidi, S.M., Nelson, R.M., Al-Awadhi, S. & Al-Shuwaie, N. (2000). The role of anticipation and fear of pain in the persistence of avoidance behavior in patients with chronic low back pain. *Spine, 25*(9), 1126-1131.

Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H., & Svensson, P., (1995). The influence of low back pain on muscle activity and coordination during gait: A clinical experimental study. *Pain, 64*, 231-240.

Bergmark, A. (1989). Mechanical stability of the lumbar spine. *Acta Orthopedica Scandinavia Supplement, 60*(230), 1-54.

Bouisset, S., & Zattara, M., (1981). A sequence of postural movements precedes voluntary movement. *Neuroscience Letters, 22*, 263-270.

Bullock-Saxton, J.E., Janda, V., & Bullock, M.I., (1993). Reflex activation of gluteal muscles in walking. *Spine, 18*(6), 704-708.

Brumagne, S., Lysens, R., Swinnen, S., & Verschueren, S. (1999). Effect of paraspinal muscle vibration on position sense of the lumbosacral spine. *Spine, 24*(13), 1328-1331.

Byl, N.N., & Sinnott, P.T. (1991). Variations in balance and body sway in middle-aged adults. *Spine, 16*(3), 325-330.

Cairns, M.C., Harrison, K., & Wright, C. (2000). Pressure Biofeedback: A useful tool in the quantification of abdominal muscle dysfunction. *Physiotherapy, 86*(3), 127-138.

Chiang, J., & Potvin, J.R. (2001). The in vivo dynamic response of the human spine to rapid lateral bend perturbations. *Spine, 26*(13), 1457-1464.

Cholewicki, J., Polzhofer, G.K., & Radebold, A. (2000). Postural control of trunk during unstable sitting. *Journal of Biomechanics, 33*, 1733-1737.

Collins, J.J., De Luca, C.J., Burrows, A., & Lipsitz, L.A. (1995). Age-related changes in open-loop and closed-loop postural control mechanisms. *Experimental Brain Research*, 104, 480-492.

Collins, J.J., & De Luca, C.J. (1993). Open-loop and closed-loop control of posture: A random-walk analysis of center-of-pressure trajectories. *Experimental Brain Research, 95*, 308-318.

Dault, M.C., Yardley, L., & Frank, J.S. (2003). Does articulation contribute to modifications of postural control during dual-task paradigms? *Brain Research and Cognitive Brain Research, 16*(3), 434-440.

Figura, F., Cama, G., Capranica, L., Guidetti, L., & Pulejo C. (1991). Assessment of static balance in children. *Journal of Sports Medicine and Physical Fitness*, 31, 235-242.

Forssberg, H., & Hirschfeld, H. (1994). Postural adjustments in sitting humans following external perturbations: muscle activity and kinematics. *Experimental Brain Research, 97*, 515-527.

Gurfinkel, V.S., Lipshits, M.I., & Lestienne, F.G. (1988). Anticipatory neck muscle activity associated with rapid arm movements. *Neuroscience Letters*, 94, 104-108

Gage, W.H., Sleik, R.J., Polych, M.A., McKenzie, N.C., & Brown, L.A. (2003). The allocation of attention during locomotion is altered by anxiety. *Experimental Brain Research, 150*(3), 385-394.

Gill, K.P., & Callaghan, M.J. (1998). The measurement of lumbar proprioception in individuals with and without low back pain. *Spine, 23*(3), 371-377.

Helewa, A., Goldsmith, C.H., Lee, P., Smythe, H.A., & Forwell, L. (1999). Does strengthening the abdominal muscles prevent low back pain – A randomized controlled trial. *The Journal of Rheumatology, 26*(8), 1808-1815.

Hides, J.A., Jull, G.A., & Richardson, C.A. (2001). Long-term effects of specific stabilizing exercises for first-episode low back pain. *Spine, 26*, 243-248.

Hides, J.A., Richardson, C.A., & Jull, G.A. (1996). Multifidus recovery is not automatic after resolution of acute first-episode low back pain. *Spine*, *21*(23), 2763-2769.

Hides, J.A., Stokes, M.J., Saide, M., Jull, G.A., & Coopers, D.H. (1994). Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine*, *19*(2), 165-172.

Hodges, P.W. (2001). Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. *Experimental Brain Research, 141*, 261-266.

Hodges, P.W., Butler, J.E., McKenzie, D.K., & Gandevia, S.C. (1997). Contraction of the human diaphragm during rapid postural adjustments. *Journal of Physiology, 505*(2), 539-548.

Hodges, P.W., Cresswell, A.G., & Thorsternsson, A. (2001). Perturbed upper limb movements cause short-latency postural responses in trunk muscles. *Experimental Brain Research, 138*, 243-250.

Hodges, P., Cresswell, A., & Thorstensson, A. (1999). Preparatory trunk motion accompanies rapid upper limb movement. *Experimental Brain Research, 124*, 69-79.

Hodges, P.W., Gandevia, S.C., & Richardson, C.A. (1997). Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. *Journal of Applied Physiology, 83*(3), 753-760.

Hodges, P.W., & Richardson, C.A. (1996). Inefficient muscular stabilization of the lumbar spine associated with low back pain – A motor control evaluation of the transversus abdominis. *Spine, 21*(22), 2640-2650.

Hodges, P.W., & Richardson, C.A. (1997a). Feedforward contraction of transversus abdominis is not influenced by direction of arm movement. *Experimental Brain Research, 114,* 362-370.

Hodges, P.W., & Richardson, C.A. (1997b). Contraction of the abdominal muscles associated with movement of the lower limb. *Physical Therapy, 77*(2), 132-143.

Hodges, P.W., & Richardson, C.A. (1999a). Transversus abdominis and the superficial abdominal muscles are controlled independently in a postural task. *Neuroscience Letters, 265*, 91-94.

Hodges, P.W., & Richardson, C.A. (1999b). Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. *Archives of Physical Medicine and Rehabilitation, 80*, 1005-1012.

Huang, Q., Andersson, E., & Thorstensson, A. (2001). Intramuscular myoelectric activity and selective coactivation of trunk muscles during lateral flexion with and without load. *Spine, 26*(13), 1465-1472.

Jackson, M., Solomonow, M., Zhou, B., Baratta, R.V., & Harris, M. (2001). Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion. *Spine, 26*(7), 715-723.

Kuo, A.D. (1995). An optimal control model for analyzing human postural balance. *IEEE Transcripts of Biomedical Engineering*, 42, 87-101.

Kollmitzer, J., Ebenbichler, G.R., Sabo, A., Kerschan, K., & Bochdansky, T. (2000). Effects of back extensor strength training versus balance training on postural control. *Medicine and Science in Sports and Exercise, 32*, 1770-1776.

Lee, T. (1998) On the dynamics of motor control research. *Research Quarterly for Exercise and Sport, 69*(4), 334-337.

Luoto, S., Aalto, H., Taimela, S., Hurri, H., Pyykko, I., & Alaranta, H. (1998). One-footed and externally disturbed two-footed postural control in patients with chronic low back pain and healthy control subjects. *Spine, 23*(19), 2081-2090.

Luoto, S., Taimela, S., Hurri, H., Aalto, H., Pyykko, I., & Alaranta, H. (1996). Psychomotor speed and postural control in chronic low back pain patients. *Spine*, *21*(22), 2621-2627.

Luoto, S., Taimela, S., Hurri, H., & Alaranta, H. (1999). Mechanisms explaining the association between back trouble and deficits in information processing. *Spine*, *24*(3), 255-261.

McGill, S. (2001). Low back stability: From formal description to issues for performance and rehabilitation. *Exercise and Sport Science Reviews*, *29*(1), 26-31.

Mientjes, M.I. (1999). Balance in chronic low back pain patients compared to healthy people under various conditions in upright standing. *Clinical Biomechanics, 14*(10), 710-716.

Miller, M.I., & Medeiros, J.M. (1987). Recruitment of internal oblique and transversus abdominis muscles during the eccentric phase of the curl-up exercise. *Physical Therapy*, *17*(8) 1213-1217.

Newell, K.M., van Emmerik, R.E.A., Lee, D., & Sprague, R.L. (1993). On postural stability and variability. *Gait and Posture*, 4, 225-230.

Ng, G.Y.F., & Richardson, C.A. (1990). The effects of training triceps surae using progressive speed loading. *Physiotherapy and Practice, 6*, 77-84.

O'Sullivan, P.B., Twomey, L., & Allison, G.T. (1997). Evaluation of specific stabilizing exercises in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine*, *22*(24), 2959-2967.

Panjabi, M.M. (1992a). The stabilising system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *Journal of Spinal Disorders*, *5*, 383-389.

Panjabi, M.M. (1992b). The stabilising system of the spine. Part II. Neutral zone and stability hypothesis. *Journal of Spinal Disorders*, *5*, 390-397.

Radebold, A., Cholewicki, J., Panjabi, M.M., & Patel, T.C. (2000). Muscle response patterns to sudden trunk loading in healthy individuals and in patients with chronic low back pain. *Spine*, *25*(8), 947-954.

Radebold, A., Cholewicki, J., Polzhofer, G.K., & Greene, H.S. (2001). Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. *Spine*, *26*(7), 724-730.

Rankin, J.K., Woollacott, M.H., Shumway-Cook, A., & Brown, L.A. (2000). Cognitive influence on postural stability: a neuromuscular analysis in young and older adults. *Journal of Gerontological and Biological Science in Medicine and Science, 55*(3), M112-9.

Richardson, C., & Bullock, M. (1986). Changes in muscle activity during fast, alternating flexion-extension movements of the knee. *Scandinavian Journal of Rehabilitative Medicine*, *18*, 51-58.

Richardson, C.A., & Jull, G.A. (1995). Muscle control – pain control. What exercises would you prescribe. *Manual Therapy*, *1*, 2-10.

Richardson, C., Jull, G., Hodges, P., & Hides, J. (1999). *Therapeutic exercise for spinal segmental stabilization in low back pain: Scientific basis and clinical approach.* London: Churchill Livingstone.

Saltzman, E., & Kelso, S.J.A. (1987). Skilled actions: A task-dynamic approach. *Psychological Review, 94*(1), 84-106.

Samson, M., & Crow, A. (1996). Intra-subject inconsistencies in quantitative assessments of body sway. *Gait and Posture, 4*, 252-257.

St. Claire-Gibson, A., Lambert, M.I., & Noakes, T.D. (2001). Neural control of force output during maximal and submaximal exercise. *Sports Medicine*, *31*(9), 637-650.

Shumway-Cook, A., & Woollacott, M.J. (2001*). Motor control: Theory and practical applications* (2nd ed.). Philadelphia: Lippincott Williams & Wilkins.

Taimela, S., Kankaanpaa, M., & Luoto, S. (1999). The effect of lumbar fatigue on the ability to sense a change in lumbar position. *Spine*, *24*(13), 1322-1327.

Taub, E., Uswatte, G., & Elbert T. (2002). New treatments in neurorehabilitation founded on basic research. *Nature Reviews Neuroscience, 3*(3), 228-236.

Vera-Garcia, F.J., Grenier, S.G., & McGill, S.M. (2000). Abdominal muscle response during curl-ups on both stable and labile surfaces. *Physical Therapy*, *80*(6), 564-569.

Vuillerme, N., Nougier, V., & Prieur, J.M. (2001). Can vision compensate for a lower limbs muscular fatigue for controlling posture in humans. *Neuroscience Letters*, *308*, 103-106.

Wilder, D.G., Aleksiev, A.R., Magnusson, M.L., Pope, M.H., Spratt, K.F., & Goel, V.K. (1996). Muscular response to sudden load. *Spine*, *21*(22), 2628-2639.

Winter, D.A. (1990). *Biomechanics and motor control of human movement* (2nd ed.). New York: John Wiley & Sons, Inc.

APPENDIX A

Participant Information Sheet

Name:_____ Date: _____

Age:_____ Weight:_____kg Height: _____cm

T9-L5 Length:_____cm

Circle the Appropriate Answer

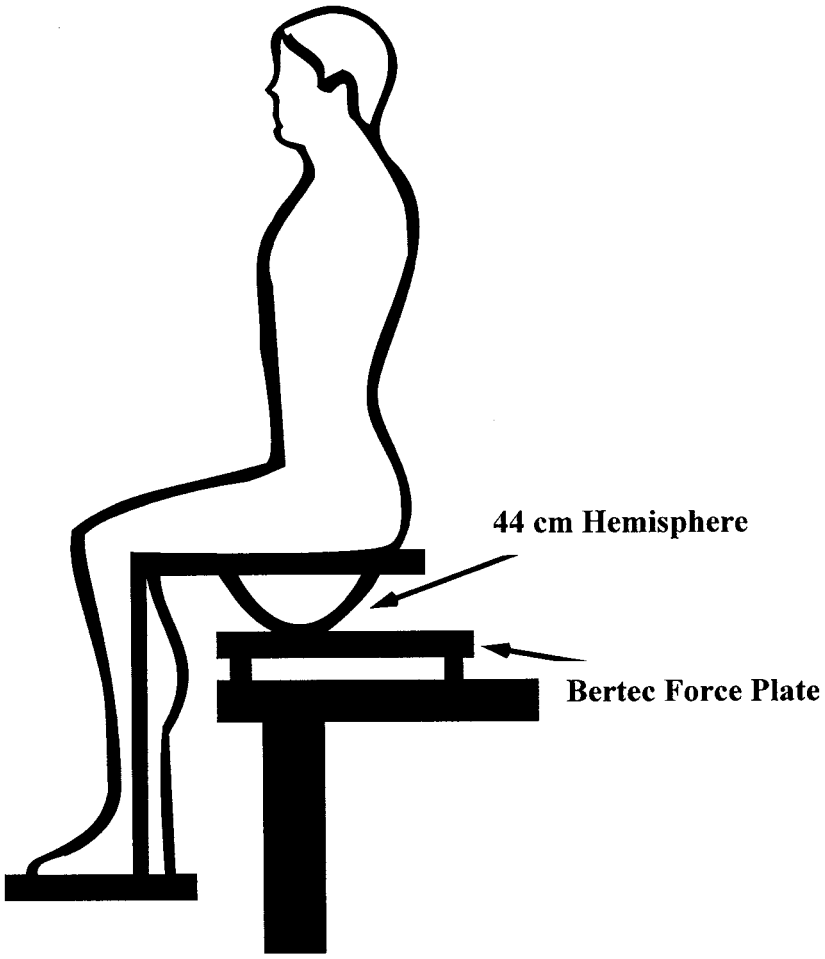
Male	Female	Sex
Left	Right	Arm Dominance Question: What arm do you use to throw a ball?
Yes	No	Do you have any visual or vestibular disorders, or any type of injury that causes shoulder or neck pain during shoulder flexion of the dominant arm?
Yes	No	Do you wear corrective lenses for anything other than reading?
Yes	No	If you wear corrective lenses for other than reading, do you have them with you today?
Yes	No	Do you have chronic, unremitting pain, neurological symptoms, pain extending beyond the gluteal fold, fractures, previous abdominal or spinal surgery, or recent pregnancy. ?
Yes	No	Do you have any history of low back pain lasting longer than three days?

If you answered no to the previous question, stop here.

Yes	No	If you have low back pain, have you been screened by an orthopedic surgeon or physiatrist within the past 3-months with no change in symptoms since the visit?

APPENDIX B

View of Unstable Platform Design


44 cm Hemisphere
Bertec Force Plate

APPENDIX C

Institutional Review Board Approval Letter

San José State
UNIVERSITY

Office of the Academic Vice President
Associate Vice President Graduate Studies and Research
One Washington Square
San José, CA 95192-0025
Voice: 408-283-7500
Fax: 408-924-2477
E-mail: gstudies@wahoo.sjsu.edu
http://www.sjsu.edu

To: Brian Bettendorf
1211 Chantal Way
Redwood City, CA 94061

From: Nabil Ibrahim
AVP, Graduate Studies & Research

Date: October 18, 2002

The Human Subjects-Institutional Review Board has approved your request to use human subjects in the study entitled:

Task difficulty as an indicator of
postural control deficits in the lumbar spine.

This approval is contingent upon the subjects participating in your research project being appropriately protected from risk. This includes the protection of the anonymity or confidentiality of the subjects' identity when they participate in your research project, and with regard to any and all data that may be collected from the subjects. The approval includes continued monitoring of your research by the Board to assure that the subjects are being adequately and properly protected from such risks. If at any time a subject becomes injured or complains of injury, you must notify Nabil Ibrahim, Ph.D. immediately. Injury includes but is not limited to bodily harm, psychological trauma, and release of potentially damaging personal information. This approval for the human subjects portion of your project is in effect for one year, and data collection beyond October 18, 2003 requires an extension request.

Please also be advised that all subjects need to be fully informed and aware that their participation in your research project is voluntary, and that he or she may withdraw from the project at any time. Further, a subject's participation, refusal to participate, or withdrawal will not affect any services that the subject is receiving or will receive at the institution in which the research is being conducted.

If you have any questions, please contact me at (408) 924-2480.

APPENDIX D

Consent Form

San José State
UNIVERSITY

College of Applied Sciences and Arts
Department of Human Performance
One Washington Square
San José, CA 95192-0054
Voice: 408-924-3010
Fax: 408-924-3053

Consent form

Agreement to Participate in Research

Brian Bettendorf, a graduate student, has asked you to participate in a research study at this institution. The title of the research is *Effects of Task Difficulty on Lumbar Spine Postural Control.*

The purpose of the research is to determine if arm movement during unstable sitting can be used to show differences in postural control between healthy individuals and those with lower back pain.

You will be asked to perform slow or fast shoulder flexion during unstable sitting. Multiple trials will take place, and it may take 30 minutes to complete your participation in the study.

There is a risk of the possibility of falling or having a feeling of falling off of the platform, and of fatigue. To minimize this risk, you will be given timed rest breaks between trials while holding onto supportive hand rails. No other risks to you are foreseen, beyond the level of risk normally found in daily life.

While you may not benefit directly from participation in the study, you may gain an understanding of postural control and the influence of the sensory system in modifying postural control.

Although the results of this study may be published, no information that could identify you will be included. In order to maintain confidentiality, the researcher will create a number to identify each participant, and the number will be known only to him. Each participant's data will be kept in a locked safe under care of the researcher.

There is no compensation for your participation in the study.

Any questions you have concerning the study or your participation in it before or after your consent, will be answered by Brian Bettendorf, (650) 851-1145 or his faculty advisor Dr. Emily Wughalter, (408) 924-3043. Complaints about the research may be presented to Dr. Greg Payne, Chair of the Human Performance Department, at (408) 924-3028. If you have questions about your rights as a research participant, or to report a research-related injury, please contact Dr. Nabil Ibrahim, Associate Vice President for Graduate Studies and Research, at (408) 924-2480.

Please initial here:_____

No service of any kind, to which you are otherwise entitles, will be lost or jeopardized if you choose to "not participate" in the study.

Your consent is being given voluntarily. You may refuse to participate in the entire study or in any part of the study. If you decide to participate in the study, you are free to withdraw at any time without any negative effect on your relations with San Jose State University.

At the time that you sign this consent form, you will receive a copy of it for your records, signed and dated by the investigator.

- **The signature of a subject on this document indicates agreement to participate in the study.**
- **The signature of a researcher on this document indicates agreement to include the above named subject in the research and attestation that the subject has been fully informed of his or her rights.**

_____ _____

Signature Date

_____ _____

Signature Date

APPENDIX E

Taped Instructions

Condition A (slow trial-fast trial)

Thank you for agreeing to participate in this study. I will be measuring your postural control during two different testing conditions. The test requires you to sit on an unstable platform while performing slow or fast shoulder flexion in a random order. I will describe then demonstrate slow and fast shoulder flexion. I will then have you demonstrate.

Stop tape

Investigator will describe the slow shoulder flexion as approximately 60° of shoulder flexion at a speed of 30° per second, then demonstrates slow shoulder flexion for 3 repetitions. Participant demonstrates slow shoulder flexion until investigator is confidant in the participant's ability to replicate slow shoulder flexion when prompted. Investigator will describe the fast shoulder flexion as approximately 60° of shoulder flexion performed as fast as possible, then demonstrates fast shoulder flexion for 10 repetitions. Participant demonstrates fast shoulder flexion until the investigator is confident in the participant's ability to replicate fast shoulder flexion when prompted.

Begin tape

You have already been shown slow and fast shoulder flexion. Do you have any questions on the shoulder flexion before we continue?

10-second pause so that investigator can stop tape if participant has any questions.

Once the testing begins, if you grab onto the handrails during a trial, allow an edge of the unstable platform to make contact with the force plate, or use either of your arms to regain balance, the trial will be stopped. You will be allowed one re-trial at each level of

difficulty before the testing is finished and a "not passed" is recorded for the given trial. Your goal is to complete both of the trials.

Are there any questions before we continue?

Stop tape to answer any questions and begin the participant set-up on the unstable platform.

Participant sits on unstable platform while investigator adjusts the footplate to have a 90-degree angle at the hip and the knee.

Begin tape

You are now sitting on top of an unstable platform. During the test, please maintain eye contact with the mark on the wall in front you.

10-pause while investigator points to the mark on the wall

Between trials please hang to the handrails. You will be performing three 10-second trials with two conditions, slow shoulder flexion or fast shoulder flexion. Each trial will be followed by a 30-second recovery. One 10-second practice trial will precede the actual measurements. You will be cued with the words "get ready" when the trial is ready to begin, at which time you will take your arms off of the hand rails and prepare yourself to perform shoulder flexion at the announced speed. The prompt to begin the trial will be "go" and the prompt to finish will be "stop". This recording will take you through each condition with cues as to when to begin or stop, and whether to perform slow or fast shoulder flexion. Do you have any questions?

10-second pause so investigator can stop tape if there are questions.

Your 10-second practice trial with slow shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your first 10-second trial with slow shoulder flexion is coming up, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your second 10-second trial with slow shoulder flexion is coming up, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your third 10-second trial with slow shoulder flexion is coming up, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your 10-second practice trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your first 10-second trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your second 10-second trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your third 10-second trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

You have completed all of the trials. Thank you for your time, you may now come off the platform.

Condition B (fast trial-slow trial)

Thank you for agreeing to participate in this study. I will be measuring your postural control during two different testing conditions. The test requires you to sit on an unstable platform while performing slow or fast shoulder flexion in a random order.

Stop tape

Investigator will describe the slow shoulder flexion as approximately 60° of shoulder flexion at a speed of 30° per second, then demonstrates slow shoulder flexion for 3 repetitions. Participant demonstrates slow shoulder flexion until investigator is confidant in the participant's ability to replicate slow shoulder flexion when prompted. Investigator will describe the fast shoulder flexion as approximately 60° of shoulder flexion performed as fast as possible, then demonstrates fast shoulder flexion for 10 repetitions. Participant demonstrates fast shoulder flexion until the investigator is confident in the participant's ability to replicate fast shoulder flexion when prompted.

Begin tape

You have already been shown slow and fast shoulder flexion. Do you have any questions on the shoulder flexion before we continue?

10-second pause so that investigator can stop tape if participant has any questions.

Once the testing begins, if you grab onto the handrails during a trial, allow an edge of the unstable platform to make contact with the force plate, or use either of your arms to regain balance, the trial will be stopped. You will be allowed one re-trial at each level of difficulty before the testing is finished and a "not passed" is recorded for the given trial. Your goal is to complete both of the trials.

Are there any questions before we continue?

Stop tape to answer any questions and begin the participant set-up on the unstable platform.

Participant sits on unstable platform while investigator adjusts the footplate to have a 90-degree angle at the hip and the knee.

Begin tape

You are now sitting on top of an unstable platform. During the test, please maintain eye contact with the mark on the wall in front you.

10-pause while investigator points to the mark on the wall

Between trials please hang on to the handrails. You will be performing three 10-second trials two conditions, slow shoulder flexion or fast shoulder flexions. Each trial will be followed by a 30-second recovery. One 10-second practice trial will precede the actual measurements. You will be cued with "get ready" when the trial is ready to begin, at which time you will take your arms off of the hand rails and prepare yourself to perform shoulder flexion at the announced speed. The prompt to begin the trial will be

"go" and the prompt to finish will be "stop". This recording will take you through each condition with cues as to when to begin or stop, and whether to perform slow or fast shoulder flexion. Do you have any questions?

10-second pause so investigator can stop tape if there are questions.

Your 10-second practice trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your first 10-second trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your second 10-second trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your third 10-second trial with fast shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your 10-second practice trial with slow shoulder flexion is ready to begin, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your first 10-second trial with slow shoulder flexion is coming up, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your second 10-second trial with slow shoulder flexion is coming up, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

Your third 10-second trial with slow shoulder flexion is coming up, **get ready** (variable time passage), **go**, *10-seconds time passage,* **stop**. Place your hands on the handrails while you rest for 30 seconds.

You have completed all of the trials. Thank you for your time, you may now come off the platform.

APPENDIX F

Summary Statistic Tables

Table F1

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of MAX +A-P

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	.001	.00	.982
P within-group error	17	(.0694)		
		Within subjects		
Task Difficulty (D)	1	71.2***	.90	.001
P x D	1	.163	.094	.692
D within-group error	17	(.00662)		
Trial (T)	2	.962	.23	.392
P x T	2	.356	.14	.703
T within-group error	34	(.00710)		
D x T	2	.926	.23	.406
P x D x T	2	1.49	.28	.239
D x T within-group error	34	(.00676)		

Note. Values enclosed in parentheses represent mean square errors.

***$p < .001$

Table F2

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of MAX +M-L

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	1.95	.32	.181
P within-group error	17	(.127)		
		Within subjects		
Task Difficulty (D)	1	63.9***	.89	.001
P x D	1	1.53	.29	.233
D within-group error	17	(.140)		
Trial (T)	2	.425	.16	.657
P x T	2	.095	.077	.910
T within-group error	34	(.00703)		
D x T	2	.388	.15	.681
P x D x T	2	.251	.12	.780
D x T within-group error	34	(.0188)		

Note. Values enclosed in parentheses represent mean square errors.

***$p < .001$

Table F3

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of MAX -A-P

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	.448	.16	.512
P within-group error	17	(.186)		
		Within subjects		
Task Difficulty (D)	1	82.3***	.91	.001
P x D	1	.309	.13	.586
D within-group error	17	(.0596)		
Trial (T)	2	.644	.19	.531
P x T	2	1.48	.10	.242
T within-group error	34	(.00430)		
D x T	2	.231	.11	.795
P x D x T	2	.118	.084	.889
D x T within-group error	34	(.00919)		

Note. Values enclosed in parentheses represent mean square errors.

***$p < .001$

Table F4

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of MAX –M-L

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	94.5***	.19	.001
P within-group error	17	(.145)		
		Within subjects		
Task Difficulty (D)	1	86.4***	.91	.001
P x D	1	.541	.18	.472
D within-group error	17	(.0831)		
Trial (T)	2	2.63	.37	.086
P x T	2	1.81	.31	.180
T within-group error	34	(.00415)		
D x T	2	1.26	.26	.296
P x D x T	2	.487	.17	.619
D x T within-group error	34	(.0137)		

Note. Values enclosed in parentheses represent mean square errors.

***$p < .001$

Table F5

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of ABS AVG A-P

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	1.23	.26	.282
P within-group error	17	(.0136)		
		Within subjects		
Task Difficulty (D)	1	34.5***	.82	.001
P x D	1	.036	.045	.852
D within-group error	17	(.0102)		
Trial (T)	2	.574	.18	.569
P x T	2	3.39*	.41	.045
T within-group error	34	(.000645)		
D x T	2	.074	.063	.928
P x D x T	2	1.35	.27	.272
D x T within-group error	34	(.000801)		

Note. Values enclosed in parentheses represent mean square errors.

*$p < .05$. ***$p < .001$

Table F6

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of ABS AVG M-L

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	2.35	.35	.144
P within-group error	17	(.0284)		
		Within subjects		
Task Difficulty (D)	1	38.6***	.83	.001
P x D	1	2.52	.36	.131
D within-group error	17	(.0307)		
Trial (T)	2	1.92	.32	.162
P x T	2	1.02	.24	.371
T within-group error	34	(.00775)		
D x T	2	2.23	.34	.118
P x D x T	2	1.26	.26	.296
D x T within-group error	34	(.0124)		

Note. Values enclosed in parentheses represent mean square errors.

***$p < .001$

Table F7

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of RMSE A-P

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	.215	.11	.649
P within-group error	17	(.0377)		
		Within subjects		
Task Difficulty (D)	1	.566	.18	.642
P x D	1	.001	.00	.971
D within-group error	17	(.000114)		
Trial (T)	2	3.90*	.43	.030
P x T	2	1.88	.32	.168
T within-group error	34	(.00000947)		
D x T	2	3.70*	.42	.035
P x D x T	2	2.46	.36	.101
D x T within-group error	34	(.0000146)		

Note. Values enclosed in parentheses represent mean square errors.

*$p < .05$

Table F8

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of RMSE M-L

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	.259	.12	.617
P within-group error	17	(.0209)		
		Within subjects		
Task Difficulty (D)	1	1.19	.26	.290
P x D	1	1.04	.24	.322
D within-group error	17	(.0167)		
Trial (T)	2	1.07	.24	.354
P x T	2	1.14	.25	.3332
T within-group error	34	(.00861)		
D x T	2	1.11	.25	.341
P x D x T	2	1.08	.25	.350
D x T within-group error	34	(.00871)		

Note. Values enclosed in parentheses represent mean square errors.

Table F9

Analysis of Variance for the 2 (History of Pain) x 2 (Task Difficulty) x 3 (Trial) analysis of PATH

Source	*df*	*F*	η	*p*
		Between subjects		
History of Pain (P)	1	.415	.15	.528
P within-group error	18	(179.6)		
		Within subjects		
Task Difficulty (D)	1	32.4***	.80	.001
P x D	1	.097	.071	.758
D within-group error	18	(1178)		
Trial (T)	2	.310	.13	.735
P x T	2	.241	.11	.787
T within-group error	36	(60.09)		
D x T	2	.282	.12	.756
P x D x T	2	.193	.10	.825
D x T within-group error	36	(80.2)		

Note. Values enclosed in parentheses represent mean square errors.

***$p < .001$