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Biomechanical characteristics of low back tissues during trunk flexion-extension

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BIOMECHANICAL CHARACTERISTICS OF LOW BACK TISSUES DURING TRUNK FLEXION-EXTENSION

A Dissertation

Submitted to the Graduate Faculty of the Louisiana State University and Agricultural and Mechanical College in partial fulfillment of the requirements for the degree of Doctor of Philosophy

in

The Department of Kinesiology

by

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ABSTRACT

Analysis of the mechanical and neural regulatory mechanisms of the flexion-relaxation phenomenon (FRP), observed in deep trunk flexion, was performed since it is believed these mechanisms provide insight into the causes of low back injury and pain. Three methods were used to analyze the behavior of the lumbar tissues during trunk flexion-extension exercises: 1) active continuous cyclic movement, 2) acute cyclic movement at different orientations, and 3) passive continuous cyclic movement. All activities were performed at a rate of 0.1 Hz (6 cycles·min\(^{-1}\)) while monitoring the surface electromyogram (EMG) of the lumbar paraspinal muscles. Abdominal, hamstring, and quadriceps muscle activities were also monitored during acute cyclic movement. Trunk inclination and lumbar flexion angles were time synchronized with the recorded EMG signals. Increases in the myoelectric silent period with respect to inclination and flexion angles were apparent over time (p < 0.001) during the active continuous cyclic activity. Muscular fatigue and constant gravitational loading of the system were thought to influence the observations. Acute cyclic movements of trunk flexion-extension were performed in standing and supine positions. In standing, abdominal EMG activity increased when silent periods were present in lumbar paraspinal and hamstring muscles. Performance of flexion-extension from the supine position resulted in sustained silence of the paraspinal muscles once gravitational load was presented as the trunk flexed beyond vertical into deep flexion. EMG activity within the paraspinal muscles increased while extension was executed. During passive continuous cyclic movement the trunk motion was controlled by an external mechanical system to isolate the movement from fatigue. A significant decrease in the torque supplied by the posterior passive tissues was observed. EMG amplitudes remained relatively low during the passive session. A significant increase in the EMG amplitude and frequency was observed during active flexion movements performed after the passive session. Evidence suggests the primary
control mechanism of the FRP to be mechanical in origin for acute loading. Decrease stability of the spine may occur with prolonged repetitive trunk flexion-extension. The increased duration of the FRP during continuous cyclic flexion-extension suggests neural mechanisms may supersede mechanical mechanisms during repetitive lifting activities.
CHAPTER 1: INTRODUCTION

Injuries to the low back are prevalent in the general population of the western world. Back injury is the highest work related incident reported by employers in the United States (US Dept of Labor, 2003; 2004) and other industrialized nations (Maetzel & Li, 2002; Maniadakis & Gray, 2000). Low back pain (LBP), a symptom of injury, also leads to the greatest number of days away from work at a cost of over $20 billion dollars a year in employee wages and treatment (Kelsey, White, Pastides, & Bisbee, 1979; Maetzel & Li, 2002). The etiology of LBP is multifaceted and requires a holistic approach to further explain how this affliction is presented. Many variables affect the incidence or progression of LBP. The primary variable is concerned with the mechanical aspects of the system (i.e., compression, tension, shear, torsion, rate of loading), but also includes physiological, psychological, socioeconomic, and psychosocial factors (Adams & Dolan, 1996; Adams, Mannion, & Dolan, 1999; Fergusson & Marras, 1997; Granata & Marras, 1999; Magni, 1984; Mangora, 1970a; Mangora, 1970b; Svensson, 1982). These factors can be separated into intrinsic and environmental considerations based upon the morphology of the individual and the demands of the task (Andersson, 1981; Chaffin & Park, 1973; McNeill, Warwick, Andersson, & Schultz, 1980; Morris, Lucas, & Bressler, 1961; Porter, Adams, & Hutton, 1989).

Many ideas related to intrinsic and environmental factors are presented to explain the incidence and etiology of LBP and disorder. Among these ideas from the standpoint of the individual are properties of the muscle, hydrostatic changes within the vertebral discs, neural impingement, and ligament laxity (Bonato, Ebenbichler, Roy, Lehr, Posch, Kollmitzer, et al., 2003; Brisby, 2003; Lu, Hutton, & Gharpuray, 1996; Solomonow, Zhou, Baratta, Lu, & Harris, 1999). Task dependent factors related to LBP and disorder include time history of movement, the repetitive nature of the task, the position of the body during movement, and the lifting technique.
Fatigue between different muscle groups may contribute to LBP and disorder (Andersson, 1981; Granata & Marras, 1999; Hermann & Barnes, 2001; Kumar, 2001; Marras, 2000; Marras, 2005). The paraspinal muscles are responsible for maintaining stability and structural integrity of the trunk-spine system. Strength deficits may occur in the muscles and delay muscle activation when the system is perturbed (Taimela, Kankaanpää, & Luoto, 1999). Susceptibility to low back injury increases when the muscles are incapable of sustaining the external loads, leading to differential activation of the trunk and leg muscles (Andersson, Örtengren, & Nachemson, 1976; Davis & Troup, 1964; Morris et al., 1961). This effect is transient, however, as the fatigued state recovers within a brief time period (Kuorinka, 1988; Petrofsky & Lind, 1980). Central and peripheral fatigue may be superficial to other underlying mechanisms that contribute to LBP. However, the fatigued state of the system presents difficulties in determining these mechanisms and their corresponding neuromuscular adaptations.

The neuromuscular response of the lumbar paraspinal muscles to anterior trunk flexion provides a window to study the intrinsic mechanisms of LBP and disorder. There is a myoelectric deactivation observed in the paraspinal muscles when deep trunk flexion is performed in healthy individuals (Allen, 1948). Contrary to these observations, when chronic LBP patients perform trunk flexion there is continuous activity observed in the electromyogram (Golding, 1952). The myoelectric differences observed between healthy and patient populations may indicate the importance of this paraspinal activity during lifting tasks. The reduced electrical activity of the paraspinal muscles may provide an opportunity during lifting activities for an increased susceptibility of the system to low back injury and subsequent pain.
Background Information

At deep flexion angles the myoelectric activity of the lumbar paraspinal muscles decreases to low amplitude levels. The low amplitude myoelectric signal remains until this movement sequence is reversed when trunk extension is performed (Allen, 1948; Floyd & Silver, 1951; Floyd & Silver, 1955; Golding, 1952; Portnoy & Morin, 1956) (Figure 1.1). The muscle activation pattern illustrated is termed the myoelectric ‘silent period’, or flexion-relaxation phenomenon (FRP). FRP is referred to as an inhibition of the paraspinal muscles (Floyd & Silver, 1951; 1955).

![Trunk angle](image)

Figure 1.1. Example of flexion-relaxation phenomenon (left) in an individual (right) who performs flexion-extension of the trunk from a standing position.

The initial activation of the posterior muscles provides an extension moment to offset the flexion moment supplied by the gravitational force applied at the lower lumbar region (Gupta, 2001; Kippers & Parker, 1984; McGill & Kippers, 1994; Morris, 1973). However, further flexion of the trunk beyond certain angles (70°, for example) results in a decreased level of electrical activity in the lumbar muscles (Mathieu & Fortin, 2000; Sarti, Lisón, Monfort, & Fuster, 2001; Schultz, Haderspeck-Grib, Sinkora, & Warwick, 1985; Shirado, Ito, Kaneda, & Strax, 1995).
The initiation of the myoelectric silent period is currently believed to be of either a mechanical or neural origin. The mechanical hypothesis regarding the myoelectric silent period incorporates the transfer of load between the lumbar muscles and the lumbar passive tissues: ligaments, dorsal-lumbar fascia, and intervertebral discs (Allen, 1948; Floyd & Silver, 1951; Floyd & Silver, 1955; Golding, 1952; Kippers & Parker, 1984). Others have argued that load sharing may also occur between muscle groups (Andersson, Oddsson, Grunström, Nilsson, & Thorstensson, 1996; Touissaint, de Winter, de Haas, de Looze, van Dieën, & Kingma, 1995) since evidence to support a complete transfer of the load to the ligaments has been inconclusive (Potvin, McGill, & Norman, 1991). Inter-abdominal pressure (IAP) is speculated to provide additional support to the spine during lifting movements without further activation of posterior muscles (Cholewicki, Juluru, & McGill, 1999; Cholewicki, Jurulu, Radebold, Panjabi, & McGill, 1999). No relationship between IAP and FRP has been noted in the literature (McGill & Kippers, 1994). As trunk flexion angle increases, the ligaments along the dorsal side of the spinal column are strained. The increased tension that accompanies the elongated tissues decreases the necessity of the lumbar muscles in resisting the influence of gravity acting at the trunk, and results in an electrical silence of the muscles.

The presence of a mechanical load-sharing mechanism is still speculative in human models. Over time, the mechanical properties of the passive tissues could be modified due to the prolonged nature of some working environments. Repetitive loading of the ligaments in animal and cadaver models results in modified mechanical behavior of the tissues. Sustained loading of ligaments also decreases the stiffness of the tissues over time (Chazal, Tanguy, Bourges, Gaurel, Escande, & Guillot, 1985; Dumas, Beaudoin, & Droi, 1987; Goel, Voo, Weinstein, Liu, Okuma, & Njus, 1988; Hukins, Kirby, Sikoryn, Aspen, & Cox, 1990). This evidence suggests that continuous loading can be detrimental to the health of the spinal system.
Research concerning the effects of continuous loading on the human trunk-spine system has indicated, indirectly, that the passive tissues may be compromised during movement. Solomonow, Baratta, Banks, Freudenberger, and Zhou (2003) reported the duration of the myoelectric silent period decreased after 10 minutes of sustained trunk flexion-extension. Dickey, McNorton, and Potvin (2003) also observed a decreased silent period duration after continuous cyclic trunk loading with external loads. Over time, the constant requirement of the ligaments to maintain tension results in tissue lengthening, and decreases their ability to maintain tension at the same flexion angles. Nevertheless, Dickey et al. were not able to factor the effects of fatigue and the confounding external load from their results. It still remains unknown what contribution the passive tissues play in resisting the gravitational torque applied at the trunk, and whether the passive tissues are compromised.

External loading during trunk flexion is used to examine the mechanical load-sharing hypothesis in acute and repetitive loading environments. Reports are not consistent regarding the results of this loading as the duration of the myoelectric silent period has been observed to either decrease (Dickey et al., 2003; Kippers & Parker, 1994; Tanii & Madsuda, 1985), or not change (Floyd & Silver, 1955; Sarti et al., 2001). This evidence indicates that mechanical load sharing is partially responsible for explaining the silent period during acute trunk flexion. These observations do not reconcile the effects of prolonged cyclic loading and the role of the flexion-relaxation phenomenon with possible neuromuscular adaptations within the system.

The second hypothesis brought forward to explain the flexion-relaxation phenomenon indicates the importance of the neural system and its integration with the passive tissues. Sensory receptors (mainly mechanoreceptors) embedded within the ligaments and joint capsules respond to length and tension changes at the articulations (Andrew & Dodt, 1953; Cavanaugh, Ozaktay, Yamashita, & King, 1996; Ekholm, Eklund, & Skoglund, 1960; Gray & Mathews, 1951; Grigg...
The small diameter afferents of the sensory receptors serve an important regulatory function in assisting with the modulation of muscle behavior (briefly illustrated in Figure 1.2 in a very simplified fashion). In addition, receptors within the muscles also provide a possible means of inhibiting the activity of the motorneurons (Floyd & Silver, 1955; Gardland, 1991; Garland, Garner, & McCombs, 1988) (Figure 1.2).

Recent animal research has identified a possible link between the mechanical changes within the passive tissues and the joint receptors (Claude, Solomonow, Zhou, Baratta, & Zhu, 2003; Solomonow et al., 1999; Williams, Solomonow, Zhou, Baratta, & Harris, 2000). Prolonged static and cyclic loading of the supraspinous ligament results in a decreased reflexive response in the multifidus muscles (Solomonow et al., 1999; Williams et al., 2000). The modified mechanical properties of the passive tissues alter the tension and length of the

Figure 1.2. Schematic of the general neural pathways from afferent receptors in active and passive tissues to the central nervous system (CNS). The CNS interprets the information from these afferents and sends electrical impulses (Efferent) to the muscles in response to the stimulus accordingly. Inhibitory responses are not denoted as these are regulated in the CNS. Blue lines denote stimulation due to tension, while red lines denote stimulation due to length changes.
biomaterial and simultaneously delay the response of the mechanoreceptors to these stimuli. These *in vivo* studies provide greater understanding of the relationship between the active and passive tissues for application in human models. Although the influence of the mechanoreceptor afferents in muscle behavior is supported in animal research the impact of this neural mechanism on the flexion-relaxation phenomenon is sparse in human models (Indahl et al., 1997).

The integration of mechanical and neural mechanisms may provide a better understanding of the behavior of the lumbar muscles. Changes of the mechanical properties within the passive tissues are believed to affect the initiation of embedded afferent joint receptors to environmental stimuli. Obviously, the control of muscle behavior is not isolated solely to one or two factors, but to the relationship between all components such as the muscles, passive tissues, peripheral and central nervous systems. These associations must be taken into consideration when researching the mechanisms underlying the flexion-relaxation phenomenon.

The flexion-relaxation phenomenon is pertinent to healthy individuals as this event may increase susceptibility to low back injury. Individuals with LBP lack a myoelectric silent period when they perform anterior trunk flexion (Golding, 1952; Shirado et al., 1995). It is unclear whether mechanical instability or modified neural adaptations lead to injury and pain in these individuals when prolonged lifting activities are performed. Recently, the flexion-relaxation phenomenon was used as an instrument to analyze the progression of patient health in a clinical setting (Neblett, Mayer, Gatchel, Keeley, Proctor, & Anagnostis, 2003). Evidence suggests that current therapy modalities that stimulate receptors in the zygapophysial joints greatly enhance the health progression of chronic LBP patients (Dreyfuss, Dryer, & Herring, 1995; Garvey, Marks, & Wiessel, 1989). Further analysis of the mechanical and neural mechanisms is necessary to assist in the prevention and rehabilitation of injury and LBP in individuals predisposed to this condition.
Experiments

The experiments reported in the following three chapters were designed to incorporate current knowledge of the flexion-relaxation phenomenon by manipulating the flexion-extension tasks. These procedures were utilized to analyze the prolonged movement and the corresponding changes in the lumbar paraspinal muscle behavior. It is believed that the modified behavior of the paraspinal muscles occurs due to a combination of mechanical and neural mechanisms. The influence of the flexion-relaxation phenomenon and the possibility of injury during prolonged flexion-extension movements were previously mentioned (Solomonow et al., 2003). This is important since many people in labor intensive work environments are introduced to continuous and repetitive tasks on a daily basis (Andersson, 1981). The factors that need to be considered involve the movement itself, fatigue and its influence on the muscle behavior, and the possibility that mechanical and neural mechanisms are intertwined in this endeavor. In an effort to determine the role of each mechanism it is important to first isolate the variables that are hypothesized to influence the activity of the paraspinal muscles.

It is important to study the flexion-extension movement in the absence of external loads to isolate how the movement itself influences the system’s behavior. Currently, it is unknown how the flexion-relaxation phenomenon will respond to constant and repetitive cyclic loading of the trunk without the application of external loads. However, the flexion-relaxation phenomenon may also be influenced by possible changes in the properties of the passive tissues due to gravitational flexion moment. Fatigue is also a consideration since this may influence the activation of the paraspinal muscles (Garland, 1991).

Gravitational load acting on the trunk influences the flexion-relaxation phenomenon. The mechanical load-sharing hypothesis states that the trunk’s load due to gravity is shared between active and passive tissues (Kippers & Parker, 1984). Increasing the length within the posterior
tissues also results in increased tension irrespective of the amount of gravitational loading. Modifying the position of the body in relation to the gravity vector can provide insight to the mechanisms that influence the myoelectric silent period.

Previously mentioned was the confounding effect of fatigue in the analysis of mechanisms that may contribute to LBP and disorder. Fatigue significantly influences the paraspinal muscle behavior during continuous cyclic loading of the trunk, thus neuromuscular adaptations due to other mechanisms are difficult to ascertain (Dickey et al., 2003). Controlling the movement of the trunk while minimizing the muscular activity during continuous motion can decrease fatigue and allow for the analysis of other mechanisms.

It is also suggested that the posterior passive lumbar tissues are compromised during prolonged cyclic flexion-extension (Solomonow et al., 1999). Indirect evidence indicates the possibility that the passive tissues become compromised and lead to instability in the spine (Toussaint et al., 1995). The hypothesized compromise in the passive tissues has not been documented in human models using force monitoring systems. If the passive tissues are compromised then a modified and compensatory neuromuscular adaptation would develop in the trunk-spine system.

Repetitive cyclic loading of the low back tissues is hypothesized to affect the neuromuscular response of the system over time (Kumar, 2001). It is further hypothesized that the mechanical properties of the posterior passive tissues are modified as a result of repetitive cyclic trunk flexion-extension movements (Lu et al., 1996). Modulation of the low back muscle activity is thought to occur when continuous trunk flexion-extension is performed while free-standing. Factors that contribute to the modified myoelectric activity are not only the cyclic repetition of the movement, but also muscular fatigue and the effects of gravity on the system. These factors influence the behavior of the system while masking any contribution the passive
tissues incorporate to mechanical and neural mechanisms. Thus, it is important to factor these variables into the scope of the neuromuscular adaptations that are believed to occur.

The following three chapters are designed to further analyze the flexion-relaxation phenomenon by examining the previously mentioned hypotheses. Each chapter identifies a specific factor, or factors, that are believed to influence the myoelectric silent period. The first experiment (Chapter 2) identifies the behavior modifications of the paraspinal muscles through continuous cyclic trunk flexion-extension movement. The influence of the gravity vector on the flexion-relaxation phenomenon and the implementation of further compensatory mechanisms are studied in the second experiment (Chapter 3). Tension within the passive tissues of the posterior low back region is analyzed in the third experiment (Chapter 4) along with a possible neuromuscular adaptation in the paraspinal muscles. Synthesis of the conclusions presented in these chapters is presented in the last discussion chapter (Chapter 5).

References


CHAPTER 2. FLEXION-RELAXATION RESPONSE TO CYCLIC LUMBAR FLEXION

Introduction

Workers subjected to occupational activities requiring prolonged periods of cyclic and static lumbar flexion (warehouse workers engaged in loading/unloading, assembly line workers, farm and concrete workers, etc.) have been reported with unusually high rates of low back disorders (LBD) (NIOSH, 1999; US Department of Labor, 1995). From an epidemiological standpoint, therefore, cyclic and static lumbar flexion are risk factors for the development of LBD (Kumar, 2001; NIOSH, 1999; US Department of Labor, 1995). The recent efforts have focused on identifying the above processes in a highly controlled feline model (Claude, Solomonow, Zhou, Baratta, & Zhu, 2003; Jackson, Solomonow, Zhou, Baratta, & Harris, 2001; Solomonow, Eversull, Zhou, Baratta, & Zhu, 2001; Solomonow, Hatipkarasulu, Zhou, Baratta, & Aghazadeh, 2003b; Williams, Solomonow, Zhou, Baratta, & Harris, 2000). To date, it has been found that both static and cyclic lumbar flexion in a feline model elicited creep in the viscoelastic tissues simultaneously with spasms in the multifidus (Claude et al., 2003; Solomonow et al., 2003b). In the rest period following static or cyclic flexion a significant hyperexcitability of the multifidus muscles developed in parallel with acute inflammation. The model developed by Solomonow et al. (2003b) predicted that full recovery of muscular hyperexcitability and creep may take up to 48 hours. The data provide experimental evidence suggesting that such occupational activities result in a neuromuscular disorder, confirming epidemiological data.

It is of interest to assess if a similar disorder could be elicited in healthy human subjects during cyclic lumbar flexion. Extensive research has been conducted regarding creep in the spinal viscoelastic tissues of humans and cadavers after periods of loading in flexion (Adams, 2001).

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Dolan, & Hutton, 1987; McGill & Brown, 1992; Towmey & Taylor, 1982). These studies have indicated that creep was evoked in the ligaments and discs during the loading. In cadavers, repeated flexion over a 5 min period was shown to also induce creep (Adams & Dolan, 1996). Recently, Solomonow, Baratta, Banks, Freudenberger, and Zhou (2003a) demonstrated that creep developed during static lumbar flexion in healthy humans resulted in a neuromuscular disorder consisting of spasms and significant changes in muscle activity. However, the link between the development of a neuromuscular disorder due to laxity/creep in the viscoelastic tissues has not been demonstrated in humans during cyclic activities.

The performance of anterior lumbar flexion in humans has been associated with the flexion-relaxation phenomenon (FRP) (Allen, 1948; Fick, 1911; McGill & Kippers, 1994; Shirado, Ito, Kenada, & Strax, 1995). As the trunk flexes anteriorly, the muscular activity in the low back diminishes to complete silence with increased flexion angles. Muscle activity resumes at some point in time during trunk extension to upright posture. The reaction of this phenomenon to prolonged cyclic motion has been reported in only a few human studies (Dickey, McNorton, & Potvin, 2003; Sarti, Lison, Monfort, & Fuster, 2001; Schultz, Haderspeck-Grib, Sinkora, & Warwick 1985). Schultz et al. (1985) demonstrated that myoelectric activity of erector spinae muscles during anterior flexion decreased even when external loads were applied. This occurred even though they predicted that greater muscular contraction forces were needed at greater flexion angles. Sarti et al. (2001) observed that increasing the flexion speed delayed the initiation of the FRP to a larger flexion angle. They also noted that the addition of loads did not affect the initiation of the FRP compared to no-load conditions. Dickey et al. (2003) reported an increase in the angle of initiation of the FRP after loads had been added during prolonged cyclic flexion-extension. The conclusions of Sarti et al. (2001), Schultz et al. (1985) and Dickey et al. (2003) are conflicting as the first group did not observe changes due to added external load whereas the
second and third groups did, but in opposing directions. Addition of external loads during a prolonged cyclic flexion may result in a transient response as well as a steady state response from the neuromuscular system which may be the source of the conflicting results. Furthermore, it is not clear if the subjects were subjected to a constant load paradigm or a constant displacement paradigm (Goel, Wilder, & Pope 1995). The first allows subjects to flex deeper at a constant load whereas the second stipulates that the deepest angle will remain constant over time. Significant differences in the results and conclusions exist between the two paradigms and should be considered carefully when comparing various studies (Goel et al., 1995).

In summary, it is evident that the duration of the silent period of the FRP is dependent on the speed of flexion (Sarti et al., 2001). External loads were also shown to change the duration of the silent period, although the data is conflicting from no change (Sarti et al., 2001) to shortened silent period (Dickey et al., 2003) and to longer silent period (Schultz et al., 1985). Data showing possible changes in the silent period over time as cyclic flexion-extension is performed without modifying the load or speed are lacking. The presence of spasms during cyclic flexion was also not addressed to date.

The purpose of the study was to investigate the response of the FRP to sustained cyclic lumbar flexion without external load. It was hypothesized that laxity of the lumbar viscoelastic tissues developing during cyclic flexion-extension in healthy normal subjects will result in an altered period of electromyographic (EMG) silence of paraspinal muscles activity during deep flexion. It was also hypothesized that spasms as well as changes in the EMG intensity may occur during the cyclic flexion-extension exercise.
Methods

Subjects

Twelve male subjects volunteered to participate in a protocol approved by the Institutional Review Board. The subjects ranged in age from 19 to 31, with a mean age of 23.9 (± 4.8) years. Mean height and weight of the participants were 181 (± 6.8) cm and 89.8 (± 29) kg, respectively. None reported previous episodes of spinal disorders.

Instrumentation

Surface EMG data were collected using RUN Technologies Equipment (Laguna Hills, CA, USA). The EMG signals were detected and collected bilaterally by pre-gelled Ag–AgCl electrode pairs applied at the L2-3 and L4-5 levels over the paraspinal musculature and 3.5 cm lateral from the midline. The interelectrode distance was 4 cm from center to center and each electrode was of 1.0 cm diameter. Electrodes were oriented longitudinally along the muscles. A reference electrode was placed on the right anterior superior iliac crest. The EMG signals were amplified up to 20,000 with a frequency bandpass of 10–480 Hz, and a CMRR of 100 dB. The resulting signal was sampled at 960 samples per second via a 12 bit analog to digital conversion board and stored for later processing.

Kinematic data were collected at 60 frames per second using a motion analysis system (Motion Analysis Corporation, Santa Rosa, CA, USA). A single camera was positioned 1.0 m away from the subject at waist height with a direct view of the subject’s left profile. Reflective spheres of 8 mm diameter were placed at the left greater trochanter, lateral midline along the iliac crest, and on the lateral midline along the lower palpable edge of the rib cage. Kinematic and EMG data were synchronized by Eva 6.0 software of the Motion Analysis System.
Protocol

The skin was cleansed and lightly abraded with alcohol prep pads before EMG electrode attachment. The electrodes and markers were positioned as described above, and a signal check was performed to ensure quality EMG signals and marker detection. The subject was given instructions regarding the task, and the experimenter demonstrated the task until satisfied that the subject understood the protocol. A cycle consisted of 5 seconds of controlled anterior flexion from erect posture to full flexion, followed by 5 seconds of controlled extension to full erect posture. Full flexion was denoted as touching the toes with the finger tips during each cycle. This range of flexion was defined to simulate flexion to the same angle, such as that found in an occupational setting when loading/unloading boxes from one height to another. The cadence of each phase was governed by a metronome which gave off an auditory signal (beep) every second. The subjects were instructed to reach the point of full flexion on the fifth beep, reach erect posture on the tenth beep, and to maintain a consistent tempo during the entire cycle. The total time for each cycle was 10 seconds. Each subject performed this cycle continuously for 9 minutes. The first three cycles of each minute were collected and used for future analysis.

Analysis

Kinematic data were tracked and smoothed by using a zero lag, dual pass, Butterworth digital low pass filter with a cut off frequency at 1 Hz. Two angles were considered to be of interest: the angle of trunk inclination, defined as the angle between the top two markers and the vertical line to ground through the marker on the iliac crest; and the angle of lumbar flexion, defined as the angle of trunk inclination minus hip flexion angle. Subsequently, flexion refers to the angle representation of lumbar flexion, and inclination refers to the trunk inclination angle relative to the vertical (Solomonow et al., 2003a).
The recorded EMG data were full-wave rectified and smoothed at 0.5 Hz with a dual pass, zero lag Butterworth filter to yield linear envelopes. The resulting EMG linear envelope and angles of interest were then plotted and further analyzed to determine the following parameters: inclination and flexion angles at EMG cessation during flexion, inclination and flexion angles at deepest anterior flexion, and the inclination and flexion angles corresponding to EMG initiation during extension. The mean amplitudes of rectified and smoothed EMG during flexion and extension were normalized with respect to the value of the EMG during the first extension phase of minute one. The EMG magnitude during extension was chosen for normalization since it was the largest magnitude observed within a cycle.

A threshold level of 5% of maximal magnitude of the linear envelope was used to initially determine the beginning and end of the flexion-relaxation silent period. The judgment of the presence of EMG activities was based on the overall comparison of the raw as well as the smoothed EMG data. The linear envelope was primarily used since raw EMG signals may have had spasms or remnants of electrocardiogram (ECG) that could distort the raw EMG threshold level. It should be understood that the linear envelope in no way replaces the raw EMG signal, but was used as a guide based on the raw signal.

Analysis of variance with repeated measures was used to evaluate the changes of each variable within subjects over time. The alpha level was set at 0.05. Trend analysis was used to analyze the relationship between concerned parameters and the progress of the experiment within each subject over time.

Results

There were no differences observed in myoelectric activity from the paraspinal EMG data collected at the four collection sites of each subject. Data from all sites were subsequently pooled in the following analysis.
A typical recording of paraspinal muscle EMG, trunk inclination, and lumbar flexion during a single cycle of flexion-extension is depicted in Figure 2.1. EMG discharge was observed from the erect position to mid-flexion followed by EMG silence from mid-flexion to mid-extension. The EMG activity initiated, thereafter, to the next erect position.

EMG magnitudes during extension were greater than the magnitudes during flexion. A paired t-test confirmed the mean magnitude of the EMG activity during extensions was significantly greater than that during flexions (1.02 ± 0.11 and 0.72 ± 0.22, respectively, p < 0.0001). In addition, it could be observed that the EMG-on angles were closer to the deepest anterior flexion angles than were the EMG-off angles. Paired t-tests were used to confirm this observation for both inclination and flexion angles. For the inclination angle, the angular difference between deepest anterior flexion and EMG-off angles (22.2 ± 11.5) was significantly greater (p < 0.013) than the angular difference between deepest anterior flexion and EMG on angles (18.8 ± 13.5). For the flexion angle, the angular difference between the deepest anterior flexion and EMG-off angles (11.3 ± 5.9) were also significantly greater (p < 0.0067) than the angular difference between the deepest anterior flexion and EMG-on angles (9.4 ± 6.9).

Figure 2.2 illustrates the change in EMG activity over time. The most important change to be observed is the increased silent period with prolonged cyclic exercise. During the silent period, spasms appeared as random EMG activities. These spasms were found to be more prominent towards the latter portion of the exercise.

The average of inclination and flexion EMG-off angles over time are depicted in Figure 2.3. There were significant differences observed in the angle of cessation of EMG activity during anterior flexion in both inclination and flexion angles over trials (F8, 100 = 2.80, p < 0.009, and F8, 100 = 3.07, p < 0.001, respectively). Inclination EMG-off angles changed from 68.6 (± 13.5) in the initial minute to 63.8 (± 17.0) in the ninth minute; while flexion EMG-off angles
changed from 46.2 (± 8.7) to 43.7 (± 10.3) degrees during the course of 9 minutes of exercise.

The trends of decreasing angles at EMG cessation over time were linear for both inclination and flexion angles (F$_{1, 81} =$ 16.3, p < 0.0001, and F$_{1, 81} =$ 18.8, p < 0.0001, respectively).

Figure 2.1. A. Typical recording of EMG activity and its linear envelope during cyclic flexion-extension. Raw EMG and the linear envelope were used to estimate EMG-off and EMG-on. The extension phase used for normalization of the EMG linear envelope is also provided. B. The inclination (gray line) and flexion (green line) angle changes during cyclic flexion-extension. The solid vertical lines intersecting the EMG and inclination angles indicate various postures during the data recording process, and the vertical dashed lines indicating the estimated EMG-off (offset), EMG-on (onset), and corresponding angles.
Mean inclination and flexion EMG-on angles during extension are shown in Figure 2.4. The inclination EMG-on angles were reduced from the initial value of 76.7 (± 13.2) to 67.8 (± 18.0) degrees at the end of exercise, 50.6 (± 7.5) to 46.2 (± 9.2) degrees at the same time. The changes in initiation of EMG activity during extension were significant for both inclination and flexion angles (F$_{8, 100}$ = 3.24, p < 0.0031; and F$_{8, 100}$ = 3.68, p < 0.0011, respectively). A significant linear trend was observed for the initiation angle of EMG activity for both the inclination and flexion angles (F$_{1, 81}$ = 17.5, p < 0.0001; and F$_{1, 81}$ = 19.8, p < 0.0001, respectively).

Figure 2.2. Depiction of a typical subject’s EMG pattern over time. A representative cycle of the flexion-extension exercise is given per minute of a nine minute session. The solid line represents the inclination angle and gives an indication of when the EMG activity of each phase of the exercise occurs. The vertical dashed line indicates the point of deepest inclination angle. Spasms were found to be sporadic during the silence period of the EMG.
The angular excursion from the angle of EMG-off to the angle of EMG-on [(deepest angle - off angle) + (deepest angle - on angle)] was prolonged with exercise time for both inclination ($F_{8,100} = 2.96; p < 0.006$) and flexion angles ($F_{8,100} = 2.61; p < 0.014$), with linear trends prominent for each ($F_{1,81} = 19.14; p < 0.0001; F_{1,81} = 15.8; p < 0.0002$, respectively). Inclination angular excursion increased from approximately 29.7 ($\pm 12.7$) to 56.1 ($\pm 19.1$) degrees, while flexion angular excursion increased from approximately 14.9 ($\pm 5.3$) to 27.7 ($\pm 10.4$) degrees. The trend of the deepest inclination and flexion angles of each flexion-extension cycle are shown in Figure 2.5. The deepest inclination and deepest flexion angles did not change significantly over time ($F_{8,100} = 0.54, p > 0.82$, and $F_{8,100} = 0.80; p > 0.60$, respectively). The deepest inclination angles from the initial minute to minute 9 for inclination were 87.5 ($\pm 16.6$) and 93.8 ($\pm 16.9$) degrees, respectively, while the deepest flexion angles were 55.9 ($\pm 9.1$) and 58.8 ($\pm 8.5$) degrees, respectively. No significant trends for either angle measurement were present.

Magnitude of EMG activity was compared to the initial values in extension since the EMG magnitude during extension was the largest during a cycle. These relative values were standardized to each subject, compared over trials and shown in Figure 2.6. The EMG magnitude within flexion increased significantly over time, from 0.62 ($\pm 0.27$) to 0.87 ($\pm 0.23$) ($F_{8,100} = 6.86; p < 0.0001$).

Trend analysis was used to confirm that a linearly increasing EMG amplitude over time was present ($F_{1,81} = 39.7; p < 0.0001$). Significant EMG magnitude changes were also observed for extension during the trials; from 1.0 ($\pm 0.05$) to 1.1 ($\pm 0.12$) ($F_{8,100} = 2.45; p < 0.02$). In addition, a significant quadratic trend was present ($F_{2,81} = 5.62; p < 0.02$). Even though the amplitude increased significantly over time during flexion, the amplitude observed during extension was always greater than that of flexion (Figures 2.1 and 2.2).
Figure 2.3. EMG-off angle changes before myoelectric silence over time. The solid squares with whiskers represent the means and standard deviations of the angles. The left and right panels show the change in inclination and flexion angles, respectively, over trials during the flexion phase of the cycle. EMG signals ceased at significantly smaller angles of inclination and flexion over time.

Figure 2.4. EMG-on angle changes after myoelectric silence over time. The solid squares with whiskers represent the means and standard deviations of the angles. The left and right panels show the change in inclination and flexion angles, respectively, over trials during the extension phase of the cycle. Onset of EMG activity was delayed in the later stages during prolonged cyclic flexion-extension.
Figure 2.5. Deepest inclination and lumbar flexion angles over time. The solid squares with whiskers represent the means and standard deviations of the angles. Both the deepest inclination (left) and flexion (right) angles did not change over trials.

Figure 2.6. Normalized EMG amplitude within flexion (left) and extension (right) phases during cyclic flexion-extension. The solid squares with whiskers represent the means and standard deviations of the amplitude. The EMG amplitude during flexion and extension significantly increased over time.

Discussion

The major findings of this investigation consist of a significant increase in the angular excursion (or the silent period) between the EMG-off and EMG-on during a cyclic exercise over
9 minutes. An early cessation during anterior flexion and a subsequent delayed initiation of EMG activity during the following extension contributed to the longer silent period. Increased EMG amplitude was also observed during flexion and extension. Spasms were observed during the silent period although they were difficult to quantify due to the sporadic nature of the events. They were, however, more frequently observed towards the end of the exercise. It is clear that interpretation of data from integrated responses of active human subjects includes interaction of many components of the neuromuscular system (vestibular, visual, etc.) including several independent spinal reflexes. Any interpretations, therefore, should be considered in this complicated light until data from isolated stimulus and response become available, and placed in the right perspective.

Working with the feline model, Claude et al. (2003) observed spasms and gradually reduced EMG over the duration of cyclic lumbar flexion. We observed the spasms yet the EMG amplitude increased over time. The difference between Claude et al.’s (2003) study and this one is the fact that they employed passive lumbar flexion whereas our subjects perform active lumbar flexion using the musculature. Since the muscles were used continuously for 9 minutes, fatigue must have developed within. Indeed, several subjects noted that effect. It is well established that the amplitude of the EMG increases and the mean frequency of the power spectrum decreases as muscles fatigue (Lindstrom, Magnusson, & Petersen, 1970; Moritani, Nagata, & Muro, 1982). The gradual increase in the EMG amplitude during the cyclic flexion, therefore, may represent the effect of fatigue.

Another possibility could revolve around changes in the type of motor units active during the flexion-extension exercise. As time elapses, small or medium motor units with low innervation ratios may be complimented or replaced with the activity of larger motor units with high innervation ratio (Nardone & Schieppati, 1988). This will result in an increase in EMG
amplitude and with the associated increase in force. Indeed, as it will be discussed below, this can explain, in part, the results obtained in this study.

The FRP was explained by the load sharing mechanism between muscles and various viscoelastic tissues (ligaments, dorsolumbar fascia, discs, etc.). As the individual begins to flex forward, the posterior muscles are activated and offer resistance in order to prevent the head and torso mass, when subjected to gravity, from collapsing forward uncontrolled. Simultaneously, the viscoelastic tissues stretch and gradually develop increasing passive forces (Dolan & Mannion, 1994). As the passive forces exceed the magnitude of the head/torso load, the muscle forces are no longer required and become silent. Further flexion is accomplished by contraction of abdominal muscles.

Static or cyclic flexion-extension over time will develop creep or tension-relaxation in the viscoelastic tissues (Claude et al., 2003; Solomonow, 2004). The laxity developed in the tissues will result in changes in the forces developed at a given flexion angle. One can anticipate that deeper flexion will be needed for the viscoelastic tissues to develop the same force. The posterior muscles, therefore, should be expected to remain active longer before the posterior viscoelastic tissues take over the head/torso load. If an external load is introduced, a similar increase in the duration the muscles are active in the flexion phase is to be expected. Furthermore, an increase in muscular force or its associated EMG is required to offset the additional load.

Our previous report describing changes in the FRP due to 10 minutes of continuous unloaded static flexion conformed to the expectations delineated above (Solomonow et al., 2003a). The present study, however, describes an increasing silent period during the cyclic flexion-extension, which is in opposition to the expectations. Furthermore, Sarti et al. (2001) and Nelson, Walmsley, and Stevenson (1995) also failed to observe a shortened silent period during flexion-relaxation, as would be expected with increased load applied to the tissues. However,
Dickey et al. (2003) found a shortened silent period as the angle at which myoelectric silence initiated increased over time. Their results were dependent on increasing the external load during the flexion-extension exercise and could indicate that increased loading causes changes in the angle at which the flexion-relaxation phenomenon occurred. However, an increase in angle at the initiation of flexion-relaxation was also observed after loads had been removed. The load may have long lasting transient effect on the recruitment pattern of the muscles active in the flexion-relaxation phenomena. Another significant difference between Dickey et al. (2003) and this study that may explain the differences in the patterns of EMG silence in flexion pertain to the experimental requirements. Dickey and colleagues requested subjects to flex as deeply as possible with or without load (e.g., load control) whereas we limited subjects to a flexion angle associated with touching the toes with the fingertips (e.g., displacement control). Indeed, they observed a significant increase in the deepest flexion angle as compared to non-significant change in our study. The range of the excursion in deep flexion may have an impact on the changes in the flexion-relaxation pattern. The addition of external load in Dickey et al. (2003) and Sarti et al. (2001) may have had an added impact on muscular fatigue and the FRP response, as well. Due to the meaningful differences in the experimental conditions, comparison of our study with the others is not productive.

Previous research (Solomonow et al., 2001; Williams et al., 2000) has indicated that tension in lumbar ligaments decreases with prolonged static and cyclic lumbar flexion while the displacement of the ligaments remained constant. Consistent loading of the passive tissues over a prolonged period of time decreases the stiffness of the tissue and increases deformation (Jackson et al., 2001; Solomonow et al., 2001). Increased laxity of the passive tissues due to a decrease in tension (i.e., the supraspinous ligament) would not elicit the same neural response as when the tissues were taut. Stretch receptors in the posterior ligaments become stimulated when the
ligaments are stretched and afferent impulses are sent to initiate a spinal reflexive response by the extensor muscles (Gupta, 2001; Floyd & Silver, 1955; Kippers & Parker, 1984; Schultz et al., 1985; Shirado et al., 1995). There is evidence to show that the decreased activity of the stretch receptors is related to the development of creep, resulting in greater inhibition of the erectors spinae (Claude et al., 2003). Thus, the threshold level with which the mechanoreceptors are stimulated would be less likely to be attained, and a reduction in the amount of afferent impulse propagated to the muscles via spinal loops would occur. This decrease in afferent input decreases activity of the muscles and together with laxity in the ligament, leads to increased laxity at the joint (Claude et al., 2003; Solomonow, 2004). This threshold level change affects the mechanoreceptors, but not necessarily the threshold of the mechanoreceptors that act as nociceptors, as spasms in the later trials were observed.

Overall, the results of this study as well as from others (Sarti et al., 2001; Schultz et al., 1985) challenge the load sharing mechanism as the only component in the explanation of the FRP. The FRP seem to be substantially more complex as it may depend on the velocity of the flexion, changes in load, duration of cyclic activity (number of cycles) and mechanical properties of viscoelastic tissues (creep, tension-relaxation, hysteresis, response to rate of stretch, etc.), (Solomonow, 2004). It is clear that much more research is required to delineate the various input–output behaviors of the FRP in various conditions.

The increase in the EMG magnitude we observed here may represent an increase in muscular force as laxity is developed in the viscoelastic tissues. That may have compensated for the laxity of the tissues as time goes by during cyclic flexion. Indeed, our previous work (Solomonow et al., 2001; Eversull et al., 2001) confirms that EMG magnitude increases as compensation for changes in viscoelastic tissues mechanical function (e.g., laxity, hysteresis, etc.), with the absence of fatigue. Such explanation is tenable when considering the increase in
EMG magnitude as compounded response of increased force and partial fatigue or shift in the pool of the muscles active motor units between the slow and fast twitch groups (Nardone & Schieppati, 1988). One must recall that a significant time delay exists between the termination of EMG and the actual decay in force (Solomonow et al., 2001), which explains why the increase in the FRP excursion angle as observed from the EMG does not precisely represent when the muscle force actually decayed completely during the flexion. Muscle forces decay somewhat after the EMG terminated and in this case, it translates to a larger flexion angle. While this explanation clarifies that muscular forces were larger and remained longer to offset the effect of viscoelastic tissue creep/laxity during flexion, it does not explain why the EMG was delayed during extension. Obviously, another explanation must be offered, when it becomes available.

It is important to note that spasms were observed in Claude et al. (2003) study and here, indicating that micro damage developed in the fibers of the viscoelastic tissues (Frank, Amiel, Woo, & Akeson 1985). The changes in EMG amplitude, timing and spasm constitute a temporary neuromuscular disorder that may resolve with rest (Solomonow et al., 2003b). Nevertheless, prolonged cyclic lumbar flexion seems to elicit a neuromuscular disorder.

The observations in the study provide important biomechanical and physiological explanations to the development of low back disorder. The FRP is probably initiated through a load sharing mechanism from developmental or evolutionary perspectives, but various central and spinal reflexes during the sustained cyclic flexion and extension might have substantial interaction due to lax or creeped ligaments. The overall negative reaction (laxity and lower muscular forces) would further increase the possibility of damage to the lumbar ligaments and reduce the stability of the lumbar region. The accumulation of this effect over time would contribute to the development of chronic low back disorder.
In summary, cyclic lumbar flexion and extension develops laxity in the lumbar viscoelastic structures, increased silent period and the increased frequency of spasms towards the end of the exercise. The neurological compensations (enhanced muscle inhibition) of the tissues laxity were more observable than the mechanical compensations for load sharing. This further confirms the importance of the neurological synergy between ligaments and muscles in the control of movement and preservation of skeletal stability.

References


CHAPTER 3: FLEXION-RELAXATION RESPONSE TO GRAVITY

Introduction

The behavior of the lumbar paraspinal muscles has been studied previously during a variety of trunk movements, particularly cyclic trunk flexion-extension commencing from a standing position. Initially, the observed electromyographic (EMG) activity rises in the paraspinal muscles when the trunk deviates from an upright stance. Progressive decline of this signal occurs resulting in a myoelectric silent period as the trunk approaches deep flexion (Ahern, Follick, Council, Laser-Wolston, & Litchman, 1988; Allen, 1948; Kippers & Parker, 1984; Portnoy & Morin, 1956; Schultz, Haderspeck-Grib, Sinkora, & Warwick, 1985). The period of myoelectric silence in the paraspinal muscles, or flexion-relaxation phenomenon (Floyd & Silver, 1955), may be attributed to mechanical and neural mechanisms. It has been hypothesized that tension in the stretched passive tissues (dorso-lumbar fascia and posterior ligaments) is sufficient to support the gravitational load of the trunk during deep flexion (Allen, 1948; Floyd & Silver, 1955; Golding, 1952). This load sharing mechanism allows the paraspinal muscle activity to decrease. A neural reflexive response, from muscle spindles or joint receptors, may also influence flexion-relaxation (Floyd & Silver, 1951).

Both mechanical (Allen, 1948, Floyd & Silver, 1955, Golding, 1952) and neural mechanisms (Floyd & Silver, 1951) responsible for the flexion-relaxation phenomenon are supported in the literature. The amount of influence each mechanism has on the behavior of the lumbar paraspinal muscles during trunk flexion is not certain. Human models (Andersson, Oddson, Grundström, Nilsson, & Thorstensson, 1996; Dickey, McNorton, & Potvin, 2003; Golding, 1952; Kippers & Parker, 1984; McGill & Kippers, 1994; Solomonow, Baratta, Banks, Freudenberger, & Zhou, 2003) provide information supporting the mechanical load sharing
hypothesis. Animal models (Claude, Solomonow, Zhou, Baratta, & Zhu, 2003; Solomonow, Zhou, Baratta, Lu, & Harris, 1999; Williams, Solomonow, Zhou, Baratta, & Harris, 2000) offer evidence suggesting the occurrence of a neural adaptive response to the changing mechanical properties of the passive tissues. Olson, Li, and Solomonow (2004) suggested the mechanoreceptor afferents within the passive tissues contributed to an increased myoelectric silent period based on their human model observations. It is possible that both mechanisms work concomitantly. The influence of intra-abdominal pressure (IAP) cannot be dismissed when discussing these mechanisms as this provides support to the lumbar spine during trunk flexion (Cresswell and Thorstensson, 1989; Grillner, Nelson, & Thorstensson, 1978, Oddsssen and Thorstensson, 1990).

These models provide a means of interpreting how the viscoelastic tissues interact when external stimuli are introduced. A significant factor in the human models has been the influence of gravity acting upon the trunk and the load. The viscoelastic tissues respond to trunk moment changes. Distinguishing whether myoelectric silence is due to load sharing or a product of neural inhibition becomes problematic. Pre-loading the tissues prior to application of a gravitational load may provide a better understanding of the mechanisms involved in flexion-relaxation.

The purpose of this study was to explore the influence of gravitational loading orientation on the anterior and posterior lumbar muscles during trunk flexion-extension. Trunk flexion-extension beginning from either standing or supine positions allowed for differentiation of the influences of kinematic trunk motion and the gravitational force. It is hypothesized that the two different gravity vector orientations will elicit two different neuromuscular responses. In turn, these responses will reflect the different load sharing strategies and reflexive muscular responses from afferents in the viscoelastic tissues. If the neural hypothesis is true then gravitational
loading of the viscoelastic tissues after pre-loading will result in myoelectric silence of the paraspinal muscle during flexion and extension. It is anticipated that the new information may further the understanding of the flexion-relaxation phenomena as well as the processes that govern it for use in studying low back disorders.

Methods

Subjects

Thirteen male college students volunteered to participate in a protocol approved by the Institutional Review Board. The mean age, height, and body mass of the participants were 20 (± 1) yrs. (range 18 to 23 yrs.), 178 (± 6) cm (range 168 to 191 cm) and 82 (± 11) kg (range 73 to 115 kg), respectively. None of the participants had reported previous episodes of spinal disorders or low back pain.

Instrumentation

The surface electromyography data (EMG) were collected from the right lumbar paraspinal (LP), rectus abdominis (RA), external oblique (EO), rectus femoris (RF), semimembranosis (SM), and biceps femoris (BF) muscles. Electrodes on LP were placed at the L3-L4 level and 3.0 cm lateral from the spinous processes. We collected EMG activity of RA and EO at 3.0 cm and 15 cm lateral from the umbilicus, respectively, RF at 25 cm inferior from the right anterior superior iliac spine, SM at 25 cm superior from the medial femoral epicondyl, and BF at 25 cm superior to the lateral femoral epicondyl. EMG data were collected using an MA-300 system (Motion Lab Systems, Inc., Baton Rouge, LA, USA). Pairs of pre-gelled Ag-AgCl bipolar electrodes of 1.0 cm diameter were used in combination with a differential amplifier that has a gain up to 20,000. The interelectrode distance was 2.5 cm from center to center and electrodes were oriented longitudinally along the muscle fiber length. The range of the frequency bandpass was set at 10 – 480 Hz. The common mode rejection ratio (CMRR) was
100 dB. The sampling frequency was 960 Hz. Data were collected using a 12 bit analog to digital conversion board and stored for future processing.

Kinematics of the trunk and pelvis segments were monitored by a single infrared camera (Motion Analysis Corporation, Santa Rosa, CA, USA) positioned 3.0 m away from the participant with a direct view of his left profile. The camera collected kinematic data at 60 frames per second. Reflective spheres, 2.5 cm in diameter, were taped onto the skin on the left side at the lateral midline edge of the twelfth rib, lateral midline of the iliac crest, and greater trochanter. Kinematic and EMG data were synchronized by Eva 6.0 software of the Motion Analysis System.

Protocol

The participant’s skin was cleansed and abraded with alcohol prep pads before application of the electrode pairs to reduce skin impedance. The experimenter then placed the electrodes and markers as described above and checked the signals before testing to ensure quality EMG and marker detection.

The two testing conditions of trunk flexion-extension were initiated from two positions, standing and supine. Each testing session consisted of five trials of each condition performed randomly. To control for the effect of different types of footwear participants performed each condition barefoot.

A cycle of trunk flexion-extension constituted one trial. One cycle included five seconds of controlled flexion to attain deepest trunk flexion by touching the toes followed by five seconds of controlled extension to the starting position. A metronome set at one auditory beat per second was used to control the tempo of the movement. Participants attempted to achieve deepest trunk flexion on the fifth beat and the starting position on the tenth beat while maintaining a constant tempo during the cycle.
The individuals positioned their feet at shoulder width apart for each trial. During the supine condition the participant’s shoulders, pelvis, and feet were supported on three wooden platforms 3 cm in height (15 cm width). These platforms were used to prevent electrodes positioned on the low back and posterior thigh from contacting the floor.

During the supine condition the legs were constrained to control counterbalance due to the movement of the trunk. A towel was placed over the distal portion of both legs, about the ankles, to provide cushioning. A wooden box (2.3 kg) containing an 11.4 kg weight was positioned over the towel to restrain lower body movement. The location of the box and the weight did not interfere with the ability of each participant to touch his toes.

Analysis

Kinematic data were tracked and smoothed by using a zero lag fourth order Butterworth digital low pass filter with a cut-off frequency of 1 Hz. Two angles were considered to be of interest. The first was the lumbar flexion angle. This was defined as the relative angle between the line connecting the rib and iliac markers (trunk) and the line connecting the iliac and greater trochanter markers (pelvis). The second angle was the inclination angle, defined as the deviation of the trunk with reference to the vertical. The inclination angle was used to determine the relationship of body position with respect to the line of gravity (trunk moment) acting at the low back.

The recorded EMG data were full-wave rectified and smoothed with a low pass filter at 10 Hz using a fourth order zero lag Butterworth filter. The resulting linear envelopes were used for further analysis.

The EMG linear envelope and angles of interest were then plotted for visual inspection. The cessation and initiation landmarks of the EMG activity for each muscle group were then determined. The angles of peak flexion and peak inclination were then calculated. Kinematic
data were also used to determine the beginning and ending of each cycle during a trial. Once a complete cycle was determined its duration was normalized to a percentage of the complete cycle. This normalization allowed for a comparison of the kinematic and EMG parameters between trials and subjects.

A threshold level of 5% of maximal linear envelope of each muscle obtained during all the trials for both conditions was used to initially determine the beginning and the end of the myoelectric activity. Review of the raw, rectified, and smoothed EMG data was used to determine the presence of EMG activity. Raw EMG signals may have had remnants of electrocardiograph (ECG) that could have distorted the detection of initiation and cessation times. The linear envelope was used as a guide based on the raw signal and in no way replaced the raw signal.

All data that were normalized to a ratio of the complete flexion-extension cycle were tested for normality using a Wilk-Shapiro test. One-way analysis of variance with repeated measures was used to evaluate the results (Statistix Analysis Program). The alpha level was set at 0.05.

Results

Typical data and their relationship to flexion and inclination angles in standing and supine conditions are shown in Figure 3.1 (A and B). No differences were observed in the timing of peak flexion and inclination angle attainments during flexion-extension cycle between the two conditions (Table 3.1). Peak flexion angle was greater in the standing condition compared to supine condition ($F_{1,128} = 19.09, P < 0.0001$). Peak inclination angles were also greater in standing condition ($F_{1,129} = 10.37, P < 0.002$).
Standing Flexion-Extension

Figure 3.2 shows the timing of muscle activation and cessation versus percentage of cyclic. Tables 3.2 and 3.3 provide an overview of the initiation and cessation times for each muscle group as a percentage of the flexion-extension cycle. Three of the five active muscle groups (LP, SM, and BF) were initially involved at the beginning of trunk flexion. The LP myoelectric cessation occurred at 34 (± 7) % of the cycle during flexion and reinitiated at 57 (± 7) % of the cycle during extension. Myoelectric cessation of the SM and BF muscles occurred at 32 (± 7) % and 19 (± 17) % of flexion during the cycle, respectively, and both were reinitiated at 50 (± 5) % of the cycle during extension.

The EMG activity of the abdominal muscles, RA and EO, were detected in the flexion-extension cycle in about half of the participants. This activity coincided with the myoelectric silent period of the LP muscles. RA EMG activity (n = 5) was initiated at 42 (± 4) % and ceased at 54 (± 5) % of the cycle. EO EMG activity (n = 7) was initiated at 39 (± 5) % and ceased 57 (± 7) % of the cycle. EMG activity of both RA and EO muscles was observed in four individuals. One individual had only RA activity and three had only EO activity detected during this time period.

Table 3.1. Observed peak flexion and inclination angles (Mean ± SD) during both standing and supine conditions.

<table>
<thead>
<tr>
<th></th>
<th>Standing</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flexion (degree)</td>
<td>53 ± 12</td>
<td>44 ± 9*</td>
</tr>
<tr>
<td>Timing (% cycle)</td>
<td>48 ± 4</td>
<td>48 ± 3</td>
</tr>
<tr>
<td>Inclination (degree)</td>
<td>112 ± 15</td>
<td>13 ± 14*</td>
</tr>
<tr>
<td>Timing (% cycle)</td>
<td>48 ± 3</td>
<td>49 ± 5</td>
</tr>
</tbody>
</table>

* P < 0.002
Figure 3.1. Exemplar data of EMG and flexion/inclination angles during standing (A) and supine (B) trunk flexion-extension conditions. The vertical axis on the left is the trunk angle measurements in degrees. The vertical axis on the right is the EMG amplitude in arbitrary units. The horizontal axis is the percentage of each cycle. The thick line represents the trunk flexion angle and the thin line represents the trunk inclination angle. The inclination angle was measured with reference to the vertical (0 degrees). EMG data were collected from lumbar paraspinal (LP), rectus abdominis (RA), external oblique (EO), rectus femoris (RF), biceps femoris (BF), and semimembranosis (SM).
Figure 3.2. EMG activities during standing as a percentage of one trunk flexion-extension cycle. The bars represent the EMG activity per muscle group, while the whiskers denote 1 standard deviation from the respective initiation and cessation. The dashed vertical line represents the mean point during the cycle at which the maximum trunk flexion angle was attained. Average EMG activity durations of lumbar paraspinal (LP), rectus abdominus (RA, N = 5), external oblique (EO, N = 7), semimembranosis (SM, N = 11), and biceps femoris (BF, N = 9) muscles during standing.

Table 3.2. Posterior muscle activity cessation and initiation times during anterior trunk flexion-extension while standing. Each time is indicative of the percentage of the cycle.

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Time EMG off (Mean ± SD)</th>
<th>Time EMG on (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lumbar Paraspinal (LP, n = 13)</td>
<td>34 ± 7</td>
<td>57 ± 7</td>
</tr>
<tr>
<td>Semimembranosis (SM, n = 11)</td>
<td>32 ± 7</td>
<td>50 ± 5</td>
</tr>
<tr>
<td>Biceps Femoris (BF, n = 9)</td>
<td>19 ± 17</td>
<td>50 ± 5</td>
</tr>
</tbody>
</table>

There was no co-activation of back and abdominal muscles observed during the standing condition. The EMG activity of the LP muscles ceased before the initiation of either RA or EO EMG activity during flexion ($F_{1,92} = 40.44$, $P < 0.0001$; $F_{1,87} = 9.91$, $P < 0.003$, respectively). No
significant period of time was detected between the re-initiation of LP EMG activity and cessation of either RA or EO EMG activities during extension. The EO EMG activity was initiated before the RA ($F_{1,60} = 10.28, P < 0.003$) and ceased after the RA EMG activity ($F_{1,60} = 4.33, P < 0.05$).

Abdominal muscles exhibited no co-contraction with the lower extremity muscle activities during flexion, but did provide observable co-contraction during extension. RA EMG activity was initiated later than the cessation of SM and BF EMG activities during flexion ($F_{1,87} = 60.15, P < 0.0001; F_{1,73} = 50.26, P < 0.0001$, respectively), but ceased later than the re-initiation of both SM and BF EMG activities during extension ($F_{1,87} = 16.30, P < 0.0002; F_{1,74} = 16.85, P < 0.0002$, respectively). Similar to RA activity, the EO EMG activity was initiated later in flexion than the cessation of SM and BF EMG activities ($F_{1,82} = 19.98, P < 0.0002; F_{1,28} = 29.65, P < 0.0001$, respectively), but ceased after the re-initiation of SM and BF EMG activities during extension ($F_{1,82} = 32.24, P < 0.0001; F_{1,69} = 30.77, P < 0.0001$, respectively).

The coordination of the back muscle with each of the two lower extremity muscles was slightly different. The EMG activity cessation of the LP was later than that of BF during flexion ($F_{1,100} = 32.78, P < 0.0001$). There was no difference observed between LP and SM cessations. The LP EMG activity was reinitiated after the SM and BF during extension ($F_{1,114} = 36.24, P < 0.0001; F_{1,101} = 32.12, P < 0.0001$, respectively). RF activity was not observed during cycles in this position.

Table 3.3. Anterior muscle activity initiation and cessation times during anterior trunk flexion-extension while standing. Each time is indicative of the percentage of the cycle.

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Time EMG on (Mean ± SD)</th>
<th>Time EMG off (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus Abdominis (RA, n = 7)</td>
<td>42 ± 4</td>
<td>54 ± 5</td>
</tr>
<tr>
<td>External Oblique (EO, n = 5)</td>
<td>39 ± 5</td>
<td>57 ± 7</td>
</tr>
</tbody>
</table>
Supine Flexion-Extension

Some unexpected LP activities were observed in this condition. Figure 3.3 shows an example of flexion and inclination angles super imposed with LP EMG during the cycle. The EMG amplitude from the LP muscles was lower in supine than in the standing condition. A burst of activity was identified during trunk extension in 11 participants.

Figure 3.3. Exemplar lumbar paraspinal EMG pattern during supine trunk flexion-extension performance. The thin line along the x-axis is the profile of the rectified and smoothed EMG signal, which was detected at low amplitude, and denoted as a percentage of the peak EMG activity observed in the same muscle group during the standing condition. The thick line represents the relative trunk flexion angle. The dashed line represents the inclination angle of the trunk and the load applied to the posterior lumbar tissues due to gravity. Note that the greatest gravitational load in the paraspinal tissues would coincide with the peak inclination angle but the EMG magnitude was at its lowest level. A burst of EMG activity was observed during the early part of trunk extension. This may indicate that an inhibitory neural reflexive response was present when the passive tissues (i.e., ligaments) in the lumbar region reach near-maximum elongation.
Figure 3.4A and Table 3.4 describes the timing of EMG initiation and cessation of four muscle groups (LP, RA, EO, and RF) detected during the supine condition. Figure 4B illustrates the response of the LP muscles to gravitational loading (trunk flexion moment) during the supine flexion-extension when the trunk was flexed beyond vertical.

Table 3.4. Muscle cessation and initiation times during trunk flexion-extension from a supine position. The time of cessation and re-initiation is indicative of the sequence in the cycle. (Note: the back muscle activity in this condition was too low to be detected systematically, thus the EMG activity during extension is reported.)

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>EMG off (Mean ± SD)</th>
<th>EMG on (Mean ± SD)</th>
<th>EMG on (Mean ± SD)</th>
<th>EMG off (Mean ± SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectus Abdominis (RA)</td>
<td>31 ± 10</td>
<td>74 ± 6</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>External Oblique (EO)</td>
<td>54 ± 11</td>
<td>76 ± 8</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Rectus Femoris (RF)</td>
<td>30 ± 12</td>
<td>74 ± 7</td>
<td>---</td>
<td>---</td>
</tr>
<tr>
<td>Lumbar Paraspinal (LP, n = 11)</td>
<td>---</td>
<td>---</td>
<td>51 ± 12</td>
<td>67 ± 11</td>
</tr>
</tbody>
</table>

The LP EMG activity was silent during trunk flexion, even during deepest flexion. EMG activity of the LP muscles increased at 51 (± 12) % and ceased at 67 (± 11) % of the cycle during extension. The other three muscle groups served as agonists during flexion and were active from the beginning of the movement. The RA EMG activity ceased at 31 (± 10) % of the cycle before the deepest flexion angle was attained and re-initiated at 74 (± 6) % of the cycle. The EO EMG ceased at 54 (± 11) % of the cycle, after the deepest trunk flexion, and re-initiated at 76 (± 8) % of the cycle during extension.

We have examined the coordination among LP and the tested anterior muscles during this exercise. There was no significant difference between the cessation (30 ± 12 %) and re-initiation (74 ± 7 %) of RF and that of RA EMG activities. The LP EMG activity was initiated after the cessation of RA EMG activity ($F_{1,94} = 80.53$, $P <0.0001$). LP EMG activity ceased before the
Figure 3.4. A. EMG activities during supine condition as a percentage of one trunk flexion-extension cycle. The bars represent the EMG activity per muscle group, while the whiskers denote 1 standard deviation from the respective initiation and cessation. The dashed vertical line represents the mean point during the cycle at which the maximum trunk flexion angle was attained. Average EMG activity durations of lumbar paraspinal (LP, N=11), rectus abdominus (RA, N = 13), external oblique (EO, N = 13), and rectus femoris (RF, N = 13) muscles during supine trunk flexion-extension. B. Average EMG activity durations of the same muscles as in A dependent upon the inclination angle (x-axis) during the trunk flexion-extension movement. The dashed vertical line represents the mean inclination angle. Vertical lines highlight the inclination angles at 0 degrees, while the shaded bars represent 1 standard deviation from which 0 degrees of inclination was attained.
re-initiation of RA EMG activity ($F_{1,94} = 18.37, P < 0.0001$). There was no significant difference between the initiation of LP EMG activity and the cessation of EO EMG activity. LP EMG activity ceased a significant time period before the re-initiation of EO EMG activity ($F_{1,93} = 17.03, P < 0.0002$). EMG activity of the LP muscles was initiated after the cessation of RF EMG activity ($F_{1,91} = 70.59, P < 0.0001$) and ceased before the re-initiation of RF EMG activity ($F_{1,91} = 12.49, P < 0.0007$).

The coordination of the anterior muscles was different between flexion and extension phases of the cycle. The EMG activity of the EO muscles ceased after the cessation of both RA and RF EMG activities ($F_{1,128} = 155.91, P < 0.0001$; $F_{1,125} = 141.98, P < 0.0001$, respectively). There were no differences between RA, EO, and RF during the re-initiation of these muscle groups during trunk extension. BF and SM activities were not observed during the supine condition.

**Discussion**

Activation of the trunk musculature varied depending upon the change in trunk moment. Muscle activity of the abdominal muscles increased with standing deep trunk flexion in half of the subjects. An increased gravitational moment applied to the lumbar tissues did not initially result in an increased EMG activity in the LP muscles during the supine condition. The change in the gravity vector emphasizes the load sharing concept between muscles and passive tissues.

It is suggested that increased tension within viscoelastic tissues during standing trunk flexion elicits the flexion-relaxation phenomenon in LP muscles. The mechanical load sharing hypothesis related to this event (Allen, 1948; Floyd & Silver, 1955; Golding, 1952) usually discusses muscle activities of the posterior lumbar region. The possibility exists that abdominal muscles contribute to the mechanical mechanism during deep trunk flexion. On the other hand, the neural hypothesis (Floyd & Silver, 1951) proposes that the elongated viscoelastic tissues
trigger an inhibition responsible for the paraspinal muscle inactivity during deep flexion. The role of neural inhibition comes into question when LP muscle activity rises during extension in the supine condition.

Few studies of the flexion-relaxation phenomenon have identified abdominal activity during deep trunk flexion (Allen, 1948; Gupta, 2001). Kippers and Parker (1984) suggested the heightened activation of the abdominal muscles is necessary to overcome the posterior tissue resistance during deep trunk flexion. The abdominal muscles are required to assist gravity during flexion and serve as antagonists to the posterior tissues. Summation of the gravitational torque and abdominal muscle flexion torque would lead to an increased flexion moment acting at the lumbar region. However, an alternative explanation of abdominal muscle function also exists. The shortened abdominal muscles, specifically RA, are at a mechanical disadvantage when the trunk is fully flexed and may not provide sufficient force to counter the lumbar passive tissue forces. The activation of the RA and EO muscles may actually assist in elevating the intra-abdominal pressure (IAP). The IAP would then aid with the preservation of stability in the lumbar region when gravitational loading increases.

It is possible the increased IAP provides further mechanical support and directly enhances the extensor moment to the lumbar vertebrae during trunk flexion. However, the mechanisms responsible for increasing IAP still remain controversial (Cholewicki, Juluru, & McGill, 1999; Hemborg, Moritz, & Löwing, 1985; McGill & Sharratt, 1990). It is assumed IAP increases with a corresponding rise in abdominal muscle activity, attributed to the resulting increased stiffness of the muscles (Cresswell, Grundstrom, & Thorstensson, 1992; Grillner et al., 1978). Co-activation of trunk agonist and antagonist muscles is recognized to increase IAP (Gardner-Morse & Stokes, 1998; Hemborg, Moritz, & Löwing, 1985). Elevated activity of the RA and oblique muscles may not determine the extent of changes in IAP (McGill & Norman,
1985). Other muscles, such as transverse abdominis (De Troyer, Estenne, Ninane, van Gansbeke, & Gorini, 1990), internal oblique (Cresswell & Thorstensson, 1989), and the diaphragm (Hodges, Gadevia, & Richardson, 1997) may also contribute significantly to IAP.

It must be understood that the oblique muscles provide a larger area of support to the abdominal cavity. Their geometric configuration may also allow for considerable force production compared to the RA (Nussbaum, Chaffin, & Rechtien, 1995). The involvement of the abdominal muscles leads to increased IAP support for the abdominal cavity resulting in a complementary reinforcement of the integrity of the lumbar vertebrae (Cresswell and Thorstensson, 1989; Hodges, Cresswell, Daggfeldt, & Thorstensson, 2001). This reinforcement may provide for balance between the extension moment contribution of the posterior tissues and the flexion moment imparted by gravity during trunk flexion.

The EO muscles may have served multiple roles in the supine condition. It is possible the EO muscles transitioned from initial trunk flexion to support the vertebral joints, and indirectly imparted extensor moment via IAP at peak trunk inclination.

McGill (1995) and McGill and Sharratt (1990) indicate the relationship between abdominal activity (RA, external, and internal oblique) and IAP is moderate. In multiple tasks including sit-up exercises and standing trunk flexions IAP varied according to the task and the individual. It would be beneficial to further explore the contributions of other abdominal muscles, not evaluated in this study, to IAP.

Gravitational loading orientation significantly influenced the behavior of the LP muscles, independent of the kinematics. As Figure 3.5 illustrates, flexion and extension were performed in both standing and supine conditions but the influence of gravity was distinct. In standing, a silent period was denoted between two episodes of elevated LP EMG activity when trunk flexion moment was the greatest. Silence in the LP muscles was maintained through gradual
gravitational loading of the posterior tissues in the supine condition. Extension of the trunk brought about an increased activation of the LP muscles. Flexion of the lumbar spine prior to application of a trunk moment pre-loaded the viscoelastic tissues allowing the LP muscles to remain silent. The mechanical properties of the viscoelastic tissues changed during extension permitting the recruitment of the LP muscles. The effect of gravity direction supports the mechanical loading hypothesis since the LP EMG pattern is affected by different loading conditions. If the neural reflexes were related to the tension and length of the posterior passive tissues then the neural mechanism hypothesis would be true.

Figure 3.5. Comparison of lumbar paraspinal (LP) activities between standing (LP ST) and supine (LP SU) conditions. Dashed vertical line indicates the peak inclination angles. Solid bars and horizontal lines indicate the elevated EMG activity level of the LP muscles and 1 standard deviation from the mean cessation and initiation of activity, respectively, as a percentage of the flexion-extension cycle. Simulated inclination (dotted line, top) angles were used to illustrate movement in both conditions. Thin line (middle) indicates gravitational loading of LP tissues in the standing condition. Thick line (bottom) represents gravitational loading of the LP tissues in the supine condition. Even though the kinematics was the same between conditions, the behavior of the LP muscles was different depending on the influence of gravity.
Sensory receptors, such as mechanoreceptors and vestibular sensors, respond to changes in the position of the trunk (Ross & Thewissen, 1987; Suzuki, Timerick, & Wilson, 1985). Mechanoreceptors may influence the behavior of the LP muscles during trunk flexion by transmitting afferent signals to the central nervous system (Solomonow et al., 1999). Previous investigations observed mechanoreceptor afferents located within the passive tissues responded to changing mechanical properties of the tissues (Gray & Mathews, 1951; Grigg & Hoffman, 1982; Grigg, Schaible, & Schmidt, 1986; Krogsgaard, Dyhre-Poulsen, & Fischer-Rasmussen, 2002; Lundberg, Malmgren, & Schomburg, 1978). Deformation and tension within the passive tissues are dependent upon the magnitude of the load on the system. Thus gravity direction is a crucial factor in determining the response of the sensory receptors to the mechanical changes within the tissues. Our previous investigation of the flexion-relaxation phenomenon suggested the possible influence of both mechanical and neural mechanisms (Olson et al., 2004). Based on the results provided in feline models we suggested that a neural adaptation influenced the early cessation and delayed initiation of LP myoelectric activity (Claude et al., 2003; Olson et al., 2004; Williams et al., 2000).

A secondary observation detected from the EMG data during the standing condition involved a myoelectric silent period of the hamstring muscles. Previous research looking into the activity of the LP muscles reported conflicting details regarding SM and BF activities (Gupta, 2001; McGorry, Hsiang, Fathallah, & Clancey, 2001; Portnoy & Morin, 1956). Fischer and Houtz (1968) and Sihvonen (1997) documented the myoelectric silent period of the hamstring muscles during studies of the trunk and hip extensors. It is essential to realize that the movement of the trunk is mechanically associated with that of the pelvis. The lumbo-pelvic rhythm is critical when explaining the interaction between the trunk and pelvic movements (Esola, McClure, Fitzgerald, & Siegler, 1996; McClure, Esola, Schreier, & Siegler, 1997; Paquet,
Malouin, & Richards, 1994; Porter & Wilkinson, 1997). The rotation of the pelvis about the hip joint allows for further flexion of the trunk segment after full lumbar flexion is achieved. The antagonist hamstring muscles impart an extensor moment at the hip to offset the hip flexion moment supplied by gravity.

The myoelectric silent period observed in the hamstring muscles during the standing condition may indicate the activation of the Golgi tendon organ. Activation of these receptors may be a consequence of increased tension due to sagittal rotation of the pelvis. Early EMG activation of the SM and BF muscles during trunk extension provides evidence to suggest the pelvis is displaced before the trunk extends. Initiation of the hamstring muscles activity may assist the trunk extensors during extension.

Despite the epidemic of low back pain reported among workers (U.S. Department of Labor, 2003 and 2004), the treatment and intervention for subacute low back pain is still in its exploratory stage (Karjalainen, Malmivaara, Mutanen, Roine, Hurri, & Pohjolainen, 2004). Increasing our understanding of the coordination among the active and passive tissues that contribute to low back motion can help us to improve the treatment of such disorder. Our observations in the current study point out the severe limitations of the literature regarding 1) the role of the mechanical structures in contributing to the low back motion, and 2) questions regarding the hypothesis of how over-stretched low back viscoelastic tissues may inhibit low back muscle activity. Further investigation along this line will help us to fill the gaps among all the clinical treatments for subacute low back pain (Pengel, Maher, & Refshauge, 2002).

In summary, gravitational loading orientation relative to the trunk affects the activation pattern of the anterior and posterior muscles. The flexion moment due to gravity directly influences the loading of the posterior lumbar tissues and affects how the system responds independent of the kinematics. Abdominal muscle activation during standing trunk flexion-
extension may provide an additional mechanical explanation related to the flexion-relaxation phenomenon. Increased abdominal muscle activity may increase intra-abdominal pressure to support the lumbar vertebrae when the lumbar paraspinal muscles are electrically inactive. Support for a compensatory mechanical process was presented that would assist in studying the FRP. The influence of mechanoreceptors and other sensory receptors on the behavior of muscles when gravitational loading is modulated deserves further investigation. Studying the tension changes within the tendon complex may assist in further developing an understanding between mechanical and neural mechanisms. Future research should focus on methods to measure and isolate mechanical and neural mechanisms to further understand the factors affecting muscle behavior.

References


CHAPTER 4: INCREASED PASSIVE TISSUE COMPLIANCE IN THE LOW BACK DURING PASSIVE CYCLIC TRUNK FLEXION-EXTENSION EXERCISE

Introduction

Low back pain and disorder is a prevalent condition in industrialized countries. Surprisingly, with the advancement of modern technologies used in the workplace there are a greater number of people currently afflicted with this condition (Marras, 2000; Marras, 2005). The U. S. Department of Labor (2004) reports that injuries to the low back are the leading cause of work days missed. The treatment of these injuries costs individuals and corporations over $10 billion per year, and this figure steadily increases each year (NIOSH, 1999). This brings to question the etiology of low back pain and disorder in individuals who work in labor intensive settings. Those individuals who are required to perform repetitive movements may be at a higher risk for low back pain and disorder.

Previous research has indicated the types of trunk movement that significantly alter the mechanical forces acting on the vertebrae and consequently modify the lumbar muscular activities (Dolan & Adams, 1993; Dolan, Earley, & Adams, 1994; Kankaanpää, Taimela, Laaksonen, Hänninen, & Airaksinen, 1998; van Dieën & Oude Vrielink, 1998). Granata and Marras (1999) indicate that multiple factors, such as dynamic compression and shear loading, muscle fatigue, and specific requirements of the work place affect low back pain. Many labor intensive tasks require either trunk flexion-extension, trunk rotation, or a combination of both (Marras, Davis, & Granata, 1998; Schultz, Andersson, Ortengrn, Haderspeck, & Nechemson, 1982). The combination of flexion and rotation during lifting activities requires co-activation of abdominal muscles (internal and external obliques, rectus abdominis) and paraspinal muscles which increase forces acting on the lumbar spine. Flexion-extension movements have been studied extensively in the literature as it is the primary motion performed during lifting tasks (Dolan & Adams, 1998; Dolan, Mannion, & Adams, 1994; Esola, McClure, Fitzgerald, &
Siegler, 1996; Gracovetsky, Kary, Levy, Said, Pitchen, & Helie, 1990; Ng, Kippers, Richardson, & Parianpour, 2001; Oddsson & Thorstensson, 1985; Schultz, Haderspeck-Grib, Sinkora, & Warwick, 1985). In laboratory settings paraspinal myoelectric activities were modified as continuous trunk flexion-extensions were performed (Caldwell, McNair, & Williams, 2003; Dickey, McNorton, & Potvin, 2003; Marras & Granata, 1997; Olson, Li, & Solomonow, 2004).

Mechanical property changes within the muscles and passive tissues directly affect the response of the system to external stimuli. Altered muscle activation due to muscular fatigue has been hypothesized to influence the incidence of low back injury and disorder (Bonato, Ebenbichler, Roy, Lehr, Posch, Kollmitzer, et al., 2003; Dolan & Adams, 1998). Muscular fatigue is influenced by the frequency of neural stimulation and the muscle fiber type (Elder, Bradbury, & Roberts, 1982; Mannion, Dumas, Stevenson, & Cooper, 1997; Moritani, Nagata, & Muro, 1982; Petrofsky & Lind, 1980). The effects of fatigue are temporary as the muscles recover from the requirements of the task over a relatively short period (Brereton & McGill, 1998; Kuorinka, 1988). The recovery process is dependent upon the muscle length (Herzog & ter Keurs, 1988), the rate of muscle contraction (Ettema, Huijing, & de Haan, 1992), and the type of muscle contraction that produce the fatigue (Hermann & Barnes, 2000; Mannion & Dolan, 1994; Potvin & Norman, 1993). Frequent breaks and extended rest periods are common strategies used to reduce the effects of fatigue in the workplace. This strategy provides little evidence to support the direct influence of fatigue on low back injuries.

The responsibility of supporting the vertebral joints increases in the passive tissues when the lumbar muscles become fatigued or inactive. As a consequence, the ligaments, discs, and fascia then become exposed to greater load and a higher probability of injury. Modification of the paraspinal muscle activity occurs as a result of muscular fatigue, but may also be due to mechanical changes within the passive tissues as suggested by Olson et al. (2004).
The absence of myoelectric activity towards the terminal end of trunk flexion has been observed and related to passive tissue loading and deformation (Allen, 1948; Floyd & Silver, 1951; Floyd & Silver, 1955; Portnoy & Morin, 1956). Hypotheses regarding this myoelectric ‘silent period’ include either load sharing between the active muscles and passive ligaments (Andersson, Oddsson, Grundström, Nilsson, & Thorstensson 1996; Floyd & Silver, 1951; Hoek van Dijke, Snijders, Stoeckart, & Stam, 1999; Touissaint, de Winter, de Hass, de Looze, & van Dieën, 1995) or neural inhibition via mechanoreceptors related to soft tissue elongation (Floyd & Silver, 1955; Olson et al., 2004). These hypothesized mechanisms may also function concomitantly. There is evidence to show that repetitive gravitational loading of the trunk during flexion modulates muscular behavior over time (Dickey et al., 2003; Olson et al., 2004).

Conflicting evidence regarding the effects of continuous cyclic trunk flexion-extension on the behavior of the lumbar paraspinal muscles is reported in the literature. Dickey and colleagues (2003) observed a decreased ‘silent period’ after individuals performed 100 cycles of flexion-extension with external loads held in the hands. The modification of the muscle behavior could not be determined from the separate entities of repetitive movement or external loading. When repetitive cyclic flexion-extension was performed for a 10 minute period without application of an external load an increased ‘silent period’ was reported (Olson et al., 2004). Olson and coworkers suggested that the muscle activation changes were due to mechanical changes in the posterior lumbar tissues that directly affected sensory receptor desensitization. It is difficult to ascertain whether this is a plausible conclusion since passive tissue properties could not be directly measured. Fatigue could not be factored out of either study as a possible influence to the modified muscle behavior since the muscles were actively recruited. The direct consequences of repetitive flexion loading on the passive tissues have not been determined in humans, although indirect evidence is present in cadaver and animal models.
The use of cadaver models provides indirect evidence that passive tissues were compromised during loading (Dumas, Beaudoin, & Drouin, 1987; Goel, Voo, Weinstein, Liu, Okuma, & Njus, 1988; Pintar, Yoganandan, Myers, Elhigediab, & Sances, 1992; Hukins, Kirby, Sikoryn, Aspen, & Cox 1990). Animal models provide information indicating that mechanoreceptor afferents and nociceptors located in the ligaments respond to length and tension changes (Claude, Solomonow, Zhou, Baratta, & Zhu, 2003; Solomonow, Zhou, Baratta, Lu, & Harris, 1999). Manipulation of cadaver and animal ligaments through cyclic loading in creep and tension-relaxation protocols result in decreased tension within the biomaterial. Isolation of the passive tissues in vitro and in vivo assures that mechanical measurements are independent of muscle activity. There is no direct evidence in human models to indicate laxity within the posterior lumbar tissues is established after repetitive loading. Evidence of this nature would support current theories of a repetitive loading mechanism responsible for low back pain and disorder.

The purpose of this study is to examine the mechanical behavior of the lumbar passive tissues during repetitive passive cyclic trunk flexion-extension. It is hypothesized that the forces within the lumbar passive tissues will decrease as a result of repetitive cyclic sagittal trunk movement. As a secondary examination, it is also hypothesized that a neuromuscular adaptation will occur in the surrounding lumbar muscles as a result of the mechanical behavior modification in the passive tissues.

Methods

Subjects

Eighteen male college students volunteered to participate in this experiment. University approval and consent from each individual were obtained before data collection. The mean (SD) height, mass, and age of the subjects were 1.79 (0.08) m (range 1.65 – 1.96m), 78.4 (11.2) kg
(range 63.6 – 99.1 kg), and 21.6 (1.9) years (range 19 – 27 yrs), respectively. None of the participants reported any previous back or lower extremity pain or disorders.

Instrumentation

Surface electromyography (EMG) recordings were collected using an MA300 system (Motion Lab Systems, Baton Rouge, LA). EMG signals were collected from the right side of the individual using pre-gelled Ag-AgCl bipolar electrode pairs. The 1.0 cm in diameter electrodes were spaced 2.5 cm apart along the length of the underlying muscle fiber. The two placement sites were the paraspinal muscles at the L2 and L4 levels, 3.0 cm lateral to the spinous processes. The EMG signals were amplified up to 18,000 with the frequency band pass width set at 10 – 480 Hz. The common mode rejection ratio (CMRR) was set at 100 dB. Data were collected at 1000 Hz using a 12 bit analog to digital board and stored for future processing.

A dynamometer (830-110, Biodex Medical Systems, Shirley, New York) was used to control the passive flexion-extension movement of each person. The System3 software (Biodex Medical Systems) collected applied torque, angular displacement, and angular velocity data. The axis of the dynamometer was aligned with the L4-L5 joint of the participant (McGill & Kippers, 1994). An attachment to the dynamometer was constructed to support the trunk during movement (see Figure 4.1). The individual was harnessed to the horizontal arm of the attachment across the shoulders just inferior to the clavicles.

Protocol

Once EMG signal checks were performed and satisfactory, the individual was positioned with his back against a secured support. A leather belt was tightly harnessed around the participant’s waist at the anterior superior iliac spines (ASIS) to minimized pelvic movement during trunk flexion. A nylon strap was also fixed around the individual’s thighs to minimize the
influence of knee flexion (McGorry, Hsing, Fathallah, & Clancy 2001; Shin, Shu, Li, & Mirka, 2004, Figure 4.1).

Figure 4.1. Example of the procedures used to secure subjects during the passive trunk flexion-extension session. The pelvis and thighs were secured during the entire testing session. The trunk was harnessed to the arm of the Biodex dynamometer during the passive session only.

The experimenter then demonstrated the flexion-extension movement required of the participant and allowed for practice of this motion while free of the dynamometer. Each participant performed trunk flexion to his peak flexion angle within 5 seconds, and then performed trunk extension to the initial erect stance for another 5 second period. A metronome set at 60 beats·min⁻¹ was used as a guide to assist the individual in maintaining a constant tempo during flexion and extension. Once the participant was comfortable with this tempo, three separate cycles of active trunk flexion-extension were performed and recorded. The participant was instructed to look at a spot on the floor 20 cm in front of his feet to control head movement.
The arms were controlled as the forearms were crossed over the chest. To control for the effect of different types of footwear participants performed the testing barefoot. This protocol was performed prior to and immediately following the passive flexion-extension procedure (see Figure 4.2).

The passive session of the experiment began as the individual was secured to the dynamometer. The range of motion for each individual was recorded after the individual had been secured. From this information, the angular velocity of the movement (degrees·sec⁻¹) was calculated to assure that 5 seconds of flexion and extension were performed in the cycle. Six cycles per minute were performed over a 10 minute period. Measurement of the applied torque to the dynamometer allowed for indirect observations of the changes in the mechanical behavior of the lumbar passive tissues. Calibration of the dynamometer was performed prior to and after data collection to check accuracy.

Each participant was instructed to remain relaxed during the passive session. He was also advised to rest his body against the attachment arm of the dynamometer. A computer monitor which displayed the real-time EMG signals from all muscle sites recorded was placed on the floor 30 cm in front of the individual at a 45° angle to ensure muscle silence with biofeedback. After the range of motion was determined, participants were actively guided through the flexion-extension movement by the experimenter. They were told to observe the EMG signals on the monitor and maintain small signal amplitude in the lumbar EMG displays.

To minimize back muscle activity due to head motion, the participant was instructed to keep his chin close to the attachment arm. If view of the monitor was obscured, then he was instructed to maintain the position of the chin.
Analysis

In addition to the EMG data collected during the three cycles before and after the passive session, EMG data was collected the first 30 sec of each minute at a rate of 1000 Hz during the passive movement session. Torque, angular displacement and angular velocity data were simultaneously collected with the EMG data.

Electrode application and testing

Pelvis and thigh fixation

Tempo familiarization

Three active flexion-extension cycle

Ten minutes passive flexion-extension

Three active flexion-extension cycle

Figure 4.2. The stepwise procedures followed in testing during active and passive trunk flexion-extension sessions.

Raw EMG signals from the passive session and pre/post active sessions were full-wave rectified. A linear envelope was constructed with a forth-order Butterworth low pass filter with a
4 Hz cut off frequency. The filtered EMG was separated in to flexion and extension phases and integrated for future statistical analysis.

Torque data were filtered with a forth-order low pass Butterworth filter at 1 Hz cut off frequency. Three dependent variables were of interest during the analysis of the torque-angle curve. These variables were the peak torque attained during the flexion phase (PTF), the torque applied at the deepest trunk flexion angle (TT), and the peak torque attained during the extension phase (PTE).

Upper body (head, arms, trunk) center of mass and inertial properties were calculated based on Dempster (1955) (see Winter, 1979). Torque due to the passive tissues of the back was estimated by using equation (4.1). The estimated passive tissue torques (ligament, fascia, and passive muscle) enable us to determine the change in the mechanical properties of the tissues over the 60 cycle passive session. This process was performed using the constant velocity portion of the flexion-extension motion.

\[ T_{pt} = T_m - T_g \]  

Where \( T_{pt} \) represents torque of the passive tissues, \( T_m \) is the measured torque, and \( T_g \) is the gravity contribution. Torque (\( T_{pt} \))-angle relationship, based upon hysteresis effects, was estimated during the separate flexion (ascending) and extension (descending) phases. The differences in the area under the ascending and descending curves were used to measure the energy dissipation of the passive tissues during each cycle (Heerkens, Woittiez, Kiela, Huijing, Huson, van Ingen Schenau, et al., 1987).

Frequency analysis (Fast Fourier transformation) was performed on the raw EMG signal from the LP muscles of the active sessions to identify possible muscular fatigue. The period of myoelectric silence was excluded from the frequency analysis and served to separate flexion and extension phases.
Integrated EMG (IEMG) (summation of EMG values x collection rate frequency\(^{-1}\)), based on the rectified data, was calculated for muscle activity during the active flexion and extension sessions. Increased IEMG without frequency reduction (shift of the power density spectrum) after the passive session would indicate a compensation for the compromised passive tissues. IEMG activities of the LP muscles during flexion and extension phases from the passive session were also calculated and compared over cycles to measure changes that may have occurred.

One-factor ANOVA with repeated measures (Statistix Corp.) was used to analyze selected torque and trunk angle parameters in the passive session (overt time), and EMG data from active trials (pre/post active sessions). IEMG during the passive session was analyzed with a two-way ANOVA (time x flexion-extension). The alpha level was set at < 0.05.

Results

To test the mechanical property changes of the passive lumbar tissues we employed passive cyclic passive motion in our testing protocol. Surface EMG data were collected before and after the passive motion at L2 and L4 levels and frequency contents of the EMG data were analyzed. Frequency data during flexion and extension were pooled together since there was no difference between them (Figure 4.3). EMG frequencies would shift to the lower end of the power density spectrum due to fatigue after the passive session if the LP muscles were actively recruited. However, mean frequency did not change from pre (45 ± 3) to post (48 ± 3) passive sessions (\(F_{1,71} = 2.90, \ p < 0.11\)), while median frequency increased from pre (114 ± 3) to post (118 ± 5) passive sessions (\(F_{1,71} = 5.13, \ p < 0.04\)). There were no signs of fatigue in the LP muscles, which supports the contention that LP muscles were not active during the passive session.
Figure 4.3. Mean and median frequency densities for LP muscles before (pre: blue) and after (post: red) the passive session. Flexion and extension phases have been pooled since no observable difference between them. Vertical lines denote standard error of the mean (SEM). No significant differences were observed between pre and post mean frequencies. Post median frequency was significantly greater than pre median frequency (*p<0.05).

The IEMG activity during the passive exercise also supports this hypothesis. There was no difference observed among the IEMG collected at different sites in the flexion or extension phase of the motion. The IEMG during flexion and extension phases at both L2 and L4 sites were pooled together. The pooled IEMG data did not change during the passive session (F_{9,179} = 1.70, p > 0.10, Figure 4.4). This observation also indicates changes in torque applied to the dynamometer were not influenced by the activity of the paraspinal muscles.

Figure 4.5 provides exemplar torque data measured during the passive conditions. A significant hysteresis loop is apparent in every trial where the measured torque values at the given trunk angle were different, depending upon if it was achieved during flexion or extension phase. Peak torque applied to the dynamometer during the flexion phase occurred between erect stance (0°) and full trunk flexion (~90°, Figure 4.5). Peak flexion torque (PTF) increased significantly from 45 (± 4) to 60 (± 4) Nm (F_{9,178} = 6.24, p < 0.0001, Figure 4.6), indicating the
posterior lumbar structures were compromised. The PTF increased linearly ($F_{1,152} = 75.91, \ p < 0.0001$). The increasing PTF was accompanied by a corresponding increase in the angle at which the PTF was attained from 29 (± 4) to 37 (± 5) degrees ($F_{9,178} = 2.72, \ p < 0.006$). This observation is consistent with the behavior of biomaterials when creep or tension-relaxation is present. The peak flexion angle also increased linearly ($F_{1,152} = 19.50, \ p < 0.0001$).

![Figure 4.4](image)

**Figure 4.4.** Illustration of the pooled IEMG from L2 and L4 paraspinal levels while combining flexion and extension phases over 10 minutes. Paraspinal muscle activity remained relatively low throughout the entire passive session. Vertical bars represent the mean IEMGs of each cycle, while the vertical lines represent the standard error of the mean (SEM).

The magnitude of TT increased from 7 (± 4) to 21 (± 4) Nm with continuous cyclic movement of the trunk ($F_{9,178} = 5.17, \ p < 0.0001$, Figure 6). The TT increased with a linear trend ($F_{1,152} = 41.16, \ p < 0.0001$). These data indicate that the tension within the lumbar tissues at the peak flexion decreased with continuous cyclic trunk movements performed over extended periods of time. The PTE increased from 79 (± 6) to 81 (± 4) Nm in a linear trend ($F_{1,152} = 6.24,$
p < 0.014) (Figure 4.6). There was no significant change observed in the extension angle at which the PTE was attained.

Figure 4.5. Exemplar data of angle-torque trajectories measured from the dynamometer for the 10 cycles analyzed for one participant. The first, third, fifth, seventh, and ninth cycles are illustrated, respectively. The most significant torque increase occurred in the peak flexion torque (PTF) and torque trough (TT), indicated by upward solid arrows. There was no significant change in the peak extension torque (PTE). Dotted arrows indicate the direction of the trunk movement.

Figure 4.6 illustrates the change in peak torque (PTE PTF, and TT,) over the 10 minutes of passive trunk flexion-extension. A correlation between the changes in PTF and TT as well as PTF and PTE were observed ($r^2 = 0.63$, $p < 0.001$; $r^2 = 0.22$, $p < 0.001$, respectively). There was no correlation between PTE and TT observed ($r^2 = 0.06$, $p > 0.42$). A significant correlation between these variables indicates their inter-relationship with passive tissue compliance as continuous passive movement is performed.
Excluding the influence of gravity and the support of the dynamometer, the behavior changes of the posterior passive tissues over time are plotted in Figure 4.7 as the modeled hysteresis curve of each participant (Figure 4.7). The area within the hysteresis curve of each cycle is related to energy dissipation. Figure 4.8 demonstrates that the energy dissipation decreased along a significant linear trend with the 10 minute exercise ($F_{1,152} = 9.57$, $p < 0.003$).

IEMG activities from flexion and extension phases were compared between pre and post passive sessions. Support for increased compliance of lumbar tissues was provided from this analysis. LP muscle activity increased from 0.031 (± 0.002) to 0.036 (± 0.002) V*s when trunk flexion was performed after the passive session ($F_{1,35} = 7.34$, $p < 0.02$, Figure 4.9). These observations suggest a muscular compensation for the increased compliance of the passive lumbar tissues during flexion of the trunk. Although LP IEMG increased during extension after the passive session the change was not statistically significant (Figure 4.9).

Figure 4.6. Graphic representation of mean (SEM) peak extension torque (PTE), peak flexion torque (PTF), and torque trough (TT) measured from the dynamometer over time during the passive session. There was a significant increase in the load applied to the dynamometer for both PTF and TT with increased trunk flexion-extension cycle ($F_{9,178} = 8.66$, $p < 0.0001$; and $F_{9,178} = 5.17$, $p < 0.0001$, respectively). There was no significant change observed in the PTE over time.
Figure 4.7. Exemplar modeled data from one individual illustrating the change in posterior passive tissue torque over time. The bold blue line indicates the initial (1st) cycle. The solid lines indicate constant velocity period while the dashed lines indicate acceleration period of the trunk motion. Representative cycles from minutes three, five, seven, and nine are superimposed upon the initial cycle. The flexion phase is (denoted by upward arrow) and the extension phase is (denoted by downward arrow).

Figure 4.8. Graphic representation of the change in area (energy dissipation) of the hysteresis loop of the modeled data over time. The squares represent the mean of each cycle, while the SEM is represented with vertical lines. There was a significant linear trend (p < 0.03) present in the data indicating a change in energy dissipation was apparent over time.
Figure 4.9. IEMG from flexion and extension phases pooled over subjects during active pre and post passive sessions. The vertical bars represent the mean IEMG values, while the vertical lines represent the SEM. The asterisk denotes a significant difference between pre and post passive session IEMG values for the flexion phase ($F_{1,35} = 7.34, p < 0.02$). There was no significant difference between pre and post IEMG values for the respective extension phases. The graphic indicates that there was a possible compensation by the active paraspinal muscles when the passive posterior lumbar tissues were compromised.

Discussion

The purpose of this study was to identify biomechanical changes in the lumbar paraspinal tissues during continuous passive cyclic trunk flexion-extension. The forces within the lumbar passive tissues were hypothesized to decrease as a result of repetitive cyclic sagittal trunk movement. It was also hypothesized that a neuromuscular adaptation would occur in the surrounding lumbar muscles as a result of the mechanical behavior modification in the passive tissues. The most significant observations included a decreased torque supplied by the passive tissues, and an increased EMG magnitude after the 10 minutes of passive trunk flexion-extension.
Torque Changes as a Function of Trunk Angle

While moving at constant angular velocity during passive low back stretch, there are three torques applied to the trunk at the lower back: extension torque produced by the passive tissue (T$_{pt}$), flexion torque due to gravitational load (T$_{g}$), and the extension torque the Biodex applied to the trunk (T$_{m}$). The magnitude of the third torque is a function of the first two during equilibrium (Equation 1). Flexion of the trunk requires an elongation of the lumbar tissues resulting in a greater magnitude of tension within the passive structures. The elevated tension allows the passive tissues to support the load of the trunk through greater torque production (T$_{pt}$) at the joint when deeper trunk flexion angles are attained (Floyd and Silver, 1955; Kippers and Parker, 1984). Anterior trunk flexion increases the T$_{g}$ applied to the trunk at the L4-L5 joint as the system center of mass deviates from the line of gravity. T$_{m}$ changes according to the position of the trunk in flexion while both T$_{pt}$ and T$_{g}$ change with trunk angle. The increased T$_{m}$ from 0 degree to half way through trunk flexion is due to the dominating increase of T$_{g}$ (flexion), but from there to the deepest flexion T$_{m}$ decreases due to the rise of the T$_{pt}$ (Figure 5).

Adaptation to Exercise

Evidence for compliance, or laxity, of the posterior lumbar tissues was indirectly provided over the 60 cycles/10 minutes of cyclic trunk flexion-extension. Increased T$_{m}$ at a given trunk angle indicates a decreased T$_{pt}$, since T$_{g}$ is solely dictated by the trunk angle, while decreased T$_{m}$ at a given trunk angle indicates an increased T$_{pt}$ for the same reason. The observed T$_{m}$ increase in this study suggests that that the resistance of the passive tissues, and thus the ability to support the trunk load, decreases over time through the continuous cycles. This result is consistent with the existing literature. For example, Hubbard and Chan (1988) observed decreased passive strength while cyclically loading canine tendons. There is a difference in the rate of strength reduction. The results here showed a reduction linearly but Hubbard and Chan
reported most of the reduction occurred at the initial phase of the process. This difference maybe
due the familiarization and active flexion our participants performed before the passive stretch.
Studies show that the most significant changes in the hysteresis loop occur within the first 10
loading cycles (Magnusson, Agard, Simonsen, & Bojsen-Møller 1998; Taylor, Dalton, Seaber, &

The decreased $T_{pt}$ over time may imply a mechanical change within the posterior tissues
that also affects the position at which PTF is attained in the cycle (Lam, Frank, & Shrive, 1993;
Solomonow et al., 1999). Greater compliance of the passive tissues would necessitate an
increased role of the active components of the lumbar muscles to maintain support and stability
of the system during an active flexion motion.

Neural and Muscular Adaptations

Loading of the viscoelastic tissues reduces the force capacity of the structures in animal
and human models. The forces within the posterior passive tissues were reduced as a function of
time in the current study. In feline models the mechanical properties of the supraspinous
ligament are compromised through continuous static and cyclic elongation (Claude et al., 2003;
Solomonow et al., 1999). The modifications in the ligaments affect the reflexive response of the
surrounding musculature. In these studies, a reduced reflexive response to mechanical
manipulation is a result of time dependent changes in the tissues. Hours after the specimen has
been loaded a modified reflexive response is still present (LaBray, Sbriccoli, Zhou, &
Solomonow, 2004; Williams, Solomonow, Zhou, Baratta, & Harris, 2000). Experiments of this
type provide evidence suggesting passive tissue laxity contributes to neuromuscular disorders in humans.

Numerous human studies have indicated a link between mechanical stimulation of the
tissues and modified neural responses. EMG results from the current study indicate there was no
change in the neuromuscular response of the paraspinal muscles during the passive session. Activation patterns of the lumbar muscles were altered after the 10 minute session leading us to speculate that the mechanical changes within the passive tissues led to a neural adaptation. Biedert and Zwick (1998) reported a reflexive response from hamstring muscles when the surgically reconstructed anterior cruciate ligament (ACL) is stretched. However, isolation of tissues (muscles and ligaments) in humans is compounded by the interaction of the surrounding tissues coupled with embedded mechanoreceptors which respond to mechanical stimulation. Tissue isolation, in this example, does not take into consideration the interaction between other passive and active tissues within the system. Chu, LeBlanc, D’Ambrosia, D’Ambrosia, Baratta, and Solomonow (2003) induced non-invasive ACL creep in both men and women and reported a significant increase in the activation of the antagonist hamstring and quadriceps muscles during knee flexion and extension exercises after creep. Sbriccoli, Solomonow, Zhou, Lu, and Sellards (2005) performed cyclic anterior translations of the ACL in men and women over a 10 minute period and reported a decrease in maximal voluntary contraction after the protocol. These studies provide evidence to indicate an acute neuromuscular disorder may increase the likelihood of injury to a joint. It is assumed that research of this nature does compromise the passive tissues within the joint capsule leading to further instability of the joint. The results of this current study support the reports of the previously mentioned literature since $T_{pt}$ in the low back decreased as a function of time. As such, the acute loading performed in this study may provide a prelude to the adaptation of the neuromuscular system and further the development of LBP and disorder if persistent.

No Reflexive Response Observed Due to Low Velocity

Reflexive responses were not observed in the lumbar paraspinal muscles during the passive session. Lu, Solomonow, Zhou, Baratta, and Li (2004) reported differences in reflexive
discharge of the paraspinal muscles in feline models during recovery periods when differing velocities of the flexed spinal ligaments were applied. There were no differences observed in the EMG discharge signal between feline specimens perturbed at 0.1 Hz versus 0.5 Hz during passive sessions. Threshold levels of afferent receptors that initiate muscle activation through stretch are dependent upon the velocities at which segments are displaced (Edin & Vallbo, 1988; Solomonow, Eversull, Zhou, Baratta, & Zhu., 2001). The forces developed in the passive tissues will be greater when higher velocities of movement are applied.

Morphological Basis for Mechanical Property Changes

The measured $T_m$ increases and decreases as a function of the mechanical properties inherent to the biological composition of the posterior passive tissues. Recruitment of the collagen fibers is dependent upon the alignment of the fibers with the line of tensile force applied to the tissues (Woo, Hollis, Adams, Lyon, & Takai, 1991). Initially, the tissues are unloaded and relaxed allowing the collagen fibers to remain in a crimped configuration. Loading of the tissues leads to unraveling of the fibers from the crimp arrangement. Increased recruitment of the collagen fibers influences the non-linear viscoelastic response of the tissues to loading (Stomberg & Wiederhielm, 1969; Viidik, 1972). Greater elongation of the tissues requires further participation of more fibers to support and sustain the integrity of the biomaterial as external forces are applied (Chalaz, Tanguy, Bourges, Gaurel, Escande, & Guillot, 1985).

Hysteresis Curve

The area within the hysteresis loop was reduced with time in this study. This can further assist in understanding the changes of the mechanical properties of the posterior lumbar tissues. Energy is absorbed by the lumbar passive tissue during the lengthening period. Reduction of $T_{pt}$ was observed when tissues shorten, but the $T_{pt}$ were not the same at specific angles between flexion and extension phases. This demonstrates the difference in the rate of energy transfer in
and out of the biomaterial between ascending and descending limbs, as is illustrated in Figure 4.7. The area between the curves allows for the measurement of the energy dissipated during cyclic loading (Butler, Grood, Noyes, & Zernicke, 1978). Shrinkage of the hysteresis loop indicated a decrease of energy dissipation due to changes in the properties of the tissues. Previous literature has also discussed the change in viscoelastic properties of the passive tissues when cyclically loaded (Asano, Kaneda, Umehara, & Tadona, 1992; Weisman, Pope, & Johnson, 1980). Specifically, in vitro and in vivo studies indicate a decrease in energy dissipation between ascending and descending limbs with continued cyclic loading (Rigby, 1964; Taylor et al., 1990; Yahia et al., 1991; Yahia, Pigeon, and DeRosiers, 1993).

Application to the Prevention and Rehabilitation of Low Back Disorders

This observation is important to the study of the etiology of low back pain and disorder. Repetitive movement has previously been link to the causation of low back pain and neuromuscular disorders of the spine. The application of these hypotheses to humans has been attributed to work with animal and cadaver models. Traditionally, EMG techniques and mechanical models are used to predict and identify changes in the mechanical properties of the lumbar tissues (Dolan et al., 1994; van Dijke et al., 1999). Mechanical modifications in the supporting passive tissues occur in these animal and cadaver models when the ligaments and fascia are manipulated repetitively (Claude et al., 2003). The results reported in this study provide evidence that laxity within the lumbar passive tissues does occur. An increased level of paraspinal muscle activity after the passive session indicates a neuromuscular compensatory mechanism was triggered to maintain the structural integrity of the spine. The mechanical and neural adaptation to repetitive loading will help us to further understand the etiology of low back disorder associated with this types of motion.
Conclusion

Reduced tension in the lumbar passive tissues was observed as a result of continuous cyclic trunk flexion-extension exercise without active muscle contraction. A consequence of the decreased tension within the passive tissues was increased muscle activity after the passive motion session. This observation is pertinent to the understanding of everyday movements that may lead to low back pain or disorder. The results of this study also filled the gap in the exploration of neuromuscular disorders in humans through the use of animal models. Changes of the hysteresis loop observed from the measured and modeled torque were in agreement. Those changes indicate prolonged trunk flexion motion influences mechanical and neural modifications in the low back. This research provides information suggesting repetitive trunk movements are significant factors in determining the mechanical and neural etiologies of low back pain.

References


CHAPTER 5. GENERAL DISCUSSION

The purpose of this series of experiments, presented in Chapters 2, 3, and 4, was to further the understanding of the mechanisms influencing the flexion-relaxation phenomenon (FRP). The role of mechanical and neural mechanisms in control of the FRP is still debated in the literature. The underlying premise for studying the FRP is to better understand the etiology of low back pain (LBP), injury, and disorder. Increased mechanical loading of the lumbar spine occurs when individuals flex forward to perform a lifting task. As such, the paraspinal muscles would participate in the task actively. Why then, at deep flexion angles, do the paraspinal muscles electrically deactivate in healthy individuals? This deactivation (myoelectric silent period) may provide a window for susceptibility to low back injury and provoke pain. Further analysis of the mechanisms that influence the FRP was performed in this thesis to reconcile current knowledge into a framework that incorporates collaboration between mechanical and neural hypotheses. It is believed that the observations presented in the previous three chapters advance our understanding of the regulatory mechanisms of the FRP and provide insight into the origins of LBP and injury.

Overview of Results

The results of Chapter 2 indicate that continuous flexion-extension movement of the trunk increases the myoelectric silent period of the paraspinal muscles. A modified duration of the myoelectric silent period has been reported previously during continuous lifting exercises (Dickey, McNorton, and Potvin, 2003). This type of observation has been discussed in light of the mechanical load sharing hypothesis. The mechanical load-sharing hypothesis states the responsibility of maintaining the trunk mass is transferred between the back extensor muscles and the posterior spinal ligaments. Increased flexion of the trunk lengthens the posterior passive tissues and therefore increases the tension within them. Prolonged flexion-extension activity
weakens the mechanical properties of the tissues through either creep or tension relaxation at a
given trunk angle (see Chapter 4). Mechanically, weakened passive tissues call for greater
muscle activity and therefore shorten the duration of the silent period over time. The observation
of a prolonged silent period did not support the prediction of the mechanical load-sharing
hypothesis.

The combination of hypothesized mechanical and neural mechanisms may provide an
alternative explanation (Floyd & Silver, 1951; 1955). The mechanical property changes within
the tissues may be reflected in the modified neuromuscular activity resulting from afferent
stimulation (Biedert & Zwick, 1998; Indahl, Kaigle, Reikerås, & Holm, 1997; Krogsgaard,
Dyhre-Poulsen, & Fischer-Rasmussen, 2002).

The influence of gravity was suggested to greatly affect the activation of the paraspinal
muscles as the posterior tissues are loaded. The suggestion was examined in Chapter 3. Pre-
loading the tissues through lengthening was demonstrated to affect the myoelectric deactivation
of the paraspinal muscles when gravitational loading was not presented. Increased flexion of the
trunk also caused an inhibition of the hamstring muscles.

Inhibition of the paraspinal muscles during flexion-extension exercises exposes the low
back to possible injury when external forces are applied. The neuromuscular system must
compensate for this electrical inactivity through other mechanisms in order maintain structural
integrity of the spine. In Chapter 3 elevated myoelectric activity of the abdominal musculature
was observed during the paraspinal silent period, and believed to assist in controlling the
movement and maintaining the integrity of the spine. Debate continues in the literature regarding
the effectiveness of intra abdominal pressure (IAP) and its application to support of the spine
during lifting activities (Cresswell and Thorstensson, 1989; Essendrop, Schibye, and Hye-
Knudsen, 2002; McGill and Norman, 1987; McGill and Sharratt, 1990). Acute loading of the
spine results in greater abdominal activity and increased IAP. Increased IAP is speculative as a compensatory mechanism during a continuous cyclic motion when tension decreases in the posterior tissues (Essendrop & Schibye, 2004).

A decrement of the posterior tissue tension is manifest when cyclic repetitive flexion-extension is performed (Chapter 4). In many active lifting procedures the posterior muscle activity increases as the task duration increases. Many times this is due to fatigue and modified recruitment of the motor units innervating the muscle fibers (Moritani, Nagata, & Muro, 1982). This was a confounding factor in Chapter 2. Passive movement of the trunk via an external mechanical control system assisted in isolating the contributions of the passive tissues to resistance of gravitational loading. Paraspinal muscle activity was maintained at a low level and did not contribute significantly to the continuous movement. Studies utilizing human models have reported diminished passive tension of the hamstrings muscles when the thigh was fixed for durations of less than two minutes (Magnusson, Aagard, Simonsen, & Bojsen-Møller, 1998; Magnusson, Simonsen, Aagard, Gleim, McHugh, & Kjaer, 1995; McHugh, Magnusson, Gleim, & Nicholson, 1992). The reduced passive tension within the low back tissues reported in Chapter 4 is significant as it has not been reported in the literature.

The mechanical properties of the lumbar passive tissues are compromised over ten minutes of continuous passive flexion-extension. It is suggested that compromised lumbar passive tissues result in an increased activation of a greater number of motor units in the lumbar muscles during active sagittal plane movement. As these tissues become more compliant the load tolerance of the spinal system also decreases leading to a compromised stability of the vertebrae. The neuromuscular adaptation to this decreased structural integrity will require either increased activation of the surrounding paraspinal musculature or a modified recruitment pattern of other muscle groups. This explanation does not reconcile the tissue compliance with the increased
silent period observed in Chapter 2. It is surmised that mechanical and neural mechanisms work collectively in the development of the FRP. If correct, the knowledge gained through studying these collaborating mechanisms will provide greater insight to the etiology of LBP and injury.

**Overall Discussion**

**Mechanical Mechanisms**

The viscoelastic properties of the ligaments change with prolonged loading at different loading magnitudes, frequencies, and repetitions (Bulter, Grood, Noyes, Zernicke, & Brackett, 1984; Burstein & Frankel, 1968; Hubbard & Chun, 1988; Yahia, Pigeon, & DeRosiers, 1993). The modification of biomaterial mechanical properties is also affected by the lengthening velocity (Noyes, DeLuca, & Torvik, 1974; Solomonow, Zhou, Baratta, Lu, & Harris, 1999; Yahia, Audet, and Drouin, 1991). It is assumed that the mechanical property changes within the passive tissues affect the ability of the trunk-spine system to maintain sufficient resistance to counter external force application.

Previously, there had been no direct force-based evidence in human models to suggest that the lumbar passive tissues are compromised during flexion-extension activities. The results of Chapter 4 filled this knowledge gap since the data indicate that the mechanical properties of the passive tissues are modified during repetitive cyclic loading. Laxity of the spinal tissues has been measured in a human model through repetitive loading and can be speculated to adversely affect the health of the lumbar spine.

The FRP is an event that can provide an indication of the mechanical changes in the passive tissues. In LBP patients there is no visible sign of the FRP during flexion-extension exercises. Mechanically, it is assumed that a decreased silent period will be present, based upon *in vitro* and *in vivo* animal and human experiments (Claude, Solomonow, Zhou, Baratta, & Zhu, 2003; Dickey et al., 2003; Solomonow et al., 1999). The FRP has been observed to decreased
after being externally loaded during repetitive movement exercises (Dickey et al., 2003). The results of Chapter 4 support the assumption of a decreased silent period due to the mechanical load-sharing hypothesis. One limitation to this experiment was the unmeasured duration of the silent period before and after the passive session. The silent period duration before and after passive movement is a measurement to consider for future research endeavors.

The literature also indicates that load-sharing may occur between regions of the body, such as the thoracic vertebrae, lower levels of the pelvis, and thighs (Andersson, Oddson, Grundström, Nilsson, & Thorstensson, 1996; Toussaint, de Winter, de Haas, de Looze, van Dieën, & Kingma, 1995; van Diëen, Toussaint, Maurice, & Mientjes, 1996). The lumbar region is highly susceptible to injury during lifting actions. Increased activation of the thoracic and lower extremity muscles may partially alleviate the loads imparted upon the lumbar spine. However, LBP and disorder prevalence in labor intensive occupations illustrates the vulnerability of the low back to repetitive loading. The lumbar vertebrae must tolerate the loading forces as the moment applied to the system is the greatest at the lower spinal regions (Chaffin & Park, 1973; Morris, 1973).

Increased abdominal activity may provide indirect evidence for increased intra-abdominal pressure (IAP) when the paraspinal muscles were inhibited. Mechanically, the greater pressure within the abdominal cavity compresses the viscera to stabilize the lumbar region, much like a corset or a work belt function during daily activities. It is unclear whether the IAP compensates for the decreased tension of the posterior paraspinal tissues as continuous cyclic movement is performed. Activation of the abdominal muscles produces both flexion and extension moments (Gardner-Morse & Stokes, 1998; McGill & Kippers, 1994). The extension moment assists the posterior muscles through the direct attachment to the lumbodorsal fascia. However, the activation of these muscles also produces a flexion moment which may cancel out
the effects of the aforementioned extension moment (Gardner-Morse & Stokes, 1998). This is a mechanical mechanism that requires adequate attention to further understand its interaction with the paraspinal muscles.

Neuromuscular fatigue is often discussed when the sources of LBP and disorder are studied. Muscular fatigue occurs as a result of sustained activity/movement which leads to a decreased force output of the muscles (Andersson, Örtengren, & Nachemson, 1976; Davis & Troup, 1964; Sparto & Parianpour, 1998; Sparto, Parianpour, Reinsel, & Simon, 1997). The elevation of myoelectric amplitude and a shift of the power density spectrum to lower frequencies are signs the muscle is fatigued. In Chapter 2 it was difficult to distinguish fatigue from neural adaptations due to mechanical property changes within both muscle and the passive tissues. However, increased myoelectric amplitude and a shift of the power density spectrum to greater frequencies were observed in Chapter 4. Thus, neuromuscular adaptations in the paraspinal muscles are present when the system is in a non-fatigued state.

Neural Mechanisms

Floyd and Silver (1951; 1955) speculated the muscle afferent receptors responded to increased length and tension by initiating an inhibitory reflexive response. These inhibitory responses are speculated to occur in animal models as continuous static or cyclic loading regimens are applied to the ligaments. Mechanoreceptors (Ruffini endings, Pacinian corpuscles, Golgi endings) embedded within the passive tissues are stimulated when a specific threshold level is detected as the length and forces are modified. Neural inhibition of the paraspinal myoelectric activity occurs through afferent stimulation from the alpha and gamma motor systems and synaptic communications with interneurons. As the results of Chapter 3 suggest, lengthening of the posterior tissues may stimulate afferent receptors to inhibit the paraspinal muscles. However, extension resulted in increased activation of the paraspinal muscles.
indicating neural response to elongation does not sufficiently explain the inhibition of the muscle.

The basis for studying the silent period is rooted in the neurophysiological observation of muscle activity. Evoking H-reflexes through external stimulation provides useful information regarding the inhibition and latencies of reflexive responses (Avela, Kyröläinen, & Komi, 1999; Gassel & Ott, 1973; Trontelj, 1973). Discussion of the inhibitory response is inclusive of an antidromic signal in the alpha motor neurons and a contribution from gamma motor neurons to sustain the silent period. Neurophysiological evidence also suggests the influence of higher brain centers and interneurons in the presence of a silent period (Barat, Katz, Lamy, Pénicaud, & Wargon, 2003; Shahani & Young, 1973). The duration of the silent period in these studies is measured in milliseconds but may provide evidence concerning the longer period of myoelectric inactivity in the paraspinal muscles.

The vestibular system is reported to affect the stimulation of the posterior muscles as the head is rotated through the three cardinal planes with respect to the gravity vector (Ali, Rowan, & Iles, 2003; Horak & Hlavacka, 2002). The sensitivity of the labyrinth to changes in the position of the head may contribute to modifications of paraspinal neuromuscular behavior. Stimulation of the vestibular system may reorient the internal reference that the CNS uses to distinguish its relationship with gravity. This argument cannot be dismissed as the inhibition of paraspinal myoelectric activity was observed during supine flexion of the trunk in Chapter 3. Sufficient data are currently lacking in the literature to definitively assert the FRP is attributed to vestibular reflexes.

Amalgamation of Neural and Mechanical Mechanisms

Stimulation of mechanoreceptors within the zygapophysial (facet) joints influences the paraspinal myoelectric silent period (Cavanaugh, Ozaktay, Yamashita, & King, 1996; Indahl et
Continuous repetitive flexion-extension exercise increases the silent period (Chapter 2) and suggests an adapted neural reflex possibly due to mechanical changes in the tissues. Acute loading of the trunk during sagittal plane movements is dictated by the mechanical properties of the posterior trunk tissues. The increased mechanical laxity in these tissues through repetitive loading may allow for further strain within the facet capsules that stimulate the embedded mechanoreceptors. The result of this stimulation would be an inhibition of the surrounding musculature. The trunk inclination angle increased over time (Figure 2.5) and possibly increased the distance between superior and inferior articulating processes. The propensity for this activity suggests that the lengthened silent period would mask any signs of injury or mechanical instability until stimulation of nociceptors (pain receptors) occurs.

Evidence of reflexive activity after cumulative loading may not be apparent immediately. The initial activation of nociceptors is suggested to occur after sufficient passive tissue compliance for injury development has occurred (LaBry, Sbriccoli, Zhou, & Solomonow, 2004; Sbriccoli, Solomonow, Zhou, Baratta, Zhu, & Berger, 2004; Solomonow et al., 1999). The response of monosynaptic protective reflexes to external perturbations could be delayed when an inhibitory signal is present. Neural inhibition of the paraspinal muscles may then be detrimental to the injury preventive mechanisms of the trunk-spine system.

Low Back Pain and Flexion-Relaxation Phenomenon

The underlying mechanisms that modulate the FRP may be instrumental in the progression of LBP. Mechanical loading of the trunk influences episodes of LBP and injury in labor intensive occupations (Andersson, 1981; Svensson, 1982; Svensson & Andersson, 1989). Repetitive trunk flexion-extension performed over a prolonged time period induces a behavior modification in the paraspinal muscles (Chapter 2). Compromised posterior passive tissues are a
consequence of continuous repetitive cyclic loading (Chapter 4). The decreased ability of the passive tissues to maintain external loads and the stability of the trunk-spine system indicate the greater activity of the parapsinal muscles is required.

The decreased ability of the passive tissues to sustain the load (Chapter 4) enhances the opportunity for instability in the trunk-spine system. The increased duration of the FRP observed in Chapter 2 occurred as laxity in the posterior passive tissues was assumed to increase. The lack of active paraspinal muscle involvement compounded with compromised passive tissues increases the possibility that continued mechanical loading will result in trauma to the trunk-spine system. Previously mentioned mechanical mechanisms, such as increased IAP and load sharing among other muscle groups, may assist in maintaining the integrity of the trunk-spine system. However, it is difficult to determine if these mechanisms contribute significantly to load redistribution when the paraspinal muscles are electrically deactivated. Thus, neural inhibition of the protective musculature of the trunk-spine system can jeopardize stability and adversely affect the health and mechanical integrity of the system leading to low back injury and pain.

Relevance of the Observations to Clinical Application

The myoelectric silent period observed in the lumbar paraspinal muscles during full trunk flexion provides a window to possible injury mechanisms during prolonged trunk loading. Mechanically, the dorsal tissues are compromised when repetitively loaded. Neural mechanisms may delay or inhibit neuromuscular activity. The affliction of LBP in individuals may be of a mechanical nature, but the neural component may override the disruption of mechanical stability.

Rehabilitation of LBP in patients through mechanical and neural modalities has been documented. The etiology of LBP, injury and disorder is not fully understood, but these therapies have provided relief to those who suffer from LBP. Stimulation of neural afferents in the posterior passive tissues is used in clinical settings to alleviate pain in patient populations.
(Dreyfuss, Dreyer, and Herring, 1995; Garvey, Marks, and Wiesel, 1989; Indahl et al., 1997).

Researching the mechanisms responsible for the FRP will further the understanding of the etiology of LBP and disorder leading to further preventative and rehabilitative measures.

Conclusions

Trunk flexion-extension activities greatly influenced the behavior of the paraspinal tissues. The increased duration of the FRP resulted from a prolonged repetitive flexion-extension exercise performed over a nine minute period. Significant force decrements within the posterior passive tissues were observed due to passive cyclic flexion-extension movements executed for 10 minutes. Increased activation of the abdominal muscles is suggested to assist in maintaining the integrity of the trunk-spine system during the FRP. The data presented in Chapter 3 support the argument that mechanical factors influence the FRP during acute loading of the spine. Chapters 4 provided further evidence to strengthen the mechanical load-sharing hypothesis as the structural support provided by the passive tissues decreased over a 10 minute period of cyclic movement. The motor control strategy of the neuromuscular system would be expected to recruit the paraspinal muscles for a greater duration of the flexion-extension cycle when the structural support is compromised. However, evidence in Chapter 2 indicates a neural inhibitory reflexive response supersedes the mechanical deficiencies acquired in the trunk-spine system during prolonged cyclic activity. Thus, mechanical mechanisms may contribute to the FRP early during trunk flexion-extension movements with further control of this event regulated by neural mechanisms later in the exercise.

The clinical application of the results presented has specific relevance for preventative and rehabilitative purposes. The FRP may provide a window of susceptibility to injury during prolonged lifting activities. Knowledge gained in the study of the mechanisms that influence the
FRP has direct implications for the underlying mechanisms believed to cause low back injury and subsequent pain.

Future Research

Future research endeavors should further investigate the interaction between the mechanical and neural mechanisms for application in preventative and rehabilitative settings. Adequate preventative measures when performing lifting tasks, such as frequent breaks and modified lifting procedures, are areas of focus. Advancing the study of the mechanisms that regulate the FRP can contribute to the understanding of the neuromuscular control of the paraspinal muscles and the development of LBP.

Currently, acute cyclic loading of the spine is performed to analyze changes in the muscle behavior. Chronic LBP and injury conditions are prevalent in individuals working in labor intensive occupations. Human research should attempt to simulate the movements that are believed to cause these afflictions. Replication of real-world movements can be performed without endangering the individual by use of mechanically controlled devises, such as dynamometers.

The duration of the FRP needs further analysis to further the understanding of the development of LBP and injuries. Mechanically, tensile forces of the passive tissues decrease during repetitive cyclic loading. If fatigue is not a factor in the silent period, then it is assumed the inhibition of the paraspinal muscles is a function of the neural stimulus of the posterior passive tissues. The frequency of the movement, as well as the time requirements to perform the task also need to be considered when looking at how the regulating mechanisms of the FRP affects LBP, injury, and disorder.
References


APPENDIX 1. EXPANDED LITERATURE REVIEW

Introduction

Chronic low back pain has become prevalent in the workplaces of industrialized nations. The routine activity of lifting loads throughout the span of hours during a work day has been assumed to add to this prevalence (Dolan & Adams, 1998; Dolan, Early, & Adams, 1997). The repetitive nature of lifting tasks has been suggested as a prime cause of low back pain reported in the literature (Bonato, Ebenbichler, Roy, Lehr, Posch, Kollmitzer, et al., 2003). Continuous flexion of the trunk has been observed to affect the activity of the low back muscles (Dickey, McNorton, & Potvin, 2003). This is a particular problem as the activity of the low back muscles has been observed to decrease when performing anterior trunk flexion. This phenomenon is known as flexion-relaxation and has been reported in the literature over the past 50 years (Allen, 1948; Floyd & Silver, 1951) (Figure A.1). The reduced paraspinal muscle activity during these flexion and extension movements may play a pivotal role in determining the incidence of idiopathic low back pain, and may provide clues to the etiology of further low back injury.

There are two major hypotheses in the literature regarding the explanation of the flexion-relaxation phenomenon. The mechanical load sharing hypothesis has indicated that the tension within the passive tissues of the lumbar spine increases as the trunk flexes anteriorly, allowing the lumbar muscles to relax (Floyd & Silver, 1951). The second hypothesis discusses a neural adaptation initiated from the stress applied to the passive tissues (Solomonow, Zhou, Baratta, Lu, & Harris, 1999).

Debate between the contributions of these two hypotheses persists in an attempt to understand the mechanisms involved with the flexion-relaxation phenomenon. In fact, these hypotheses should be thought of as providing a composite explanation for the flexion-relaxation
phenomenon. To understand the importance each hypothesis contributes to the explanation of the flexion-relaxation phenomenon it is necessary to look at each separately.

Figure A.1. EMG activity of the lumbar paraspinal muscles during trunk flexion-extension from a standing posture. The flexion-relaxation phenomenon occurs from EMG cessation until re-activation of the EMG signal. The curved line above the EMG signal represents the flexion angle of the trunk during the exercise.

The purpose of this paper is to review evidences related to both hypotheses and how these hypotheses explain the physiology related to prolonged cyclic anterior trunk flexion (Figure A.2). The mechanical load sharing hypothesis has been the predominant explanation for the flexion-relaxation phenomenon. This is based upon invasive and non-invasive measurements of the system’s behavior. Neural adaptation has been reported to a lesser extent, but it has been reported that a reflex response to mechanical and electrical stimulation is present in the human body. Therefore, a second purpose is to report the importance of the neural adaptation when discussing the flexion-relaxation phenomenon.
Figure A.2. Flow chart of the mechanisms hypothesized to influence the flexion-relaxation phenomenon (FRP). Mechanical load-sharing and neural adaptations relating to myoelectric silence have been shown in animal and human models. To better understand the mechanisms involved with the FRP there must be an understanding of the mechanical and neural properties associated with each mechanism, respectively. Once the mechanisms responsible for the FRP are understood, along with the kinematics of the movement, this phenomenon can be applied to work related injury and to understanding their etiologies, as well as being able to better assist patients in the clinical setting.

Work Related Injuries

The latest data from the United States Department of Labor (USDL) reported that injuries to the back have the highest rate of reported injury by employees (USDL, 2003). The current data reflect numbers reported from 2001 and reported that sprains and strains were the leading category for injury and illness when discussing the time away from work (699.9 hours, in 1,000s of hours), while back pain ranks fifth with 42.7 hours of work missed (USDL, 2003).
The ambiguity of categorizing injuries is apparent given the general classification of sprains/strains and back pain. Statistics compiled by the government also subcategorized specific injuries into those that involve musculoskeletal disorders. A musculoskeletal disorder (MSD) is defined as an injury to the muscles, tendons, nerves, ligaments, joints, cartilage, or spinal discs (USDL, 2003). They are not considered acute disorders, but rather injuries that have accumulated over time (chronic). The latest statistics show that one-third (34%) of all work related injuries are of this nature (USDL, 2003).

Injury to the trunk (back and shoulder), incorporating strains and sprains, was reported as being the most instrumental in causing the greatest amount of time away from work, when observing the anatomical location of injuries (USDL, 2003). A worker’s motion and position during an activity was the second leading cause of injury reported. However, repetitive motion in the workplace ranked relatively low as a source of injury (USDL, 2003). The report of a low incidence of injury caused by repetitive motion runs contradictory to the report concerning the number of missed days of work. Carpel tunnel syndrome, one example of a repetitive loading related injury, accounted for the longest number of median days away from work (26 days) (USDL, 2003). In fact, repetitive motions accounted for a median of 18 days away from the workplace, the longest amount among all categories (the second highest was related to falls with 11 median days missed). Those with a categorized sprain/strain or back injury, however, reported that they missed a median of six days of work (USDL, 2003).

Repetitive motions are often prolonged over time, such as a work day, while completing manual tasks. Over time, these day to day motions affect the physiological characteristics of the muscles and other surrounding tissues, which affect the performance of the task (Bonato et al., 2003).
Previous studies using feline models have reported that neuromuscular response to perturbations of the spinal ligaments change with the stiffness of the ligament over time (Jackson, Solomonow, Zhou, Baratta, & Harris, 2001; Solomonow et al., 1999; Williams, Solomonow, Zhou, Baratta, & Harris, 2000). Taken together, the increased strain on tissues and the repetitive nature of a task could be a combination that affects the ability of workers to maintain employment, particularly if the task in question involves movements of the back. This factor is important to keep in mind when studying the flexion-relaxation phenomenon because the repetitive nature of manual labor affects how efficiently a worker performs on the job, as well as time spent outside the workplace.

Flexion-Relaxation Phenomenon

Historical Relevance

Anatomists have discussed the structure and functional importance of the erectors spinae muscle for centuries (Borelli, 1710; Duchenne, 1867, as cited in Floyd & Silver, 1955). They believed that the low back erectors (paraspinal muscles) acted as antigravity muscles, initiating movement in the direction opposite of gravity when humans were in an erect stance. When the upper body (head, arms, and trunk) is flexed anteriorly these muscles are activated to balance the torque acting at the trunk’s flexion axis. The muscles become elongated as the movement proceeds and contract eccentrically in order to control the velocity of the system. Conversely, when the trunk is moved through an extension movement the muscles contract concentrically and allow an action that overcomes the force of gravity. The erector spinae muscles in both situations act as antagonists to the effect of gravity.

It had been assumed that these low back muscles were active throughout the motions of flexion and extension within the sagittal plane. This paradigm was challenged in the late 19th or early 20th century by at least one scientist. According to Floyd and Silver (1951), in 1911 a
German anatomist named Fick hypothesized that the erector muscles were inactive during full anterior flexion of the trunk. Allen (1948) was the first to report the activity of the erector muscles during a flexion-extension exercise performed from a standing position. Electromyography (EMG), via surface electrode placement, was used to record electrical activity from the rectus abdominus and gluteus maximus muscles, as well as the muscles of the low back between the thoracic 10 (T10) and the sacrum from male and female volunteers. The results of his experiment gave substantial evidence to support the hypothesis of Fick that muscle activity of the erectors spinae does in fact decrease close to base-line level (very minimal levels of activity) at full trunk flexion. This observation, however, does not imply that the muscle activity (myoelectric activity) is completely inactive as very low levels of activity were recorded (Allen, 1948).

The reason for this decrease in myoelectric activity at or near full anterior flexion is not well understood. Why would muscles that functioned as antagonists to gravity deactivate when the trunk was at a position of producing peak torque at the lumbar level? In 1955 Floyd and Silver performed an experiment that employed flexion-extension of the trunk in two conditions: without external load, and while lifting a weight from full flexion. Floyd and Silver wanted to understand what exactly was occurring that caused the erector muscles to deactivate at positions near full flexion. Superficial and deep lumbar muscle activities were recorded with surface and needle electrodes, respectively. Cinematic recordings were taken to coincide with EMG data. The results of Floyd and Silver showed that myoelectric silence occurred during both no-load and load conditions. It was reported that the additional weight did increase the activity of the erector muscles during the initial lift, but this activity decreased as the trunk was extended. The activity of the muscles increased again at a position similar to that of the no-load condition.
during extension. Muscle activity recorded from the needle electrodes did not differ from the recordings of the surface electrodes.

Floyd and Silver (1951; 1955) termed the myoelectric silence during full anterior trunk flexion the ‘flexion-relaxation’ phenomenon. They denoted this event when myoelectric activity ceased during anterior flexion and when myoelectric activity resumed during extension. This phenomenon was very perplexing. Floyd and Silver could not find electrical evidence from the deep muscle groups to show that other muscles compensated for the silence of the lumbar muscles. Radiographic recordings taken during different phases of the flexion-extension exercise reported that the erector spinae and intervertebral ligaments did lengthen. Based on this evidence, Floyd and Silver stated that they believed tension increased in the passive tissues.

Not long after Floyd and Silver (1955) published their observations regarding what is currently known as the ‘flexion-relaxation’ phenomenon, Portnoy and Morin (1956) published a study that observed the same phenomenon. Their subjects performed flexion-extension from a standing position while surface EMG were collected from lumbar, rectus femoris, gastrocnemius, gluteal, and hamstrings muscles. Portnoy and Morin noted that the activity of the lumbar and hamstring muscles occurred at the same time as the initiation of flexion. At the beginning of the lumbar ‘flexion-relaxation’ the hamstring muscle activity was maintained. Portnoy and Morin suggested that the hip extensors were controlling the movement of the pelvis during the last portion of full flexion and at the initiation of extension.

Two points of interest were brought forward in order to explain the muscle silence, or ‘flexion-relaxation’ phenomenon in Floyd and Silver (1955) and Portnoy and Morin (1956). Since it was believed that tension within the intervertebral ligaments increased, the ligaments were hypothesized to support the weight of the trunk during anterior flexion, thus allowing the lumbar muscles to ‘relax’ (Floyd & Silver, 1951; Floyd & Silver, 1955; Golding, 1952). These
studies also mentioned a possible reflex inhibition brought about by the increase in tension of the ligaments. Stimulation of mechanoreceptors within the paraspinal muscles and spinal ligaments was hypothesized to be a component of the myoelectric silence (Floyd and Silver, 1955; Portnoy & Morin, 1956). These early studies into the flexion-relaxation phenomenon were indecisive as to how the mechanical load sharing and the neural adaptation hypotheses worked together to explain the myoelectric silence.

Quadruped and human models have been used in an attempt to disseminate the information for both hypotheses. In fact, quadruped models have assisted in explaining the mechanical and physiological functions of the human system. Caution must be taken when applying a quadruped model to humans. The gravitational force acting at a quadruped versus a biped is rotated 90 degrees during stance. This indicates that the erect stature of the human is parallel with gravity and the loads applied to the longitudinal axis are absolutely and relatively greater. Thus, the passive tissues of the human vertebrae must be able to maintain the structural integrity of the system. The function of the upper body is also different, since humans are constantly changing the amount of load maintained by the trunk during such activities as lifting. This affects the amount of tension and the length of the passive tissues. The neural networks that control motion may also be different since the strategies used to compensate for neurological deficiencies may be different.

Kinematics

Lumbar Range of Motion

Measurements of the vertebral column motion, which are widely debated, have included many techniques that attempt to determine the true representation of flexion, extension, and lateral flexion through goniometers, skin surface markers, lumbar tracking systems, and radiography (Ahren, Follick, Council, Laser-Wolston, & Litchman, 1988; Dickey et al., 2003;
Gracovetsky, Kary, Levy, Said, Pitchen, & Helie, 1990; Gracovetsky, Newman, Pawlowsky, Lanzo, Davey, & Robinson, 1995; Gupta, 2001; McClure, Esola, Schreier, & Siegler, 1997; McGorry et al., 2001; Ng, Kippers, Richardson, & Parnianpour, 2001; Porter & Wilkinson, 1997; Sihvonen, 1997; Solomonow et al., 2003; Toussaint et al., 1995; Tully, Wagh, & Galea, 2002; Zhang, Xiong, & Bishop, 2003). These methods of measurement have been very reliable in assessing movement of the lumbar spine. It has been argued that specific means of measuring lumbar range of motion may be more effective than others, however.

Identifying the specific movement pattern of the spine is important in understanding its functional significance during normal activities. In living systems it is very difficult to correctly determine the movement of the individual vertebrae since the boney structure is so deep within the body. Radiography has assisted in determining the flexion angles at which the flexion-relaxation phenomenon occurs (Floyd & Silver, 1955; Gracovetsky et al., 1990). Comparison of the centers of the intervertebral discs with surface markers has allowed researchers to differentiate between rotation of the lumbar spine superficially and the actual rotation of the vertebrae during anterior flexion (Gracovetsky et al, 1990).

There are a number of issues that need to be addressed when looking at these studies. First, it must be understood that superficial markers placed on the skin overlying the spinous processes represent a relative perceived angle of a motion external to that of the vertebrae. The actual angle of motion is represented by this relative measurement. Second, the movement of the markers placed on the skin also complicates the analysis of motion since relative motion exists between the skin and the bone. The skin is a very loose organ that allows for the free movement of the body segments. As such, when movements are performed the deep tissues of bone, muscle, and adipose are able to glide underneath the skin. The third point important to remember is that radiographs can only be taken when a person is in a static position, and this should be
done infrequently since the danger of radiation poisoning does exist. Overall, it must be understood that the angular measurements reported are in fact relative representations of the actual vertebral motion.

Debate remains as to the best methodology for observing spinal motion. Gracovetsky et al. (1990) and Gracovetsky (1995) placed passive reflective markers on skin over the spinous processes from the seventh cervical (C7) to the second sacral (S2) of 40 normal subjects and compared the relative movement of the markers with radiographic data. These researchers observed that the movement of the superficial markers correlated well with the radiographs and the movement of the skin added a low rate of error. However, the amount of adipose tissue to lean muscle mass was not taken into consideration and may affect how the skin movement represents the vertebral motion.

Mayer, Tencer, Kristoferson, and Mooney (1984) performed a test-retest of reliability using the two inclinometer and single inclinometer techniques from normal and patient populations. It was reported that both techniques had low error rates compared to radiographic data. Although, standardization of clinicians/researchers was controlled in both studies no between session testing was performed to confirm repeatability. One drawback of the inclinometer method is that measurements must be recorded with the subject in a static position and this may affect the repeatability if partitioning of angles within the total range of motion is desired. Also, the reading of the inclinometer is dependent upon the hand position of the clinician, and this may change from day to day and patient to patient.

A new method to come forth in the past decade is a 3-D tracking system. This instrument uses electromagnetic signals to record lumbar motion and allows the subject to move through a normal range of motion. Two sensors are rigidly fixed on the skin over the first sacral (S1) and the first lumbar (L1) vertebrae. Movement artifact has been reported to be very low with this
device (Pearcy & Hindle, 1989). Compared with radiographic data the 3-D tracking system has been reported to overestimate the movement range, but has been reported to be very accurate and reliable (Pearcy & Hindle, 1989).

Noninvasive examination of the motion of the spine allows researchers and clinicians the advantage of quantifying healthy and patient spinal range of motion. Flexion and extension of the trunk is not the product of isolated motion of the spine. The muscles and other connective tissues of the spine are integrally connected with the pelvis segment. Thus the movements of the lumbar spine and pelvis are factors in determining the range of motion during trunk flexion-extension and affect the tension application in the tissues of the low back.

Lumbo-Pelvic Rhythm

The concerted effort of the posterior tissues of the human lumbar spine and pelvis must be included in order to explain anterior trunk flexion. The active and passive tissues of the lumbar spine (muscles: multifidus, longissimus, iliocostalis; ligaments: supraspinous, interspinous, anterior longitudinal ligament, posterior longitudinal ligament, ligamentum flavum, etc.) are not isolated during motion. The effects of the forces applied to the system and the resulting motion are dependent upon the relationship between these tissues (Gudavalli & Triano, 1999; Thorstensson, Carlson, Zomlefer, & Nilsson, 1982; Tully et al., 2002). Likewise, motion occurring at one joint affects the neighboring joints as well.

The spine and pelvis are both responsible for initiating the motion of anterior flexion of the trunk (Esola et al., 1996; Porter & Wilkinson, 1997). Esola and colleagues observed that lumbar spinal motion was the major contributor to trunk flexion during the first 50% of the movement in asymptomatic individuals. Towards the middle of the flexion phase the lumbar spine and the pelvis contributed equally to the movement. As this motion continued the role of
the spine decreased, while the role of the pelvis increased towards the completion of the movement.

The lumbar passive tissues become responsible for maintaining the tension and balance the load of the upper body, as stated previously (Floyd & Silver, 1955; Schultz, Haderspeck-Grib, Sinkora, & Warwick, 1985; Tanii & Masuda, 1985; Toussaint et al., 1995). Control of the pelvis is maintained by the tension provided by the hip extensor muscles (gluteals and hamstrings) to the trunk flexion, and this resistance provided by the posterior tissues increases with an increase in the tension level during eccentric contraction (Gupta, 2001). The ability of the hip extensor muscles, such as the hamstrings, to lengthen enables further flexion of the trunk to levels that some in the medical field deem normal for most individuals (Tully et al., 2002). The activity of these muscles, however, has not been shown to affect the onset of the flexion-relaxation phenomenon (Gupta, 2001). In fact, studies have observed a myoelectrical silence within the hamstring muscles during full anterior flexion of the trunk (McGorry et al., 2001; Sihvonen, 1997).

When executing extension from the position of full anterior flexion the hamstring muscles may provide a vital role in initiating this motion. An important element that was discussed earlier was the relationship between the pelvis and the spine. During the last 25% of anterior flexion to the deepest position the pelvis is the primary segment responsible for flexion while performing rotation about the hip joints. When extension is initiated from this deep position the pelvis is responsible for most of the early motion, thus the hamstring muscles and other posterior tissues of the pelvic-femoral complex are responsible, in part, for allowing the trunk to extend (McClure et al., 1997; McGorry et al., 2001). After the initial 25% of the extension, the lumbar spine performs an increasingly important role during extension, and gradually a decrease in the passive tissue tension occurs as the distance between the origin and
insertion decreases. In essence, the rhythm of the spine and pelvis become mirror images between flexion and extension (McClure et al., 1997). Once this passive tissue laxity is assumed, then the myoelectric activity of the paraspinal muscles commences to elevate the trunk to the original erect position.

The ability of the ligaments to provide tensile force may affect how the lumbar spine and pelvis behave during anterior flexion. Mechanical mechanisms have been a focal point when discussing the flexion-relaxation phenomenon. Just as important in the explanation of this phenomenon is the neural control of the muscle. Based upon the mechanical behavior of the passive tissues a neural adaptation may be present, leading to a further understand of why myoelectric silence is occurs during trunk flexion.

Mechanical Mechanisms (Load Sharing Hypothesis)

Debate continues as to the mechanistic explanation of the flexion-relaxation phenomenon and its implications for the integrity of the lumbar spine. Allen (1948), Golding (1952), and Floyd and Silver (1951; 1955) hypothesized that increased stress within posterior ligaments of the spine released muscle tension. In order to facilitate this transfer (load sharing) the position of the lumbar spine must be flexed to a specified angle which allows the spinal ligaments to increase the level of tension due to elongation comparable to that of the tension within the paraspinal muscles.

Andersson, Oddsson, Grundström, Nilsson, and Thorstensson (1996) contended that deep layered muscles of the erector spinae and the quadratus lumborum were responsible for maintaining the load of the upper body during anterior flexion due to increased deep muscle activity they reported. Toussaint et al. (1995) suggested that the thoracic erector spinae may bear the load of the upper body. They reported that thoracic erector spinae activity increased as trunk flexion angle increased thus presenting a possible increase in moment at the thoracic region and
not the lumbar region. McGill and Kippers (1994) suggested that the lumbar muscles did not necessarily diminish their importance since these tissues may provide passive support for the trunk during flexion. Dolan, Mannion, and Adams (1994) have speculated that in addition to the ligament participation in passive extensor moment the lumbar fascia and other non-contractile tissues may also factor into load transfer. However, the majority of the literature has focused on the importance of the ligaments during this process since they cannot be excluded when discussing the load sharing mechanism.

Initially, when the trunk is in the erect position very little muscle activity is needed to maintain posture. The vector of gravity force acting at the center of mass of the upper body passes very close to the center of mass of the whole body and the vertical axis, thus torque produced at the lumbar region is minimal. As the trunk begins anterior flexion the center of mass of the upper body deviates anteriorly from the body’s vertical axis. The activity of the paraspinal muscles increases as the moment arm of the upper body weight increases (Kippers & Parker, 1984). Between mid-flexion and full flexion the activity of the paraspinal muscles decreases to near base-line (myoelectric silence) at trunk inclination angles between 80 and 90 degrees for healthy individuals (Allen, 1948; Floyd & Silver, 1955; Sarti, Lisón, Monfort, & Fuster, 2001; Shirado, Ito, Kaneda, & Strax, 1995). It is at this point during the motion where the passive tissues of the lumbar region are thought to provide enough tension to balance the forces exerted by gravity and therefore the paraspinal muscle activity is no longer required (Floyd & Silver, 1955; Schultz et al., 1985; Tanii & Masuda, 1985; Toussaint et al., 1995).

After sufficient tension in the passive tissues has been established further movement of the trunk in the anterior direction increases this tension to allow the myoelectric activity of the paraspinal muscles to remain low. Once full flexion has been achieved the trunk must then initiate extension in order to return to an erect posture.
During trunk extension the paraspinal muscles must increase myoelectric activity in order to overcome the force of gravity. As the passive tissue length decreases the muscles are activated to provide the force needed to offset the extension moment of the trunk as the initial erect posture is regained.

Golding (1952) assessed the myoelectric activity of healthy individuals and those with low back pain diagnosis. It was believed that the injury to those with low back pain was caused by injury to the passive tissues of the spine, especially the posterior ligaments which were thought to sustain the load of the trunk during flexion. Both groups of individuals performed anterior flexion of the trunk as EMG activity was recorded from the levels of T10, L3, and L4. Healthy subjects showed myoelectric silence during full flexion positions. This silence period was not observed in low back pain (LBP) patients. Golding speculated that the EMG activity of the paraspinal muscles remained high to protect the damaged passive tissues in LBP patients. Based upon the load sharing hypothesis, the activity of the muscles would prevent the increased tension in the ligaments and intervertebral discs, thus preventing any additional trauma to these tissues.

Mechanical Properties of Ligaments

The posterior ligaments of the lumbar spine have been proposed to provide the tension necessary to maintain the torque at the lumbar level to offset the opposing load of the trunk and the gravity force (Allen, 1948; Floyd & Silver, 1955; Schultz et al., 1985; Tani & Masuda, 1985; Toussaint et al., 1995). Previous research conducted in this area has reported that tension within the ligaments increases as the length of the tissues increases (Chalaz, Tanguy, Bourges, Gaurel, Escande, & Guillot, 1985; Dumas, Beaudoin, & Drouin, 1987; Panjabi, Goel, & Takata,
The structural components of these tissues allow the ligaments to strain due to specific tension application (Figures A.3 and A.4).

Figure A.3. Stress-strain curve indicating the mechanical response of a viscoelastic tissue to loading.

Figure A.4. Hysteresis curve indicating the mechanical response of a viscoelastic tissue to loading (arrow pointing up) and release of tension (arrow pointing down). Notice that strain in the tissue is greater at stresses that initially strained the tissue.
Ligaments are constructed of two types of protein fibers that are distinctly different from one another in their mechanical properties (Nachemson & Evans, 1968). Collagen protein fibers, which are concentrated into bundles, make up the minority of the tissue. These proteins are responsible for giving the ligament its resilience to tension, or its viscosity (Sanjeevi, Somanathan, & Ramaswamy, 1982). Elastin, or elastic, fibers make up the majority of the tissue and are responsible for providing the elastic properties which allow the tissue to comply with added tension (Sanjeevi, 1982). These fibers are intertwined within the collagen bundles (Chalaz et al., 1985; Hukins, Kirby, Sikoryn, Aspden, & Cox, 1990).

Creep and Tension-Relaxation

The behavior of the passive tissues is dependent upon the nature of the movement and forces responsible for the movement. Ligaments are viscoelastic (visco – the force production is related to velocity, the greater the velocity the greater the force required; and elastic – the force production is related to length, the greater the length, the greater the force production) which implies that their material properties allow for resistance of mechanical loading, but return to their original state with cessation of mechanical loading (Figure A.4). Tension-relaxation and creep are the two processes commonly referred to when discussing tension and length changes, respectively (Thornton, Oliynyk, Frank, & Shrive, 1997).

The response of the passive tissues to mechanical loading does change over time. One way of testing the properties of the ligaments is to control the length of the tissue (Lam, Frank, & Shrive, 1993). If the ligament is stretched repeatedly to a given length, then the same amount of ligament elongation will produce less stress within the tissue over time (Figure A.5). This manifestation of mechanical manipulation is termed tension-relaxation. This is closely related to the second terminology, creep.
Ligament creep refers to the elongation of the tissue with the same stress due to repeated or constant loading (Thornton et al., 1997) (Figure A.6). Others have defined ligament creep as the abnormal elongation of the ligament due to a constant static or cyclic load applied to the tissue (Panjabi et al., 1982; Solomonow et al., 1999). As stated previously, tension-relaxation is induced by the prolonged application of an external load statically or cyclically, and has been observed to affect the amount of tension a ligament can maintain (Solomonow et al., 1999). These two components are important when explaining the behavior of the ligament to specific perturbations.

![Hysteresis Curve](image)

**Figure A.5. Hysteresis curve indicating the mechanical response of a viscoelastic tissue to loading and release of tension to a constant length over time.** Notice that tension in the tissue decreases (arrow) over time indicating that the tissue cannot sustain constant loads when it is displaced at a constant length. The dashed, dotted, and thinner lines represent the change in the curve over time from the initial (dark line) curve.

The terms tension-relaxation and creep are used to explain the changes in the mechanical properties of the passive tissues, but in fact denote the same result, either by maintaining a constant tension or constant length. The tension-relaxation of a tissue refers to the decrease in the
amount of tension in that tissue due to static or cyclic lengthening to a predetermined position. Tissue creep refers to the change in length of the tissue during either static or cyclic loading in order to maintain a specific tension level. In fact, these two terms can be used to explain the same shift in the hysteresis curve when observing cyclic movements over time. As the number of lengthening and shortening (increased tension application followed by a release of tension) cycles of viscoelastic tissues increases the tension within the tissues decreases (Figure A.5), while the length of the tissues increases (Figure A.6). The decrease in tension and increase in length occurring within the tissues is shown with a shift of the hysteresis curve down and to the right. During the release of applied tension (Figure A.4) and with an increase in the number of cycles, the tension level within the tissue decreases earlier once the shortening phase (release of applied tension) of the subsequent cycle begins.

Figure 6. Hysteresis curve indicating the mechanical response of a viscoelastic tissue to loading and release of tension to a constant stress over time. Notice that strain, creep (arrow), in the tissue progresses over time indicating that the tissue must deform to a greater extent in order to maintain a specific level of stress. The dashed, dotted, and thinner lines represent the change in the curve over time from the initial (dark line) curve.
Many studies have been performed to test the tensile strength of ligaments in order to understand these properties (Chalaz et al., 1985; Dumas et al., 1987; Hukins et al., 1990; Panjabi et al., 1982; Panjabi, Oxland, Yamamoto, & Crisco, 1994; Pintar, Yoganandan, Myers, Elhagediab, & Sances 1992). In vivo experiments using animals have revealed the complex nature of passive tissues. These studies have also assisted in advancing the understanding of the mechanical properties of ligaments in humans. The mechanical behavior of the ligaments, however, is dependent upon their structural composition.

Quadruped Studies

The components of the ligament dictate the response of the tissue to external and internal force applications. Modeling of the tissue behavior has become a fundamental tool to understanding its properties (Liao & Belkoff, 1999; Sanjeevi, 1982). The forces applied to the tissues correspond to the stresses developed and measured within the structure. When the tissue is stressed it adapts to the force application by deforming. This deformation, or strain, allows the tissue to withstand the magnitude of loading and the velocity at which the tissue is being manipulated. Thus, its viscoelastic properties allow the tissue to comply with these loads.

In modeling the ligaments it is important to keep these issues in mind. Such modeling incorporates the stress-strain curve to develop ways in which to understand the viscous and elastic components of the tissue (Sanjeevi, 1982; Sanjeevi, Somanathan, & Ramaswamy, 1982). The primary question that needs to be answered concerns how the ligament responds to loading. Strain increases when stress increases but this relationship has its limits (Panjabi et al., 1982). The elastic component is compromised when the strain is too great when stress continues to increase. The elastic limit is reached when the tissue cannot return to its original length after loading has ceased. Any additional loading of the tissue in excess of the stress applied at the
elastic limit can lead to mechanical failure (Liao & Belkoff, 1999). Over time, the tissue is able to repair itself, but its structural integrity will not be the same as it was previous to the mechanical failure (Abramowitch, Yagi, Tsuda, & Woo, 2003; Bray, Leonard, & Salo, 2003).

A single loading cycle can alter the mechanical behavior and function of the passive tissue. However, ligaments function during bouts of continuous cyclic loading on a daily basis. It has been established that tension-relaxation affects the amount of tension within the ligament when a ligament is fixed at a constant length. Lam et al. (1993) have reported that in vitro cyclic loading of rabbit medial-collateral ligament at a constant length, within physiological limits, also reduces the amount of stress within the tissue over time. This is an important observation since the ligaments and other passive tissues are believed to be very important in providing stability to joints (Oxland, Panjabi, Southern, & Duranceau, 1991). Decreased stress within the ligament would negatively affect the properties of the tissue since laxity would develop within the ligaments over time. The tension-relaxation of the tissue provides an initial understanding of the adaptation to cyclic loading, but creep also negatively affects the behavior of the ligament.

Ligament creep and the cyclic nature of movement further affect the response of the tissue to loading. Thornton et al. (1997) hypothesized that low stresses applied to the tissue during in vitro tension-relaxation tests would correspond to the amount of creep in the medial-collateral ligament of rabbits. An initial prediction was that the tension-relaxation would indicate the amount of creep in the ligament. It was reported that tension within the tissues decreased, but this decreased tension did not correlate with the amount of ligament creep projected in the model. In fact, the model overestimated the creep behavior of in vitro experiments. Thornton et al. stated this observation was due to the creep being further from a linear event than the tension-relaxation. Realize that the initial and final ends of the stress-strain curve are non-linear, but the middle portion is linear. Creep is a time dependent process and as such the level of tension
within the tissue must be maintained at a constant level in order for this process to occur. Conversely, tension within the tissue decreases with tension-relaxation and the possibility of creep development is lowered. Thornton, Frank, and Shrive (2001) suggested that inclusion of fiber recruitment within the ligament may be a variable necessary for predicting creep through tension-relaxation.

The protein fibers that constitute the ligament are grouped into bundles along the length of the tissue. The length of these fibers differs within the bundles. Shorter fibers are recruited first and respond to the initial lengthening of the tissue. When these fibers are stressed near their elastic limits the longer fibers are then recruited to resist the loading within the tissue (Chalaz et al., 1985; Hukins et al., 1990). When shorter fibers cannot maintain the tension level longer fibers are recruited to maintain the tension (Chalaz et al., 1985). The fibers that are not recruited maintain a relaxed, non-tensile, configuration known as crimp. When ligaments are not loaded most fibers within the bundles assume this crimped orientation (Chazal et al.; Hukins et al.; Thornton, Shrive, & Frank, 2002). These animal studies have been in agreement with results obtained from the experiments performed on human cadavers and tissues dissected from living humans.

Human Studies

Information regarding the influence of the ligaments of the spine to human movement has incorporated information from cadavers, as well as quadrupeds. Invasive procedures of studying the passive tissues of quadrupeds cannot be accomplished within humans due to the ethical concerns related to this issue. However, tissues from living individuals have been dissected during corrective surgeries, but these are very few in comparison to cadaver studies. Therefore, most of the information regarding the application of compression, tension, and shear forces has been supplied with the use of cadaver models.
Studies observing the properties of human ligaments have generally been in agreement with those of quadruped experiments. Stress-strain curves have been used to compare the ligament behavior between quadrupeds and humans and have reported that these curves are relatively similar.

Nachemson and Evans (1968) reported that the human cadaver ligaments responded to stress in the same way quadruped ligaments responded. They stressed human ligamentum flavum in vitro, from individuals ranging in age from 13 to 79 years, to mechanical failure. It was observed that ligaments from older individuals failed at lower stresses compared to younger tissues. The stress in the tissue at rest was also less in the older tissues than the younger ligaments. The differences over age groups may be due to fluid concentration and possible changes in the elastic properties of the tissue with age (Dumas et al., 1987; Lam et al., 1993; Liao & Belkoff, 1999).

Post-mortem preservation has been a concern in these types of experiments. However, it has been reported that tissue that had been preserved up to 96 hours and fresh tissue from a living system performed similarly in mechanical stress-strain tests (Chazal et al., 1985).

Isolation of single tissues to understand their behavior is a first step in determining how the tissue responds to stress. Tissues do not perform in isolation and this must be understood when applying the results of cadaver studies to living tissue. Panjabi et al. (1982) isolated a functional unit of the spine by defining what constituted a functional unit. In this case, a functional spinal unit (FSU) was considered as two adjacent lumbar vertebrae, one fixed and the other manipulated via force application, with only the spinal ligaments and intervertebral disc providing the support for the unit. The percent strain in each ligament was observed when linear and rotary forces were applied to the unit in order to understand the how these loads affected the ligament.
Strain in the suprasinous and interspinous ligaments has been a primary interest during anterior flexion motion (Panjabi et al., 1982). As flexion moment increases in the lumbar spine the strain in the supraspinous and interspinous ligaments has been observed to be the greatest (Panjabi et al., 1982). Conversely, when the spine is performing extension the strain in the supraspinous and interspinous ligaments decreases since these tissues are not being stressed. This would indicate that the primary function of these tissues was to resist anterior flexion (Panjabi et al., 1982). These tissues are the most posterior ligaments from the vertebral column. Does this factor into their ability to resist high levels of stress during anterior flexion? What about tissues closer to the vertebral axis of rotation? The longitudinal ligaments, which run along the anterior and posterior of the vertebrae, cannot be factored out of this process of resisting flexion. The greater cross sectional area, compared to the supraspinous and interspinous ligaments, allows these ligaments to resist greater stress while straining very little (Hukins et al., 1990). It has been suggested that the stiffness of the longitudinal ligaments and their close proximity to the axis of rotation during flexion allows the tissue to resist straining versus the supraspinal ligament which was reported to produce great strains at lower stress levels (Hukins et al., 1990).

Chazal et al. (1985) studied 43 spinal ligament tissues from various levels of cadaver and living humans. They performed stress-strain testing in vitro and also observed the behavior of the structural components during various points along the stress-strain curve. It is important to indicate that their procedures fixed the bones with which each ligament was attached, and not the ligament or its boney attachment. They reported that the largest stresses were applied at the intertransverse ligament during the entire stress-strain curve. A limitation to this observation was that only two of these ligaments were tested, and these ligaments were only found in the thoracic region. Panjabi et al. (1982) observed that the transverse ligament was only strained significantly
during lateral bending and was not strained more or less than other lumbar ligaments during flexion-extension or rotation movements.

Chazal et al. did report that the posterior longitudinal ligament and ligamentum flavum maintained the largest amount of stresses during failure tests, while the supraspinous and interspinous ligaments were strained the most. The observation of the stresses reported in ligamentum flavum runs contradictory to that reported in Nachemson and Evans (1968) due to the fact that the ligament was not separated into right and left halves. The reports regarding the supraspinous and interspinous ligaments were in agreement with Panjabi et al. (1982) indicating that these ligaments were strained to a higher degree during anterior flexion. This agreement does not indicate that these tissues were in fact supporting more load since their stiffness levels, or resistance to loading, were lower than that reported in the ligamentum flavum (Chazal et al., Hukins et al., 1990; Nachemson & Evans, 1968; Pintar et al., 1992).

The mechanics of the passive tissues is important when observing the movement of the spine non-invasively. Invasive procedures allow researchers to study the mechanical properties and functions of the tissues, but these procedures may also strain the tissues beyond their physiological limits, something not seen during normal functional activities. In addition, the mechanical properties of the tissues may be compromised if they are taken out of their normal physiological conditions. Non-invasive techniques allow researchers to investigate the functioning of the system in question while maintaining the integrity of its structure.

Neural Adaptation Hypothesis

Mechanical factors influencing the properties of the ligaments have been used to explain the flexion-relaxation phenomenon. Other factors related to these tissues may also affect the behavior of the system. Neural input from joint afferents has been shown to influence the neuromotor activity of muscles (Grigg, Schaible, & Schmidt, 1986; Lundberg, Malmgren, &
Schomberg, 1978; Zimny, Schutte, & Dabezies, 1986). Early in the research of the flexion-relaxation phenomenon it was suggested that neural inhibition of the muscles via ligament afferents played a role in the silence of the muscles (Floyd & Silver, 1955; Portnoy & Morin, 1956). However, these authors could not state with certainty that a reflexive neural response, or adaptation, was present.

Control of voluntary and reflex responses to cutaneous and deep muscle pain have been shown to affect the electrical activity of the paraspinal muscles (Arendt-Neilsen, Graven-Neilsen, Svarrer, & Svensson, 1995; Zedka, Prochazka, Knight, Gillard, & Gauthier, 1999). It has also been suggested that the vestibular system regulates the posture of the body and is an important factor into the neural control of the paraspinal muscles during movement (Ali, Rowen, & Iles, 2003; Hodges, 2001). The extent to which the vestibular system and other supraspinal centers have control over the neuromuscular system at the lumbar level is debatable. The higher brain center control of the paraspinal muscles cannot be questioned, but local reflexive neural responses also influence the muscle behavior.

The response of the neural system to physiological changes in tissues, such as ligaments, intervertebral discs, and muscle, has been reported in previous studies (Biedert & Zwick, 1998; Claude et al., 2003; Eversull, Solomonow, Zhou, Baratta, & Zhu, 2001; Freemont, Peacock, Goupille, Hoyland, O’Brien, & Jayson, 1997; Grigg, et al., 1986; Jackson et al., 2001; Solomonow et al., 1999; Weddell, Feinstein, & Pattle, 1944; Williams et al., 2000). The direct electrical stimulation of neurons with the modulation of rate and magnitude has provided valuable information to understanding neural response to perturbations (Boerboom, Hof, Halbertsma, van Raaij, Schenk & Diercks, 2001; Gossard, Floeter, Kawai, Burke, Chang, & Schiff, 1994; Kang, Choi, & Pickar, 2002; Lafleur, Zynicki, Horcholle-Bossavit, & Jami, 1993; Sakamoto, Yamashita, Takebayashi, Sekine, & Ishii, 2001; Sekine, Yamashita, Takebayashi,
Excitatory post-synaptic potentials (EPSPs) and inhibitory post-synaptic potentials (IPSPs) have been identified by these means of investigation and are useful in the discrimination of the effects of afferent input to the system (Lundburg et al., 1978).

This discrimination is observed when studying a triphasic coordination pattern of agonist and antagonist muscle activity during a movement such as elbow flexion in the transverse plane (Miles, Ives, & Vincent, 1997). The biceps brachii act as the agonist primary mover, while the triceps brachii act as the antagonist. Neural activity in the biceps brachii is very high at the beginning of the movement, compared to the triceps brachii. A neural inhibitory signal is sent to triceps by the central nervous system through the alpha motor system (reciprocal inhibition). This inhibition limits the ability of the opposing muscle group to interfere with the primary movement. The triceps are not entirely devoid of contributing to this movement, however. The velocity of the forearm must decrease near the end of the motion if control of the movement is to be attained. In order for the movement velocity to be controlled the triceps are activated to oppose the movement while biceps activity is reduced. Once the movement has been terminated the activity of the triceps again decreases, while the activity of the biceps increases again to maintain the final position and offset the activity of the triceps (Miles et al., 1997). This example illustrates the ability of the neuromuscular system to coordinate and modulate the activity of opposing muscle groups, which is dependent upon the goal of the movement task.

Neuromotor control of the muscles has also been shown to be dependent upon the passive tissues within and surrounding the joints. Mechanoreceptors within the passive tissues have been reported as being responsible for reflexive responses to changes in length and tension in the passive tissue (Cavanaugh, el-Bohy, Hardy, Getchell, Getchell, & King, 1989; Claude et al.,
The proportion of these receptors differs with respect to the location along the tissues (Bohy, Cavanaugh, Getchell, Bulas, Getchell, & King, 1988; Rhalmi, Yahia, Newman, & Isler, 1993; Schutte, Dabezies, Zimney, & Happle, 1987; Yahia, Newman, & Rivard, 1988). Five receptor types have been reported in these neurohistological studies: Golgi endings, Ruffini end-organs, Ruffini corpuscles, Pacinian corpuscles, and a fifth group of free (non-myelinated) nerve endings were also distinguished and believed to behave as nociceptors (Schutte et al., 1987; Skoglund, 1956; Zimny et al., 1986).

**Feline Experiments: Histology and Physiology**

Early feline studies assisted in understanding the innervations and physiological function of neural endings within the joints (Boyd, 1954; Gardner, 1944). Gardner’s research, in particular, distinguished the types of nerve endings within the joint capsule. Through three staining methods he was able to determine where in and around the joint the nerves terminated, while also reporting the diameters of the cells in order to categorize the signal output frequency. Myelinated axons of the Ruffini type were reported in the joint capsule, but the presence of the Pacinian type was not observed (Gardner, 1944). Although Golgi tendon-organ type endings were reported in the cruciate ligaments other sensory receptors were not observed (Boyd, 1954). It was reported that Ruffini endings were observed in medial collateral ligaments and the regions outside of the joint capsule, but not in the cruciate ligament (Skoglund, 1956). According to Skoglund, small numbers of Pacinian corpuscles were reported as innervating the anterior and posterior of the joint capsule. One method used to indicate the presence of joint receptors actually increased the synovial cavity pressure within the feline knee and reported increased sensory unit recruitment with slow adaptability (Andrew & Dodt, 1953). This observation would
then suggest receptors within the passive tissues served a regulatory function in joint proprioception.

Boyd (1954) reported that two morphological types of receptors were present in the knee joint. One type, identified as the spray type because the numerous endings of the nerve expand or fan-out (spray) over the area, was reported to be responsible for the slowly adapting response to stimulation. The second type was lamellated and believed to initiate the rapid response. Histological data collected from the specimen revealed the small abundance of lamellated in comparison to spray type endings. Based on the evidence reported by Gardner (1944), Boyd noted that the lamellated endings were visually similar to previous observations of Pacinian corpuscles, which give rise to rapidly adapting responses (Gray & Matthews, 1951). However, the presence of Pacinian receptors in the joint capsule was not observed during his experiment. It was also hypothesized that the spray type endings were representative of Ruffini endings, which were thought to respond to deformations in the joint capsule (Boyd, 1954).

Feline Experiments: Neural Behavior

The isolation of nerve endings has been beneficial to the understanding of the importance of the joint afferents. Andrew and Dodt (1953), Boyd (1954), and Lundberg, Malmgren, and Schomburg (1975) isolated nerve endings from the joint capsule and ligaments and then recorded physiological responses to electrical and mechanical stimuli. These studies, although beneficial in distinguishing specific neural responses (adaptations), did not control for input from the higher brain centers which would mask isolated contributions of monosynaptic responses. Skoglund (1956), Holmqvist and Lundberg (1961), and Engberg, Lundberg, and Ryall (1968) assisted in the advancement of isolating neural responses. In vivo experiments were performed on decerebrate felines to observe monosynaptic responses to electrical and mechanical perturbations.
The results of in vivo studies illustrate the intricacies involved in the neural pathways. Skoglund (1956) demonstrated that mechanical stimulation of the feline knee joint by rotation of the tibia about the femur produced specific responses by the neurons to specific displacement. The activity of nerve endings in the passive tissues of the joint were monitored during motions involving flexion/extension, rotation, and abduction/adduction. Skoglund observed that two types of neural adaptive responses, slow and rapid, were present. The slowly adapting response was characterized as a reaction to joint position while the rapidly adapting response was initiated by changes in tension. It has been reported that the response of the nerve endings were sensitive to specific position changes and tension in the ligaments (Andrew & Dodt, 1953).

Joint afferents have also been observed to affect the reflexive response during in vitro and in vivo testing (Biedert & Zwick, 1998; Dyhre-Poulsen & Krogsgaard, 2000; Kang et al., 2002; Sakamoto et al., 2001; Solomonow & Lewis, 2002; Stubbs et al., 1998; Tsuda et al., 2001). In feline studies the passive tissues at such joints as the knee, ankle, and lumbar spine have been manipulated to provide evidence for the existence of reflex arcs between the passive tissues and surrounding musculature (Andrew & Dodt, 1953; Holmqvist & Lundberg, 1961; Skoglund, 1956; Solomonow & Lewis, 2002; Stubbs et al., 1998). These studies are important to understanding the regulatory function of monosynaptic responses versus long-loop responses. The presence of reflexive arcs initiated from the passive tissues would strengthen the argument for neuromuscular regulation at a local level.

In recent years, studies regarding the role of the passive tissues in low back disorders have become a focal issue. Manipulations of feline tissue were conducted through mechanical displacement of spinal ligaments. It was reported that the neural reflexive response to the perturbation at the joint decreased as a function of time (Jackson et al., 2001). Displacement of the supraspinous ligament at constant lengths has been reported to affect the myoelectric activity
of the multifidus muscles suggesting that afferents within the tissue had reached threshold and stimulated the surrounding musculature to prevent further injury via a reflexive response (Jackson et al., 2001; Williams et al., 2000). Within five minutes the muscular reflexive activity was observed to decrease, and was maintained at a low level during the remaining time period as the tension within the ligament decreased (Jackson et al., 2001). Muscle spasms were observed to be sporadic during electrical silence and indicated that possible micro damage to the tissue was occurring. Maintaining a constant displacement of the tissues revealed that neural input to the muscles decreased over time. Would this same event also occur if the tissue load remained constant?

Solomonow et al. (2002) reexamined this issue and fixed the amount of load applied to the tissue in each specimen to either a 20, 30, 50, or 70 Newton (N) load. The observations reported were similar to those regarding constant displacement of the tissue and indicated that a neural adaptation was present in the mechanoreceptors of the ligament (Solomonow et al., 2002). It was also stated that spasms were the product of the nociceptors which responded to pain and damage to the tissues.

Further research has reported that cyclic loading of the ligaments to a constant length also decreases the myoelectric activity of the surrounding musculature (Solomonow et al., 1999). Laxity within the ligament increased over time with each cycle and was hypothesized to be responsible for the decreased afferent output of the mechanoreceptors (Solomonow et al., 1999). Decreased neural input to the muscles from the mechanoreceptors would adversely affect the ability of the surrounding musculature to respond to additional loading of the system and would increase the likelihood of injury.

This information has been vital to the understanding of neural mechanisms within the mammalian system. Based on the evidence provided in feline studies regulation of the paraspinal
musculature may be provided by mechanoreceptors within the spinal ligaments. Further evidence of neural adaptations within humans has been reported to coincide with those of feline experiments.

Human Experiments: Histology and Physiology

Studies involving humans have also examined the nerve supply and monosynaptic reflexive pathways generated in passive tissue. Kennedy, Alexander, and Hayes (1982) performed histological procedures on the passive tissues of human knees that had been amputated. Previously, histological evidence from feline knees indicated a minimal amount of receptors were present in the passive tissues, and no receptor endings were observed in the cruciate ligaments (Skoglund, 1956). However, Golgi-like receptors were identified in both the posterior and anterior cruciate ligaments (Boyd, 1954). Kennedy et al. reported that no specialized receptors were identified in the anterior or posterior cruciate ligaments, but within the joint capsule specialized receptors were identified.

Zimney et al. (1986) researched the passive tissues of the knee through histological methods as well and reported different observations than Kennedy et al. (1982). Based on previous works on feline knee preparations it was thought that mechanoreceptors would be present in the cruciate ligaments (Boyd, 1954; Gardner, 1944; Skoglund, 1956). The staining technique utilized by Zimney et al. was able to distinguish nerve endings within the ligaments. The identification of the morphology of each ending was based on previous animal studies. From these morphological characteristics it was concluded that free nerve endings and two types of mechanoreceptors, Ruffini end organs and Pacinian corpuscles, innervated these tissues.

Schutte et al. (1987) isolated the anterior cruciate ligament to confirm that the histological methods used previously were reliable. They were able to replicate the methodology and identified free nerve endings, Pacinian corpuscles, Ruffini end organs, and another Ruffini-
type mechanoreceptor called Ruffini corpuscles, similar in morphology to Golgi tendon organs. These results indicate that mechanoreceptors that sense tension and movement changes are embedded within the ligaments of the knee. These examples provide evidence to suggest that passive tissues, i.e., ligaments and intervertebral discs, are necessary for the function of the joints in proprioception.

Similar histological studies were performed in the ligaments of the lumbar spine. Yahia et al. (1988) isolated surgically acquired supraspinous, interspinous, and ligamentum flavum ligaments from patients who had disc herniation or idiopathic scoliosis pathologies. Histological analysis showed that two types of mechanoreceptors were present in the ligament specimen. Conversely, Rhalmi et al. (1993) did not observe evidence for the existence of mechanoreceptors in the lumbar spinal ligaments. It was determined that the staining method used between the two studies reflected the difference in the results since the immunoreactive methods by Rhalmi et al. were focused on distinguishing pain receptors. Overall, evidence suggesting the presence of mechanoreceptors within the passive tissues of the spine has been conclusive. This evidence may indeed lead to a further explanation to the behavior of the paraspinal muscles.

Human Experiments: Neural Behavior

In vivo experiments with human subjects have been conducted to identify afferent pathways similar to those researched in felines. Mechanical and electrical stimulation techniques have been utilized to provide definitive evidence that a ligament-muscle reflex arc is present in humans (Biedert & Zwick, 1998; Baerboom et al., 2001 Dyhre-Poulsen & Krogsgaard, 2000; Tsuda et al. 2001; Tsuda et al., 2003). Through direct electrical stimulation of the anterior cruciate ligament (ACL) it was reported that the musculature surrounding the knee (hamstrings and quadriceps) responded with either increased activity or inhibition of activity (Biedert & Zwick, 1998; Dyhre-Poulsen & Krogsgaard, 2000).
Tsuda et al. (2001) reported that stimulation of the ACL in subjects who did not have a history of knee injury produced increased muscle activity. When the hamstring muscles were relaxed a definite change in activity level was seen during ACL stimulation for both healthy individuals and those recovering from arthroscopic knee surgery (Dyrhe-Poulsen & Krogsgaard, 2000; Tsuda et al., 2001). Dyhre-Poulsen and Krogsgaard observed that muscles undergoing voluntary isometric contraction of either flexors or extensors prior to ACL stimulation became inhibited when the ACL was stimulated. The functional significance of this reflexive response, especially in the hamstrings, was explained in terms of muscle regulation during normal movements and posture. The idea that these reflex loops acted as protective responses was dispelled since latencies of over 100 ms were reported and injury to the tissue would occur before the muscles could respond accordingly (Dyhre-Poulsen & Krogsgaard, 2000; Krogsgaard, Dyhre-Poulsen, & Fischer-Rasmussen, 2002). It should be noted that the subjects were recovering from surgical procedures from hours earlier, and the anesthetic used may have hindered the response of the neural system to the electrical stimulation.

Mechanical stimulation of the ACL was performed by Beidert and Zwick (1998) and their results did not support the notion that mechanical loading of the ligament provided a reflexive response in the hamstrings muscles. Beidert and Zwick determined the mechanical load for the human ligament stimulation from empirical data used in feline experiments. A corresponding load was applied to the human ligament, but may not have been enough to attain the threshold level of the mechanoreceptors. Also, small amounts of local anesthesia were used for the skin incisions to allow hook placement around the ligament. Tsuda et al. (2001) argued that artificial mechanical stimulation of the ACL presented two methodological problems: 1) stimulation of mechanoreceptors surrounding the joint capsule was possible and any direct
correlation with the ACL movement and the muscles’ activity would be impaired, and 2) direct contact of the stimulation devise could inadvertently stimulate the mechanoreceptors.

Current evidence in the literature strongly suggests that a ligament-muscle reflex arc does exist in the human knee. Based on this support for afferent control of muscles, which was derived from feline experiments, it can be deduced that such a reflex response is present in the vertebral joints. The behavior of the reflexive response is dependent upon the amount of tension applied to the tissues. This tension is dictated by the amount of lengthening allowed by the lumbar spine. The level of tension would therefore be dependent upon the range of motion of the individual since a greater range of lumbar flexion would allow the ligaments to maximize the amount of tension application.

Research looking into the neural adaptation to passive tissue response during static and cyclic movements in humans has been indecisive. Dickey et al. (2003) observed that the myoelectric silence period decreased over a period of 100 cycles during a controlled 11 second per cycle trunk flexion-extension exercise. Dickey et al. also changed the load condition so that no external loads were lifted during the first and last 10 cycles. Between cycles 10 and 90 external loads were lifted by the subjects. Electromyographic recordings observed from the initial 10 cycles and the last 10 cycles showed that myoelectric silence and the resumption of myoelectric activity occurred at greater angles of trunk flexion after the loads had been lifted. There was no significant difference in the timing of EMG silence and resumption during the loading trials. Dickey et al. suggested that ligament creep had occurred during the loading trials. This suggestion was used to explain the difference between the initial and final 10 no-load trials. However, it was also suggested that a neural mechanism may have been involved since the angles of the initiation of myoelectric silence and the consequent resumption of the EMG did not change during the loading trials.
This may be important when discussing the rate and amount of loading reported to affect the initiation of the flexion-relaxation phenomenon (Sarti et al., 2001). Specifically, rate of motion has been reported to influence the initiation of the flexion-relaxation phenomenon and this was thought to be due to support provided by the elastic forces of the passive tissues to stabilize the vertebrae (Sarti et al., 2001). If these passive tissues are compromised over time through constant tension application then the myoelectric activity of the paraspinal muscles would be maintained during anterior flexion.

Solomonow et al. (2003) recorded the low back EMG from individuals before and after maintaining a seated static flexed lumbar position for 10 minutes. Similar to the findings of Dickey et al. (2003), the EMG amplitude diminished and resumed at trunk flexion angles greater than in the pre-test. It was suggested that creep was being produced in the passive tissues and that this process negatively affected the ability of the passive structures to provide enough support for the load of the trunk.

Olson, Li, and Solomonow (2004) looked at cyclic flexion-extension of the trunk over a 10 minute period in a no-load condition and reported contrary results to the previous reports. The rate of the cycle was controlled at six cycles per minute. As the number of cycles increased over time, the period of myoelectric silence increased. This occurred even though the amplitude of the EMG signal increased, indicating muscle fatigue was present. The initial myoelectric silence and the resumption of EMG activity occurred at decreased trunk flexion angles. This may in fact indicate that a neural adaptation to mechanical lengthening of the tissues is present. The precise neural control of the muscle activity is not so clear. However, based on the fact that mechanoreceptors are located in passive tissues it may be suggested that deformations to the passive tissues affect the neural input to the central nervous system from the mechanoreceptors.
Mechanical Load Sharing and Neural Adaptation Interaction

Floyd and Silver (1951; 1955) indicated in their studies of the flexion-relaxation phenomenon that both mechanical load sharing and neural adaptation hypotheses were somehow responsible for the myoelectric silence during deep trunk flexion. Initially, it seemed logical to explain the flexion-relaxation phenomenon as a mechanical load sharing between the active muscles and the passive posterior lumbar ligaments. However, the lengthening of the ligaments and active tissues would trigger a neural response from afferents sensitive to tension and length changes within both tissues. Evidence for an expected neural response to these length and tension changes were not observed from EMG recordings of the paraspinal muscles. Researchers have postulated that an inhibitory neural response observed in the lumbar paraspinal muscles was due to the influence of mechanoreceptor afferents located within the vertebral passive tissues (Dickey et al., 2003; Kippers & Parker, 1984; Portnoy & Morin, 1956; Solomonow et al., 2003). The question that still remains unanswered is how these mechanical and neural mechanisms interact and result in a ‘relaxed’ lumbar paraspinal muscle group.

Mentioned previously in this discussion was the role of the posterior lumbar passive tissues. Figure A.4 illustrates the mechanical behavior of the passive posterior lumbar ligaments to increased tension. It is this tension within the ligaments, discs, fascia, and zygapophysial joints that may allow the myoelectric activity of the muscles to decrease, initially. However, the tension maintained by the passive tissues is of a very high magnitude and believed to surpass the activation threshold of the afferent receptors (Floyd & Silver, 1955; Kippers & Parker, 1984; Portnoy & Morin, 1956). When the trunk is in maximum flexion the ligaments must not only support the load of the trunk, but must also maintain the integrity of the vertebral joints of the lumbar region. Before discussing this topic any further it must be understood that the myoelectric activity of the lumbar paraspinal muscles may be very low during deep trunk flexion, but the
passive tension (via elastic components) within the muscles may also contribute to the support of
the trunk and the vertebral joint integrity, although to a lesser extent relative to the active muscle
(McGill & Kippers, 1994).

The distance between the insertion and origin of the posterior lumbar tissues increases
with an increase in the anterior trunk flexion angle (McGill & Kippers, 1994). Internal tension
(stress) in the passive tissues also increases as the trunk flexion angle increases. Increased
tension should stimulate mechanoreceptive afferents in the passive tissues in order to prevent
damage to the tissues. Mechanoreceptor afferents primarily respond to changes in tension, and to
a lesser extent changes in length (Boyd, 1954; Gray & Matthews, 1951; McLain & Pickar, 1998;
Yamashita, Cavanaugh, el-Bohy, Getchell, & King, 1990). An increase in the tension level
within the posterior lumbar ligaments would stimulate rapidly adapting Pacinian corpuscles
(afferents) to transmit neural responses to the surrounding musculature via the central nervous
system (Holmquist & Lundberg, 1961). Previous studies with feline models have observed an
increase in the EMG signal of multifidus muscles during the initial increase in ligament tension
(Claude et al., 2003; Solomonow et al., 1999). The behavior of the reflexive response from the
afferents, and that of the surrounding muscles, changes over time during cyclic bouts of loading.

Changes in the mechanical behavior of ligaments during repeated bouts of cyclic loading
have been observed over time (Figures A.5 and A.6). Tension levels within the ligament
decrease as a function of the number of cycles and time (Hukins et al., 1990; Lam et al., 1993). If
the mechanical load sharing hypothesis were solely responsible for the flexion-relaxation
phenomenon, then the myoelectric silence period would decrease since the tension within the
ligaments would not attain a magnitude great enough to support the load of the trunk until later
in the lengthening phase (this is illustrated in Figure A.6). This behavior change in the muscle
EMG has been documented in previous research (Dickey et al., 2003; Solomonow et al., 2003).
The inclusion of a neural influence in the flexion-relaxation phenomenon during cyclic loading introduces a different interpretation to the behavior. Mechanically, the tension level within the ligaments decreases. Mechanoreceptor afferents located within the passive tissues initially respond to changes in the tension level of the tissues, but once the tension levels decrease the stimulus to the afferents also decreases allowing for a reflexive inhibitory response to be sent to the muscle. However, the inhibitory neural reflex is thought to initiate when tension in the passive tissues is the greatest, and this has been observed during the myoelectric silence period of healthy individuals (Floyd & Silver, 1955; Golding, 1952; Shirado et al., 1995). Individuals with chronic low back pain do not show signs of myoelectric silence in the lumbar paraspinal muscles during trunk flexion-extension (Nouwen et al., 2001; Shirado et al., 1995; Sihvonen et al., 1991). This may be due to damage inflicted in the passive tissues which could stimulate the response of free nerve endings and mechanoreceptors that act as pain receptors. (Ahren et al., 1988; Cavanaugh et al., 1989; Lafleur et al., 1993). This passive tissue damage may lead to the inability of these tissues to maintain enough tension to support the load of the trunk. Thus, any changes in the mechanical properties of the passive tissues will affect the type of response (excitatory or inhibitory) evoked from the afferent endings embedded in these tissues.

Clinical Relevance

The flexion-relaxation phenomenon occurs during anterior flexion of the lumbar spine in most individuals. Differences arise in the occurrence of this phenomenon when individuals with chronic low back pain (CLBP) perform this movement (Ahern et al., 1988; Neblett, Mayer, Gatchel, Keeley, Proctor, & Anagnostis, 2003; Shirado et al., 1995; Sihvonen et al., 1991). The incidence of this phenomenon in CLBP patients has been reported infrequently and the explanations for this have been focused on the instability at the vertebral joints. Panjabi et al. (1982) and Panjabi (2003) have suggested that neutral zones (NZ) within the spinal structure
allow minimal motion at the joints without muscle activation. The NZ is a range of normal motion within the vertebral column that does not need muscular activity to maintain integrity. When the outer ranges of the NZ are neared the muscles are activated to prevent possible injury. If the muscles are not activated and the joint motion is beyond the scope of the NZ, then the stability of the joint is compromised and injury will occur.

The stability of the joints is dependent upon the compliance of the surrounding tissues to provide support. Patients with CLBP have infrequently been observed as having the flexion-relaxation phenomenon purportedly due to tissue damage around the joint (Neblett et al., 2003; Shirado et al., 1995; Sihvonen et al., 1991). This may in part be due to the lack of a full range of motion, brought about by decreased joint stability, since the range of motion during anterior flexion was also shown to differ between patients and normal controls (Ahrens et al., 1988; Shirado et al., 1995). Trunk and hip flexion angles in CLBP patients have been shown to be significantly smaller than pain free individuals when performing this task.

Reliability tests that measure range of motion for patients who report chronic low back pain have been questioned in the past few years. The reliability and validity of the clinical test measures of lumbar range of motion have been challenged based on their ambiguity (Zuberbier, Hunt, Kozlowski, Berkowitz, Schultz, Crook, et al., 2001). The test and retest protocols have been reported to have serious flaws that hamper the true diagnosis of chronic low back pain. These testing procedures are dependent upon the repeatability of the range of motion protocols and impairment rating scores. If the measured range of lumbar motion is inconsistent between trials, then the whole procedure must be retested at a later time so that a true determination of range of motion and impairment rating can be performed. This process has implications for those who are being evaluated for work related injuries.
Parks, Crichton, Goldford, and McGill (2003) provided results of lumbar range-of-motion (LROM) testing for individuals who had been referred to a rehabilitation center for determination of disability. These participants were presented with a variety of tests to analyze their functional abilities. This analysis was performed primarily to determine if the range of motion and pain/test scores were correlated. It was also used to assess worker disability due to workplace injuries. A three-dimensional lumbar motion analysis devise securely attached to the first sacral (S1) and twelfth thoracic (T12) vertebrae was used to calculate LROM. Testing consisted of a questionnaire, hand grip, isometric pull and push strength, amount of time they could walk, stand, and sit without reporting pain, and the ability to lift a ‘blind’ weighted box onto a shelf. The authors observed that there was a low correlation between most of the LROM values and the functional test scores. In fact, over half of the participants were advised that they could return to work due to the results of the clinical assessment.

These evaluations of the lumbar range of motion may not be an indicator for the diagnosis of chronic low back pain. Another method of detecting idiopathic low back disorder may provide a better means of diagnosis. Analyses of EMG recordings have reported a constant myoelectric activity of paraspinal muscles is present in CLBP individuals in comparison to the FRP observed in healthy subjects. These EMG recordings may allow for a better assessment of patients with CLBP.

The flexion-relaxation phenomenon has been suggested as being a very useful assessment of individuals with low back pain. It has been reported that muscle activity during flexion-extension exercises distinguished normal individuals and CLBP patients (Golding, 1952; Nouwen, van Akkerveeken, & Versloot, 1987). The implementation of this phenomenon in the clinical setting has not been widely reported. A study by Neblett et al. (2003) was, in fact, the
first reported research using the flexion-relaxation phenomenon to assess the progress of low back pain individuals before and after rehabilitation.

Chronic low back patients were compared to asymptomatic controls during anterior flexion of the trunk. The angle at which the myoelectric activity of the paraspinal muscles ceased was compared between the two groups. After a multifaceted treatment program that lasted three weeks, patients were reevaluated during the anterior flexion task. The results of the patient group to the rehabilitation program indicated that normal muscle activity and function were apparent. The analysis of the onset of the flexion-relaxation phenomenon in patients after treatment was not significantly different from the asymptomatic group. It should be noted that the patient group consisted of individuals who were disabled due to work related injury and the treatment was constructed to allow these patients to resume work.

In the rehabilitative setting it has been thought that range of motion was the best indicator in assessing low back disorders. This method may not be ideal for all conditions, however. Consistent reports of the recordings of paraspinal EMG have indicated that differences between asymptomatic and patient populations are apparent. Thus, the analysis of paraspinal EMG may be a better determinant of assessing low back disorders. This analysis may also assist in developing reasoning behind the CLBP and instituting rehabilitative strategies to allow individuals to function normally.

Conclusion

The flexion-relaxation phenomenon has been explained previously either as purely mechanical load sharing or as a neural adaptation. Early research provided evidence that such a phenomenon was physiologically normal in healthy individuals. Allen (1948), Floyd and Silver (1951; 1955), and Golding (1952) established through electrical recordings that the myoelectric activity of the lumbar paraspinal muscles diminished when individuals performed anterior trunk
The cause of this ‘flexion-relaxation’ phenomenon, as coined by Floyd and Silver, has been discussed for decades.

The mechanical load sharing hypothesis has been cited most often in the literature to explain the occurrence of the flexion-relaxation phenomenon. The passive tissues have been identified as maintaining the load of the trunk during full trunk flexion. Other tissues, such as the deep erector spinae muscles, fascia, and even the elongated superficial paraspinal muscles cannot be discounted and may provide support for the trunk during this position. However, during the evaluation of patients with chronic low back pain (CLBP) the myoelectric activity of the paraspinal muscles has been reported to remain at a high level (Shirado et al, 1995). The explanation for this increased activity was based on the belief that the passive tissues were indeed responsible for maintaining the load of the trunk. Damage to the passive tissues would disable their ability to sustain increased loading. This situation would cause the paraspinal muscles to maintain a high level of activity during full trunk flexion.

The processes of tension-relaxation and creep further complicate the load sharing hypothesis. Increased strain in a ligament will induce a decrease in the amount of tension within the tissue. Dickey et al. (2003) and Solomonow et al. (2003) suggested that creep within the passive tissues delayed the initiation of myoelectric silence. Whether the passive tissues are statically or cyclically loaded did not matter since the results were similar. A contradictory result was observed by Olson et al. (2004). They reported that the initiation of the myoelectric silence occurred at smaller trunk flexion angles over time. Creep of the passive tissues cannot be factored out of this observation, but how is this related to the mechanical load sharing hypothesis?

The results of Olson et al. (2004) may lead to an explanation that incorporates both the mechanical load sharing and neural adaptation hypotheses. Earlier studies of the flexion-
relaxation phenomenon suggested that a neural component may have been involved in the myoelectric silence during full trunk flexion (Allen, 1948; Floyd & Silver, 1955; Portnoy & Morin, 1956). The extent to which the neural system plays a role in this phenomenon is not well understood. Either a monosynaptic or long loop reflex response is a possible explanation, but these can not be discerned since the activity of higher brain centers in humans cannot be factored out from a control perspective. It is known that mechanoreceptors are present within the passive tissues and do respond to changes in tension and displacement, for proprioception. Many questions regarding the function of the mechanoreceptors need to be answered. One of these questions should be do these mechanoreceptors adapt to changes in length and tension within the passive tissues?

Previous studies have monitored the paraspinal muscles during active movement for short and long periods of time (Ahren et al., 1988; Dickey et al., 2003; Sarti et al., 2001). Fatigue may be a problem when analyzing the results from subjects performing continuous trunk flexion-extension (Dickey et al., 2003; Olson et al., 2004). In order to better understand the contribution of the neural system to the flexion-relaxation phenomenon it may be necessary to control the movement of the trunk passively. In so doing, the range of motion of the trunk can be controlled and the creep and/or tension-relaxation of the passive tissues can be simulated. This allows for the monitoring of the myoelectric activity of the paraspinal muscles while factoring out fatigue as a possible determent to the analysis.

Further research into the contribution of the neural system in the flexion-relaxation phenomenon during continuous cyclic motions is needed based upon the evidence presented. The future areas to research should be driven by the following hypotheses: 1) The mechanical loading (magnitude, displacement, and rate) of the passive tissues affects the mechanoreceptor signal output (neural adaptation), 2) Active muscle activity affects the ability of the interpretation of
EMG (neural activity of the muscles), so passive movements of the trunk will provide a better assessment of the importance of the neural adaptation (passive lengthening of tissues: creep/tension-relaxation affect neural adaptation).

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APPENDIX 2. CONSENT FORMS

Louisiana State University-Baton Rouge Campus Consent Form I: Cyclic Flexion-Extension

Study Title: The influence of low back function on the coordination between upper and lower extremities during locomotion.

Performance Sites: Department of Kinesiology, Louisiana State University

Investigators: The following investigators are available for questions.

Name: Li Li, Ph.D.
Department of Kinesiology
Telephone Number: 578-2036

Name: Michael Olson, M.S.
Department of Kinesiology
Telephone Number: 578-2036

Purpose of the Study: To investigate how the low back function influences the coordination control of upper and lower limbs during locomotion.

Subject inclusion: The study includes a group of subjects aged 19-40 years old with no apparent symptoms of low back or extremity pain who agree to participate in the investigation.

Subject exclusion: If the answer to any of the following questions is “YES”, or those who are not in the specified age range are excluded.

1). Has your doctor ever said you have heart trouble?
2). Do you frequently have pains in your heart or chest?
3). Do you often feel faint or have spells of severe dizziness?
4). Has your doctor ever said your blood pressure was too high?
5). Has your doctor ever told you that you have a bone or joint problem, arthritis that has been aggravated by exercise, or might be made worse with exercise?
6). Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
7). Have you been diagnosed with or had any past episodes of severe low back pain?

Description of the Study: The testing will take place in room 3 Gym-Armory on LSU campus. Sites of EMG placement will be cleaned with alcohol and bipolar pre-amplified electrodes will be placed on these sites accordingly to record electric activity of the muscles. Reflective markers will be placed on anatomical landmarks to allow for movement analysis. You will be involved in a warm-up procedure consisting of walking on a motorized treadmill at a comfortable speed.
for five minutes. You will first perform three 20 second walking trials at 2.0, 2.5, and 3.0 miles per hour. You will then participate in one of the following low back stretch protocols: static flexion of the trunk for 20 minutes, or cyclic trunk flexion-extension for 10 minutes at a rate of 6 per minute. A second set of walking trials will then be performed so that the movements of both walking sessions can be collected and analyzed. A recovery session lasting at least 30 minutes will be included to allow the spinal ligaments to recover. In this session you will be lying on your back on a table.

Benefits: The study will not benefit you directly, but may benefit others by its findings.

Risks: You will be exposed to minimum discomfort. A warm up walk will help your muscles get ready for the test. An experimenter will stand next to you, as a spotter, to prevent possible falling in case you should lose balance during testing.

Right to Refuse: Participation in the study is voluntary and you may change your mind and withdraw from the study at any point in the study without penalty.

Privacy: The results of the study may be published. The privacy of participating will be protected and your identity will not be revealed unless legally compelled.

Release of Information: Investigators may review your records in this study, but your identity will be kept confidential.

Financial Information: There is no charge or payment to you regarding any part of the study.

Signatures:

The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to investigators listed above. I understand that if I have questions about subject rights, or other concerns, I may contact Robert Mathews, chairman (578-8692), Institutional Review Board. I agree with the terms above and acknowledge I have been given a copy of the consent form.

________________________  ____________________
Signature of the Subject    Date
Study Title: Affects of the flexion-relaxation phenomenon to trunk flexion and extension performed from different positions.

Performance Sites: Department of Kinesiology, Louisiana State University

Investigators: The following investigators are available for questions.

Name: Li Li, Ph.D.
Department of Kinesiology
Telephone Number: 578-2036

Name: Michael Olson, M.S.
Department of Kinesiology
Telephone Number: 578-2036

Purpose of the Study: To investigate the neuromuscular behavior of the trunk muscles.

Subject inclusion: The study includes a group of subjects aged 19-40 years old with no apparent symptoms who agree to participate in the investigation.

Subject exclusion: If the answer to any of the following questions is "YES", or those who are not in the specified age range are excluded.

1). Has your doctor ever said you have heart trouble?
2). Do you frequently have pains in your heart or chest?
3). Do you often feel faint or have spells of severe dizziness?
4). Has your doctor ever said your blood pressure was too high?
5). Has your doctor ever told you that you have a bone or joint problem, arthritis that has been aggravated by exercise, or might be made worse with exercise?
6). Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
7). Have you been diagnosed with or had any past episodes of severe low back pain?

Description of the Study: The testing will take place in room B2 Gym-Armory on the LSU campus. Sites of EMG placement will be cleaned with alcohol and bipolar pre-amplified electrodes will be placed on these sites accordingly to record electric activity of the muscles. Reflective markers will be placed on anatomical landmarks to allow for movement analysis. You will be involved in a warm-up procedure consisting of walking on a motorized treadmill at a comfortable speed for five minutes. You will perform three exercises five times each. In one exercise you will lie on your back with your legs extended and the arms across the chest. You will be instructed to perform a sit-up from this position and bend as far forward as possible, and then return to the lying position. The second
exercise is the same as the first, only the arms are to be straight along the sides of the body. You will be instructed to perform a sit-up and touch your toes, and then return to the lying position. The third exercise involves starting from an upright standing position and bending down to touch you toes, and then coming back up to the starting position. The speed of your movement will be determined by the cadence of a metronome. You will have five minutes of rest between exercises.

Benefits: The study will not benefit you directly, but may benefit others by its findings.

Risks: You will be exposed to minimum discomfort. A warm up walk will help your muscles get ready for the test.

Right to Refuse: Participation in the study is voluntary and you may change your mind and withdraw from the study at any point in the study without penalty.

Privacy: The results of the study may be published. The privacy of participating will be protected and your identity will not be revealed unless legally compelled.

Release of Information: Investigators may review your records in this study, but your identity will be kept secret.

Financial Information: There is no charge or payment to you regarding any part of the study.

Signatures:

The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to investigators listed above. I understand that if I have questions about subject rights, or other concerns, I may contact Robert Mathews, chairman (578-8692), Institutional Review Board. I agree with the terms above and acknowledge I have been given a copy of the consent form.

___________________________________________________  __________________________
Signature of the Subject                                   Date
Study Title: Passive trunk flexion-extension performed from standing and supine positions.

Performance Sites: Department of Kinesiology, Louisiana State University

Investigators: The following investigators are available for questions.

Name: Li Li, Ph.D.
Department of Kinesiology
Telephone Number: 578-2036

Name: Michael Olson, M.S.
Department of Kinesiology
Telephone Number: 578-2036

Purpose of the Study: To investigate the neuromuscular behavior of the trunk muscles.

Subject inclusion: The study includes a group of subjects aged 19-40 years old with no apparent symptoms who agree to participate in the investigation.

Subject exclusion: If the answer to any of the following questions is "YES", or those who are not in the specified age range are excluded.

1). Has your doctor ever said you have heart trouble?
2). Do you frequently have pains in your heart or chest?
3). Do you often feel faint or have spells of severe dizziness?
4). Has your doctor ever said your blood pressure was too high?
5). Has your doctor ever told you that you have a bone or joint problem, arthritis that has been aggravated by exercise, or might be made worse with exercise?
6). Is there a good physical reason not mentioned here why you should not follow an activity program even if you wanted to?
7). Have you been diagnosed with or had any past episodes of severe low back pain?

Description of the Study: The testing will take place in room B13 Gym-Armory on the LSU campus. You will be involved in a warm-up procedure consisting of walking on a motorized treadmill at a comfortable speed for five minutes. Sites of EMG placement will be cleaned with alcohol and bipolar pre-amplified electrodes will be placed on these sites accordingly to record electric activity of the muscles. An elgon (electrogoniometer) will be placed over the low back to measure the angle of the spine during movement. You will perform two exercises of trunk flexion-extension (bending forward and backward). In one exercise you will lie on your back in a reclining chair with your trunk and pelvis secured and your arms across your chest. You will be instructed to relax while a motor
controls the movement of the chair from a lying position to a position that allows the trunk to bend as far forward ("sit-up") as comfortably possible, and then return to the lying position. No energy is required on your part to perform the "sit-up" exercise. This movement will continue for 20 minutes. The second exercise is the same as the first, only you will be performing the exercise from a standing position. Your trunk will be secured to a rotating arm that will control the movement of your trunk. Your chest will rest against this arm during the entire exercise and the hands should be placed upon the rotating arm. Your pelvis will be secured to a bench which will be securely fastened to the wall. You will be instructed to relax while a motor controls the movement of the rotating arm. This exercise simulates the movement of bending over and touching your toes. No energy is required of you. This movement will continue for 20 minutes.

Benefits: The study will not benefit you directly, but may benefit others by its findings.

Risks: You will be exposed to minimum discomfort. A warm up walk will help your muscles get ready for the test.

Right to Refuse: Participation in the study is voluntary and you may change your mind and withdraw from the study at any point in the study without penalty.

Privacy: The results of the study may be published. The privacy of participating will be protected and your identity will not be revealed unless legally compelled.

Release of Information: Investigators may review your records in this study, but your identity will be kept secret.

Financial Information: There is no charge or payment to you regarding any part of the study.

Signatures:

The study has been discussed with me and all my questions have been answered. I understand that additional questions regarding the study should be directed to investigators listed above. I understand that if I have questions about subject rights, or other concerns, I may contact Robert Mathews, chairman (578-8692), Institutional Review Board. I agree with the terms above and acknowledge I have been given a copy of the consent form.

__________________________________________  ______________________  
Signature of the Subject                           Date
Global dat(4, 30720)
Global col
Sub EMGpr()
For sn = 0 To 2
   sname = "sub0" + Trim(Str(sn + 1))
   Sheets(sname).Select
   For col = 2 To 7
      meanremoval
      rectification
      DFilter
      rescale
   Next col
Next sn
End Sub

Sub meanremoval()
'readin data and remove mean
mean = 0
For I = 1 To 30720
   dat(1, I) = Cells(11 + I, col)
   mean = mean + dat(1, I)
Next I
mean = mean / 30720
For I = 1 To 30720
   dat(2, I) = dat(1, I) - mean
Next I
'For I = 1 To 30720
'Cells(11 + I, 16) = dat(2, I)
'Next I
End Sub

Sub rectification()
For I = 1 To 30720
   dat(3, I) = Abs(dat(2, I))
Next I
End Sub
Sub DFilter()
'Following are the variables needed to be defined outside
'of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 30720      ' Number of points in the set of data being passed in
Const DFPi = 3.1415926
Const DFcutoff = 6       ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfiltertype = "hp"  ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 960       ' Sampling rate of the original data
Const DTypef = "Butterworth"
Dim DFti    ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFA0, DFA1, DFA2
Dim DFb1, DFb2
Dim Dffiltroption As String
ReDim DFDat(DFNumpnt)    ' original data set need to be passed in to the sub
ReDim DFnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
' generate data
For Dfi = 1 To DFNumpnt
    DFDat(Dfi) = dat(3, Dfi)
Next Dfi
' Fourth order, zero lag filter
' correction to cutoff for high-pass filter
Dffiltroption = DFfiltertype
DFti = 1 / DFSrate
If Dffiltroption = "hp" Then
    DFpcut = (1 / (2 * DFti)) - DFcutoff
Else
    DFpcut = DFcutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
' Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
' J. of Applied Biomechanics, 10:374-381
' And also, Robertson, Barden and Dowling
' NACOB II, 1992
If DTypef = "Butterworth" Then
    DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
    DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DTypef = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else

DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
' correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
   DFa1 = -DFa1
   DFb1 = -DFb1
End If
' Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For Dfi = 1 To DFNumpnt
   DFTemp(Dfi + 2) = DFdata(Dfi)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
   DFprime(Dfi) = DFTemp(Dfi)
Next Dfi
For Dfi = 3 To DFNumpnt + 4
   DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi - 1) + DFa2 * DFTemp(Dfi - 2) + DFb1 * DFprime(Dfi - 1) + DFb2 * DFprime(Dfi - 2)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
   DFTemp(Dfi) = DFprime(Dfi)
Next Dfi
For Dfi = DFNumpnt + 2 To 1 Step -1
   DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi + 1) + DFa2 * DFTemp(Dfi + 2) + DFb1 * DFprime(Dfi + 1) + DFb2 * DFprime(Dfi + 2)
Next Dfi
For Dfi = 1 To DFNumpnt
   DFnewdata(Dfi) = DFprime(Dfi + 2)
Next Dfi
For Dfi = 1 To DFNumpnt
   dat(4, Dfi) = DFnewdata(Dfi)
Next Dfi
End Sub
Sub rescale()
Max = -100000
Min = 100000
For I = 51 To 30720 - 51
    If dat(4, I) > Max Then Max = dat(4, I)
    If dat(4, I) < Min Then Min = dat(4, I)
Next I
For I = 1 To 50
    dat(4, I) = 0
Next I
For I = 51 To 30720 - 51
    dat(4, I) = (dat(4, I) - Min) / (Max - Min)
Next I
For I = 30720 - 50 To 30720
    dat(4, I) = 0
Next I
For I = 1 To 30720
    Cells(I + 11, 7 + col) = dat(4, I)
Next I
End Sub

Sub emgpr2() 'low pass filter
For sn = 0 To 11
    sname = "sub0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    For col = 9 To 14
        filter2
        Next col
    Next sn
End Sub

Sub filter2()
Dim dat(6, 30720)
    For I = 1 To 30720
        dat(1, I) = Cells(11 + I, col)
    Next

'Following are the variables needed to be defined outside
'of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 30720      ' Number of points in the set of data being passed in
Const DFPi = 3.1415926
Const DFcutoff = 0.5      ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfiltertype = "lp"  ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 960          ' Sampling rate of the original data
Const DFtypef = "Butterworth"
Dim DFti    ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFa0, DFa1, DFa2
Dim DFb1, DFb2
Dim DFfiltoption As String
ReDim DFdata(DFNumpnt) ' original data set need to be passed in to the sub
ReDim DFNewData(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFPrime(1 To DFNumpnt + 4) As Single
'generate data
For Dfi = 1 To DFNumpnt
    DFdata(Dfi) = dat(1, Dfi)
Next Dfi
'Fourth order, zero lag filter
'correction to cutoff for high-pass filter
DFfiltoption = DFfiltertype
DFti = 1 / DFSrate
If DFfiltoption = "hp" Then
    DFpcut = (1 / (2 * DFti)) - DFcutoff
Else
    DFpcut = DFcutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
'Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
'NACOB II, 1992
If DFtypef = "Butterworth" Then
    DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
    DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DFtypef = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else
    DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
'correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
'Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For Dfi = 1 To DFNumpnt
    DFTemp(Dfi + 2) = DFdata(Dfi)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
    DFprime(Dfi) = DFTemp(Dfi)
Next Dfi
For Dfi = 3 To DFNumpnt + 4
    DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi - 1) + DFa2 * DFTemp(Dfi - 2) + Dfb1 * DFprime(Dfi - 1) + Dfb2 * DFprime(Dfi - 2)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
    DFTemp(Dfi) = DFprime(Dfi)
Next Dfi
For Dfi = DFNumpnt + 2 To 1 Step -1
    DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi + 1) + DFa2 * DFTemp(Dfi + 2) + Dfb1 * DFprime(Dfi + 1) + Dfb2 * DFprime(Dfi + 2)
Next Dfi
For Dfi = 1 To DFNumpnt
    DFnewdata(Dfi) = DFprime(Dfi + 2)
Next Dfi
'***************add ins ****************************
'For Dfi = 1 To DFNumpnt
'    dat(col - 1, Dfi) = DFnewdata(Dfi)
'Next Dfi

'Next Dfi

For I = 1 To DFNumpnt
    Cells(11 + I, 39 + col) = DFnewdata(I)
Next I

End Sub
Sub kin()
For sn = 0 To 2
    sname = "sub0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    resample
Next sn
End Sub

Sub resample()
    ReDim dt(7, 30720)
    For sn = 0 To 2
        sname = "sub26" + Trim(Str(sn + 1))
        Sheets(sname).Select
        For I = 1 To 30720
            dt(1, I) = Cells(11 + I, 1)
            For J = 1 To 6
                dt(J + 1, I) = Cells(11 + I, J + 1)
            Next J
        Next I
        stepsize = 20
        For I = 1 To 30720 Step stepsize
            Cells(12 + (I - 1) / stepsize, 30) = dt(1, I)
            For J = 1 To 6
                Cells(12 + (I - 1) / stepsize, 30 + J) = dt(J + 1, I)
            Next J
        Next I
    Next sn
End Sub

Sub resample2()
    ReDim data(14, 30720)
    For sn = 0 To 2
        sname = "sub26" + Trim(Str(sn + 1))
        Sheets(sname).Select
        For I = 1 To 30720
            data(1, I) = Cells(11 + I, 8)
            For J = 8 To 13
                data(J + 1, I) = Cells(11 + I, J + 1)
            Next J
        Next I
        stepsize = 20
        For I = 1 To 30720 Step stepsize
            Cells(12 + (I - 1) / stepsize, 39) = data(1, I)
            For J = 8 To 13
                Cells(12 + (I - 1) / stepsize, 39 + J) = data(J + 1, I)
            Next J
        Next I
    Next sn
End Sub
Cells(12 + (I - 1) / steps, 32 + J) = data(J + 1, I)
Next J
Next I
Next sn
End Sub
Chapter 2 Computer Programs: Angular Kinematics Calculations

Sub kin()
For sn = 0 To 11
    sname = "sub0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    For col = 3 To 8
        anglecal1
    Next col
Next sn
End Sub

Sub anglecal1()
Pi = 3.1415926
Numbpt = Cells(1, 1)
For I = 1 To Numbpt
    ribx = Cells(6 + I, 3)
    riby = Cells(6 + I, 4)
    iliacx = Cells(6 + I, 5)
    iliacy = Cells(6 + I, 6)
    hipx = Cells(6 + I, 7)
    hipy = Cells(6 + I, 8)
    'trunk angle(alpha)
    If iliacy < riby Then
        Cells(I + 6, 10) = (180 / Pi) * Atn((ribx - iliacx) / (riby - iliacy))
        Else
        Cells(I + 6, 10) = 90 + (180 / Pi) * Atn((iliacy - riby) / (ribx - iliacx))
    End If
    'rib/hip angle(beta)
    If hipy < riby Then
        Cells(I + 6, 11) = 180 + (90 - (180 / Pi)) * Atn((ribx - hipx) / (riby - hipy))
        Else
        Cells(I + 6, 11) = 180 - (180 / Pi) * Atn((hipy - riby) / (ribx - hipx))
    End If
    'beta-180=gamma
    Cells(I + 6, 12) = Cells(I + 6, 11) - 180
Next I
End Sub
Sub EMGpr()
For sn = 0 To 4
    sname = "sub09sit" + Trim(Trim(sn + 1))
    Sheets(sname).Select
    For col = 2 To 7
        meanremoval
        rectification
        DFilter
        rescale
    Next col
Next sn
End Sub

Sub meanremoval()
    'read in data and remove mean
    mean = 0
    For I = 1 To 11520
        dat(1, I) = Cells(11 + I, col)
        mean = mean + dat(1, I)
    Next I
    mean = mean / 11520
    For I = 1 To 11520
        dat(2, I) = dat(1, I) - mean
    Next I
    'For I = 1 To 30720
    'Cells(11 + I, 16) = dat(2, I)
    'Next I
End Sub

Sub rectification()
For I = 1 To 11520
    dat(3, I) = Abs(dat(2, I))
Next I
End Sub
Sub DFilter()
' Following are the variables needed to be defined outside
' of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 11520 ' Number of points in the set of data being passed in
Const DFpi = 3.1415926
Const DFcutoff = 4 ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfiltertype = "hp" ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 960 ' Sampling rate of the original data
Const DTypef = "Butterworth"
Dim DFTi ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFA0, DFA1, DFA2
Dim DBf1, DBf2
Dim DFFiltption As String
ReDim DFdata(DFNumpnt) ' original data set need to be passed in to the sub
ReDim DFnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
' generate data
For Dfi = 1 To DFNumpnt
    DFdata(Dfi) = dat(3, Dfi)
Next Dfi
' Fourth order, zero lag filter
' correction to cutoff for high-pass filter
DFfiltption = DFfiltertype
DFTi = 1 / DFSrate
If DFFiltption = "hp" Then
    DFpcut = (1 / (2 * DFTi)) - DFcutoff
Else
    DFpcut = DFcutoff
End If
DFWC = Tan(DFpi * DFpcut * DFTi)
' Wc need to be corrected for the dual pass
' Murphy and Robertson (1994),
' J. of Applied Biomechanics, 10:374-381
' And also, Robertson, Barden and Dowling
' NACOB II, 1992
If DTypef = "Butterworth" Then
    DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
    DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DTypef = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else
    DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
'correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
'Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For Dfi = 1 To DFNumpnt
    DFTemp(Dfi + 2) = DFdata(Dfi)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
    DFprime(Dfi) = DFTemp(Dfi)
Next Dfi
For Dfi = 3 To DFNumpnt + 4
    DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi - 1) + DFa2 * DFTemp(Dfi - 2) + DFb1 * DFprime(Dfi - 1) + DFb2 * DFprime(Dfi - 2)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
    DFTemp(Dfi) = DFprime(Dfi)
Next Dfi
For Dfi = DFNumpnt + 2 To 1 Step -1
    DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi + 1) + DFa2 * DFTemp(Dfi + 2) + DFb1 * DFprime(Dfi + 1) + DFb2 * DFprime(Dfi + 2)
Next Dfi
For Dfi = 1 To DFNumpnt
    DFnewdata(Dfi) = DFprime(Dfi + 2)
Next Dfi
For Dfi = 1 To DFNumpnt
    dat(4, Dfi) = DFnewdata(Dfi)
Next Dfi
End Sub
Sub rescale()
Max = -100000
Min = 100000
For I = 51 To 11520 - 51
    If dat(4, I) > Max Then Max = dat(4, I)
    If dat(4, I) < Min Then Min = dat(4, I)
Next I
For I = 1 To 50
    dat(4, I) = 0
Next I
For I = 51 To 11520 - 51
    dat(4, I) = (dat(4, I) - Min) / (Max - Min)
Next I
For I = 11520 - 50 To 11520
    dat(4, I) = 0
Next I
For I = 1 To 11520
    Cells(I + 11, 7 + col) = dat(4, I)
Next I
End Sub

Sub emgpr2() 'low pass filter
For sn = 0 To 4
    sname = "sub0sit" + Trim(Str(sn + 1))
    Sheets(sname).Select
    For col = 2 To 7
        filter2
        Next col
    Next sn
End Sub

Sub filter2()
Dim dat(6, 11520)
For I = 1 To 11520
    dat(1, I) = Cells(11 + I, col)
Next
'Mean subtraction and rectification
mn = 0
For I = 1 To 11520
    mn = mn + dat(1, I)
Next
mn = mn / 11520
For I = 1 To 11520
    dat(1, I) = Abs(dat(1, I) - mn)
Next

'Following are the variables needed to be defined outside
'of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 11520 ' Number of points in the set of data being passed in
Const DFPi = 3.1415926
Const DFCutoff = 12  ' Cutoff frequency for either a hi-pass or a low-pass
Const DFFilterType = "lp"  ' can be either "lp" for low pass or "hp" for high pass
Const DFSRate = 960     'Sampling rate of the original date
Const DFType = "Butterworth"
Dim DFt     ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFA0, DFA1, DFA2
Dim DBf1, DBf2
Dim DFFilterOption As String
ReDim DFdata(DFNumpnt)   ' original data set need to be passed in to the sub
ReDim DFnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
'generate data
For Dfi = 1 To DFNumpnt
  DFdata(Dfi) = dat(1, Dfi)
Next Dfi
'Fourth order, zero lag filter
'correction to cutoff for high-pass filter
DFFilterOption = DFFilterType
DFti = 1 / DFSRate
If DFFilterOption = "hp" Then
  DFpcut = (1 / (2 * DFti)) - DFCutoff
Else
  DFpcut = DFCutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
'Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
 'NACOB II, 1992
If DFType = "Butterworth" Then
  DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
  DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DFType = "Butterworth" Then
  DFk1 = Sqr(2) * DFWC
Else
  DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFA0 = DFk2 / (1 + DFk1 + DFk2)
DFA1 = 2 * DFA0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
' correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
' Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For Dfi = 1 To DFNumpnt
    DFTemp(Dfi + 2) = DFdata(Dfi)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
    DFprime(Dfi) = DFTemp(Dfi)
Next Dfi
For Dfi = 3 To DFNumpnt + 4
    DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi - 1) + DFa2 * DFTemp(Dfi - 2) + DFb1 * DFprime(Dfi - 1) + DFb2 * DFprime(Dfi - 2)
Next Dfi
For Dfi = 1 To DFNumpnt + 4
    DFTemp(Dfi) = DFprime(Dfi)
Next Dfi
For Dfi = DFNumpnt + 2 To 1 Step -1
    DFprime(Dfi) = DFa0 * DFTemp(Dfi) + DFa1 * DFTemp(Dfi + 1) + DFa2 * DFTemp(Dfi + 2) + DFb1 * DFprime(Dfi + 1) + DFb2 * DFprime(Dfi + 2)
Next Dfi
For Dfi = 1 To DFNumpnt
    DFnewdata(Dfi) = DFprime(Dfi + 2)
Next Dfi
' ******************* add ins **************************
' For Dfi = 1 To DFNumpnt
'    dat(col - 1, Dfi) = DFnewdata(Dfi)
' Next Dfi

For I = 1 To DFNumpnt Step 20
    Cells(11 + (I + 19) / 20, 44 + col) = DFnewdata(I)
    Cells(11 + (I + 19) / 20, 45) = (1 / 46) * (I - 1) / 20
Next I
End Sub
Chapter 3 Computer Programs: Standing Angular Kinematics Calculations

Sub sheet()
For sn = 0 To 4
    sname = "sub0stand" + Trim(Str(sn + 1))
    Sheets(sname).Select
Next sn
End Sub

Sub anglecal1()
Pi = 3.1415926
Numbpt = Cells(1, 1)
For I = 1 To Numbpt
    shoulderx = Cells(6 + I, 3)
    shouldery = Cells(6 + I, 4)
    ribx = Cells(6 + I, 5)
    riery = Cells(6 + I, 6)
    iliacx = Cells(6 + I, 7)
    iliacy = Cells(6 + I, 8)
    hipx = Cells(6 + I, 9)
    hipy = Cells(6 + I, 10)

    'time
    Cells(I + 6, 16) = Cells(I + 6, 2)

    'trunk angle1
    If riery < shouldery Then
        Cells(I + 6, 17) = (180 / Pi) * Atn((shoulderx - ribx) / (shouldery - riery))
    Else
        Cells(I + 6, 17) = 90 + (180 / Pi) * Atn((riery - shouldery) / (shoulderx - ribx))
    End If

    'trunk angle2
    If iliacy < riery Then
        Cells(I + 6, 18) = (180 / Pi) * Atn((ribx - iliacx) / (riery - iliacy))
    Else
        Cells(I + 6, 18) = 90 + (180 / Pi) * Atn((iliacy - riery) / (ribx - iliacx))
    End If

    'rib/hip angle
    If hipy < riery Then
        Cells(I + 6, 19) = (90 - (180 / Pi)) * Atn((ribx - hipx) / (riery - hipy))
    Else
        Cells(I + 6, 19) = 51 - (180 / Pi) * Atn((hipy - riery) / (hipx - ribx))
    End If
Next I
End Sub
Sub sheet()
For sn = 0 To 4
    sname = "sub0sit" + Trim(Str(sn + 1))
    Sheets(sname).Select
Next sn
End Sub

Sub anglecal1()
Pi = 3.1415926
Numbpt = Cells(1, 1)
For I = 1 To Numbpt
    shoulderx = Cells(6 + I, 3)
    shouldery = Cells(6 + I, 4)
    ribx = Cells(6 + I, 5)
    riaby = Cells(6 + I, 6)
    iliacx = Cells(6 + I, 7)
    iliacy = Cells(6 + I, 8)
    hipx = Cells(6 + I, 9)
    hipy = Cells(6 + I, 10)

    'trunk angle 1
    If riaby < shouldery Then
        Cells(I + 6, 17) = (180 / Pi) * Atn((shoulderx - ribx) / (shouldery - riaby))
    Else
        Cells(I + 6, 17) = -90 + (180 / Pi) * Atn((riaby - shouldery) / (shoulderx - ribx))
    End If

    'trunk angle 2
    If iliacy < riaby Then
        Cells(I + 6, 18) = (180 / Pi) * Atn((ribx - iliacx) / (riaby - iliacy))
    Else
        Cells(I + 6, 18) = -90 + (180 / Pi) * Atn((iliacy - riaby) / (ribx - iliacx))
    End If

    'rib/hip angle
    If hipy < riaby Then
        Cells(I + 6, 19) = 50 + (90 - (180 / Pi)) * Atn((ribx - hipx) / (riaby - hipy))
    Else
        Cells(I + 6, 19) = (180 / Pi) * Atn((hipy - riaby) / (hipx - ribx))
    End If

    'time
    Cells(I + 6, 16) = Cells(I + 6, 2)
Next I
End Sub
Sub EMGpr() 'EMG analysis
For sn = 0 To 7
    sname = "sub0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    For col = 2 To 5
        meanremoval
        rectification
        DFilter
        rescale
    Next col
Next sn

For sn = 0 To 7
    sname = "amiller0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    For col = 9 To 12
        filter2
    Next col
Next sn
End Sub

Sub meanremoval()
' read in data and remove mean
Mean = 0
For col = 2 To 5
    For I = 1 To 12000
        dat(1, I) = Cells(2 + I, col)
        Mean = Mean + dat(1, I)
    Next I
Mean = Mean / 12000
For I = 1 To 12000
    dat(2, I) = dat(1, I) - Mean
Next I
    For I = 1 To 12000
        Cells(2 + I, col + 10) = dat(2, I)
    Next I
Next col
End Sub
Sub rectification()
For col = 12 To 15
    For I = 1 To 12000
        dat(3, I) = Abs(dat(2, I))
        dat(2, I) = Cells(2 + I, col)
        dat(3, I) = Abs(dat(2, I))
    Next I
Next I
Next col
End Sub

Sub DFilter()
' Dim dat(6, 12000)
For col = 17 To 20
    For I = 1 To 12000
        dat(1, I) = Cells(2 + I, col)
    Next
' Following are the variables needed to be defined outside
' of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 12000  ' Number of points in the set of data being passed in
Const DFpi = 3.1415926
Const DFcutoff = 4      ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfiltertype = "hp"  ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 10000    ' Sampling rate of the original data
Const DFtypef = "Butterworth"
Dim DFti    ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFA0, DFA1, DFA2
Dim DFb1, DFb2
Dim DFFiltoption As String
ReDim DFdata(DFNumpnt)  ' original data set need to be passed in to the sub
ReDim DFnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
' generate data
For DFi = 1 To DFNumpnt
    DFdata(DFi) = dat(3, DFi)
Next DFi
'Fourth order, zero lag filter
' correction to cutoff for high-pass filter
DFFiltoption = DFfiltertype
DFti = 1 / DFSrate
If DFFiltoption = "hp" Then
    DFpcut = (1 / (2 * DFti)) - DFcutoff
End If
Next DFi
Else
  DFpcut = DCutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
' We need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
'NACOB II, 1992
If DFtypef = "Butterworth" Then
  DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
  DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DFtypef = "Butterworth" Then
  DFk1 = Sqr(2) * DFWC
Else
  DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
' correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
  DFa1 = -DFa1
  DFb1 = -DFb1
End If
' Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For DFi = 1 To DFNumpnt
  DFTemp(DFi + 2) = DFdata(DFi)
Next DFi
For DFi = 1 To DFNumpnt + 4
  DFprime(DFi) = DFTemp(DFi)
Next DFi
For DFi = 3 To DFNumpnt + 4
  DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi - 1) + DFa2 * DFTemp(DFi - 2) + DFb1 * DFprime(DFi - 1) + DFb2 * DFprime(DFi - 2)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFTemp(DFi) = DFprime(DFi)
Next DFi
For DFi = DFNumpnt + 2 To 1 Step -1
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi + 1) + DFa2 * DFTemp(DFi + 2) + DFa1 * DFprime(DFi + 1) + DFa2 * DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    DFnewdata(DFi) = DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    dat(4, DFi) = DFnewdata(DFi)
Next DFi

For I = 1 To DFNumpnt
    Cells(2 + I, 15 + col) = DFnewdata(I)
Next I

Next col

End Sub

' total number of rows: 12000
Sub rescale()
    Max = -100000
    Min = 100000
For col = 17 To 20
    For I = 1 To 12000
        dat(4, I) = Cells(2 + I, col)
    Next I
    For I = 51 To 11993 - 51
        If dat(4, I) > Max Then Max = dat(4, I)
        If dat(4, I) < Min Then Min = dat(4, I)
    Next I
    For I = 1 To 50
        dat(4, I) = 0
    Next I
    For I = 51 To 12000 - 51
        dat(4, I) = (dat(4, I) - Min) / (Max - Min)
    Next I
    For I = 12000 - 50 To 12000
        dat(4, I) = 0
    Next I
    For I = 1 To 12000
        Cells(I + 2, col + 5) = dat(4, I)
    Next I
Next col
End Sub
Sub filter2()
Dim dat(6, 12000)
For col = 22 To 25
  For I = 1 To 12000
    dat(1, I) = Cells(2 + I, col)
  Next
'Following are the variables needed to be defined outside
'of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 12000 ' Number of points in the set of data being passed in
Const DFPi = 3.1415926
Const DFCutoff = 4 ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfilterType = "lp" ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 10000 ' Sampling rate of the original date
Const Dftypef = "Butterworth"
Dim DFti ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFA0, DFA1, DFA2
Dim DFB1, DFB2
Dim DFFiltroption As String
ReDim DFdata(DFNumpnt) ' original data set need to be passed in to the sub
ReDim DFnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
'generate data
For DFi = 1 To DFNumpnt
  DFdata(DFi) = dat(1, DFi)
Next DFi
'Fourth order, zero lag filter
'correction to cutoff for high-pass filter
DFFiltroption = DFfilterType
DFti = 1 / DFSrate
If DFFiltroption = "hp" Then
  DFpcut = (1 / (2 * DFti)) - DFCutoff
Else
  DFpcut = DFCutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
'Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
'NACOB II, 1992
If Dftypef = "Butterworth" Then
  DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else

DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DTypef = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else
    DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
'correction to coefficients for high-pass filter
If DFiltoption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
'Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For DFi = 1 To DFNumpnt
    DFTemp(DFi + 2) = DFdata(DFi)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFprime(DFi) = DFTemp(DFi)
Next DFi
For DFi = 3 To DFNumpnt + 4
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi - 1) + DFa2 * DFTemp(DFi - 2) + DFb1 * DFprime(DFi - 1) + DFb2 * DFprime(DFi - 2)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFTemp(DFi) = DFprime(DFi)
Next DFi
For DFi = DFNumpnt + 2 To 1 Step -1
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi + 1) + DFa2 * DFTemp(DFi + 2) + DFb1 * DFprime(DFi + 1) + DFb2 * DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    DFnewdata(DFi) = DFprime(DFi + 2)
Next DFi
'***************add ins **********************

' For Dfi = 1 To DFNumpnt
'  dat(col - 1, Dfi) = DFnewdata(Dfi)

' Next Dfi

  For I = 1 To DFNumpnt
    Cells(2 + I, 5 + col) = DFnewdata(I)
  Next I

Next col
End Sub
Chapter 4 Computer Programs: EMG Mean Removal, Rectification, High and Low Pass Filtering, and Rescaling from Passive Session

Global dat(4, 60000)
Global col
Sub EMGpr() 'EMG analysis
    For sn = 0 To 9
        sname = "amiller0" + Trim(Str(sn + 1))
        Sheets(sname).Select
        For col = 2 To 5
            meanremoval
            rectification
            DFilter
            rescale
        Next col
    Next sn
    For sn = 0 To 9
        sname = "amiller0" + Trim(Str(sn + 1))
        Sheets(sname).Select
        For col = 9 To 12
            filter2
        Next col
    Next sn
    End Sub

Sub meanremoval()
' read in data and remove mean
    Mean = 0
    For col = 2 To 5
        For I = 1 To 10502
            dat(1, I) = Cells(2 + I, col)
            Mean = Mean + dat(1, I)
        Next I
    Mean = Mean / 10502
    For I = 1 To 10502
        dat(2, I) = dat(1, I) - Mean
    Next I
    For I = 1 To 10502
        Cells(2 + I, col + 10) = dat(2, I)
    Next I
    Next col
End Sub
Sub rectification()
For col = 12 To 15
  For I = 1 To 10502
    dat(3, I) = Abs(dat(2, I))
    dat(2, I) = Cells(2 + I, col)
    dat(3, I) = Abs(dat(2, I))
  Next I
For I = 1 To 10502
  Cells(2 + I, col + 5) = dat(3, I)
Next I
Next col
End Sub

Sub DFilter()
' Dim dat(6, 30002)
For col = 17 To 20
  For I = 1 To 10502
    dat(1, I) = Cells(2 + I, col)
  Next
' Following are the variables needed to be defined outside
' of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 10502    ' Number of points in the set of data being passed in
Const DFPi = 3.1415926    
Const DFcutoff = 10        ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfiltertype = "hp" ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 10000      ' Sampling rate of the original data
Const DTypef = "Butterworth"
Dim DFi      ' time interval (period) of the original data, 1/sampling rate
Dim DPFcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFa0, DFa1, DFa2
Dim DFb1, DFb2
Dim DFfiltroption As String
ReDim DFdata(DFNumpnt)  ' original data set need to be passed in to the sub
ReDim Dnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DPrime(1 To DFNumpnt + 4) As Single
' generate data
For DFi = 1 To DFNumpnt
  DFdata(DFi) = dat(3, DFi)
Next DFi
' Fourth order, zero lag filter
' correction to cutoff for high-pass filter
DFfiltroption = DFfiltertype
DFTi = 1 / DFSrate
If DFTi = "hp" Then
  DFpcut = (1 / (2 * DFTi)) - DFcutoff
Else
   DFpcut = DFcutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
'Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
'NACOB II, 1992
If DFtypef = "Butterworth" Then
   DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
   DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DFtypef = "Butterworth" Then
   DFk1 = Sqr(2) * DFWC
Else
   DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
'correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
   DFa1 = -DFa1
   DFb1 = -DFb1
End If
'Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For DFi = 1 To DFNumpnt
   DFTemp(DFi + 2) = DFdata(DFi)
Next DFi
For DFi = 1 To DFNumpnt + 4
   DFprime(DFi) = DFTemp(DFi)
Next DFi
For DFi = 3 To DFNumpnt + 4
   DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi - 1) + DFa2 * DFTemp(DFi - 2) + DFb1 * DFprime(DFi - 1) + DFb2 * DFprime(DFi - 2)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFTemp(DFi) = DFprime(DFi)
Next DFi
For DFi = DFNumpnt + 2 To 1 Step -1
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi + 1) + DFa2 * DFTemp(DFi + 2) + DFb1 * DFprime(DFi + 1) + DFb2 * DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    DFnewdata(DFi) = DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    dat(4, DFi) = DFnewdata(DFi)
Next DFi

For I = 1 To DFNumpnt
    Cells(2 + I, 15 + col) = DFnewdata(I)
Next I
Next col
End Sub

' total number of rows: 30002
Sub rescale()
    Max = -100000
    Min = 100000
For col = 17 To 20
    For I = 1 To 10502
        dat(4, I) = Cells(2 + I, col)
    Next I
    For I = 51 To 10502 - 51
        If dat(4, I) > Max Then Max = dat(4, I)
        If dat(4, I) < Min Then Min = dat(4, I)
    Next I
    For I = 1 To 50
        dat(4, I) = 0
    Next I
    For I = 51 To 10502 - 51
        dat(4, I) = (dat(4, I) - Min) / (Max - Min)
    Next I
    For I = 10502 - 50 To 10502
        dat(4, I) = 0
    Next I
    For I = 1 To 10502
        Cells(I + 2, col + 5) = dat(4, I)
    Next I
Next col
End Sub
Sub filter2()
Dim dat(6, 10502)
For col = 22 To 25
    For I = 1 To 10502
        dat(1, I) = Cells(2 + I, col)
    Next
Next

'Following are the variables needed to be defined outside
' of the sub in order to get the sub "DFilter" to operate
Const DFNumpnt = 10502      ' Number of points in the set of data being passed in
Const DFpi = 3.1415926
Const DFcutoff = 4      ' Cutoff frequency for either a hi-pass or a low-pass
Const DFfiltertype = "lp" ' can be either "lp" for low pass or "hp" for high pass
Const DFSrate = 10000       'Sampling rate of the original date
Const DFtypef = "Butterworth"
Dim DFti ' time interval (period) of the original data, 1/sampling rate
Dim DFpcut
Dim DFWC
Dim DFk1, DFk2, DFk3
Dim DFA0, DFA1, DFA2
Dim DBf1, DBf2
Dim DFfiltoption As String
ReDim DFdata(DFNumpnt)    ' original data set need to be passed in to the sub
ReDim DFnewdata(DFNumpnt) ' Filtered data at the end of the sub
ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
'generate data
For DFi = 1 To DFNumpnt
    DFdata(DFi) = dat(1, DFi)
Next DFi

'Fourth order, zero lag filter
'correction to cutoff for high-pass filter
DFfiltoption = DFfiltertype
DFti = 1 / DFSrate
If DFfiltoption = "hp" Then
    DFpcut = (1 / (2 * DFti)) - DFcutoff
Else
    DFpcut = DFcutoff
End If
DFWC = Tan(DFpi * DFpcut * DFti)
'Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
'NACOB II, 1992
If DFtypef = "Butterworth" Then
    DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
    DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
If DFTypef = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else
    DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFb3
DFb2 = 1 - (2 * DFa0) - DFk3
' correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
' Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 2))
For DFi = 1 To DFNumpnt
    DFTemp(DFi + 2) = DFdata(DFi)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFprime(DFi) = DFTemp(DFi)
Next DFi
For DFi = 3 To DFNumpnt + 4
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi - 1) + DFa2 * DFTemp(DFi - 2) + DFb1 * DFprime(DFi - 1) + DFb2 * DFprime(DFi - 2)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFTemp(DFi) = DFprime(DFi)
Next DFi
For DFi = DFNumpnt + 2 To 1 Step -1
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi + 1) + DFa2 * DFTemp(DFi + 2) + DFb1 * DFprime(DFi + 1) + DFb2 * DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    DFnewdata(DFi) = DFprime(DFi + 2)
Next DFi
For Dfi = 1 To DFNumpnt
    dat(col - 1, Dfi) = DFnewdata(Dfi)
Next Dfi

For I = 1 To DFNumpnt
    Cells(2 + I, 5 + col) = DFnewdata(I)
Next I

Next col
End Sub
Sub IEMG() ‘trunk flexion
‘r = sample rate
r = 1000

‘flexion
For I = 12 to 12
  Cells (1, I) = “=sum(L3:L4770)”
  Cells (2, I) = Cells (1, I)/r
Next I

For I = 13 to 13
  Cells (1, I) = “=sum(M3:M4770)”
  Cells (2, I) = Cells (1, I)/r
Next I

‘extension
For I = 12 to 12
  Cells (1, I) = “=sum(L4771:L12002)”
  Cells (2, I) = Cells (1, I)/r
Next I

For I = 13 to 13
  Cells (1, I) = “=sum(M4771:M12002)”
  Cells (2, I) = Cells (1, I)/r
Next I

Cells (2, 14) = “=average(L2:M2)”

End Sub
Chapter 4 Computer Programs: Trunk Inertia and Passive Torque Calculations

Sub Model() 'model analysis
For sn = 0 To 9
    sname = "0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    torque1
    torque2
Next sn
End Sub

Sub torque1() ' trunk flexion
n = Cells(4, 12)
Pi = 3.1415926
'lt = length of trunk segment from axis
lt = 0.4045 * Cells(2, 5)
'mass = total body mass
mass = Cells(2, 6)
'mp = mass of trunk segment
mp = 0.5676 * mass
'a = acceleration
a = (-9.81)
'd = distance of COM from axis
d = 0.504 * lt
'angle offset = ao
ao = Cells(1, 3)

For I = 1 To n
    If Cells(I + 6, 8 - 4) > 90 Then
        Cells(I + 6, 8) = -(mp * a * d) * Cos((Cells(I + 6, 8 - 4) - (90 - ao)) * Pi / 180)
    Else
        Cells(I + 6, 8) = -(mp * a * d) * Cos((Cells(I + 6, 8 - 4) - (90 - ao)) * (-Pi / 180))
    End If
Next I

For I = 1 To n
    Cells(I + 6, 9) = Cells(I + 6, 8) + Cells(I + 6, 5)
Next I

End Sub
Sub torque2() ' trunk extension
n = Cells(4, 26)
Pi = 3.1415926
'lt = length of trunk segment from axis
lt = 0.4045 * Cells(2, 5)
'mass = total body mass
mass = Cells(2, 6)
'mp = mass of trunk segment
mp = 0.5676 * mass
'a = acceleration
a = (-9.81)
'd = distance of COM from axis
d = 0.504 * lt
'angle offset = ao
ao = Cells(1, 3)

For I = 1 To n
    If Cells(I + 6, 21 - 4) > 90 Then
        Cells(I + 6, 22) = -(mp * a * d) * Cos((Cells(I + 6, 22 - 4) - (90 - ao)) * Pi / 180)
    Else
        Cells(I + 6, 22) = -(mp * a * d) * Cos((Cells(I + 6, 22 - 4) - (90 - ao)) * (-Pi / 180))
    End If
Next I

For I = 1 To n
    Cells(I + 6, 23) = Cells(I + 6, 22) + Cells(I + 6, 19)
Next I
End Sub
Sub DFT()
' Flexion Phase
Const tpnts = 4000 'total data points
Const pi = 3.1415926
Const st = 2 'number of data points preceed the start
Const samp = 1000 'Sampling rate (Hz)
Dim dftpnts
Dftpnts = tpnts
ReDim s(dftpnts), c(dftpnts), dat1(dftpnts), Power(dftpnts)
For I = 1 To dftpnts
    dat1(I) = Cells(I + st, 2) 'dat1(I)
Next I

'DFT
w = (2 * pi) / samp
m = dftpnts / 2 + 1
For k = 1 To m
    k1w = (k - 1) * w
    For J = 1 To dftpnts
        alpha = k1w * (J - 1)
        s(k) = s(k) + dat1(J) * Sin(alpha)
        c(k) = c(k) + dat1(J) * Cos(alpha)
    Next J
    s(k) = s(k) + s(k)
    c(k) = c(k) + c(k)
    Power(k) = s(k) * s(k) + c(k) * c(k)
    Cells(k, 17) = k - 1
    Cells(k, 18) = Power(k) / 1000000
Next k

tt = 0
For k = 1 To Int(0.5 * samp)
    tt = tt + Cells(k, 18)
Next k

med = 0
For k = 1 To Int(0.5 * samp)
    med = med + Cells(k, 18)
    If med > tt Then Exit For
Next k

Cells(1, 19) = k - 1
tf = 0
For k = 1 To Int(0.5 * samp)
    tf = tf + k * Cells(k, 18)
Next k

Cells(2, 19) = tf / tt
End Sub
Sub DFT2()
'Description of the code
Const tpnts = 6000 'total data points
Const pi = 3.1415926
Const st = 6000 'number of data points proceed the start
Const samp = 1000 'Sampling rate (Hz)
Dim dftpnts
ReDim s(dftpnts), c(dftpnts), dat1(dftpnts), Power(dftpnts)
For I = 1 To dftpnts
    dat1(I) = Cells(I + st, 2) 'dat1(I)
Next I
'DFT
w = (2 * pi) / samp
m = dftpnts / 2 + 1
For k = 1 To m
    k1w = (k - 1) * w
    For J = 1 To dftpnts
        alpha = k1w * (J - 1)
        s(k) = s(k) + dat1(J) * Sin(alpha)
        c(k) = c(k) + dat1(J) * Cos(alpha)
    Next J
    s(k) = s(k) + s(k)
    c(k) = c(k) + c(k)
    Power(k) = s(k) * s(k) + c(k) * c(k)
    Cells(k, 20) = k - 1
    Cells(k, 21) = Power(k) / 1000000
Next k
End Sub
Sub DFilter() ' trunk flexion
For sn = 0 To 9
    sname = "0" + Trim(Str(sn + 1))
    Sheets(sname).Select
        filter1
        filter2
Next sn
End Sub

Sub filter1()
    For col = 4 To 4
        ' Following are the variables needed to be defined outside
        ' of the sub in order to get the sub "DFilter" to operate
        ' *************Next three lines might need to be modified**************
        ' Const DFNumpnt = 58 ' Number of points in the set of data being passed in
        DFNumpnt = 600
        Const DFcutoff = 1 ' Cutoff frequency for either a hi-pass or a low-pass
        Const DFSrate = 100 ' Sampling rate of the original data
        ' ***********************************************
        Const DFPi = 3.1415926
        Const DFfiltertype = "lp" ' can be either "lp" for low pass or "hp" for high pass
        Const DTypef = "Butterworth"
        Dim DFti ' time interval (period) of the original data, 1/sampling rate
        Dim DFpcut
        Dim DFWC
        Dim DFk1, DFk2, DFk3
        Dim DFA0, DFA1, DFA2
        Dim DBF1, DBf2
        Dim DFFiltroption As String
        ReDim DFdata(DFNumpnt) ' original data set need to be passed in to the sub
        ReDim DNewdata(DFNumpnt) ' Filtered data at the end of the sub
        ReDim DFTemp(1 To DFNumpnt + 4), DFprime(1 To DFNumpnt + 4) As Single
        ' *************Next three lines might need to be modified**************
        ' get the data from the spreadsheet
        For DFi = 1 To DFNumpnt
            ' For K = 1 To 16
                DFdata(DFi) = Cells(DFi + 6, col)
            ' Next K
        Next DFi
        ' Fourth order, zero lag filter
        ' correction to cutoff for high-pass filter
        DFFiltroption = DFfiltertype
        DFti = 1 / DFSrate
If DFfiltoption = "hp" Then
    DFpcut = (1 / (2 * DFti)) - DFcutoff
Else
    DFpcut = DFcutoff
End If
DFWC = Tan(DFPi * DFpcut * DFti)
'Wc need to be corrected for the dual pass
'Murphy and Robertson (1994),
'J. of Applied Biomechanics, 10:374-381
'And also, Robertson, Barden and Dowling
'NACOB II, 1992
If DFtypef = "Butterworth" Then
    DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
    DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DFtypef = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else
    DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
'correction to coefficients for high-pass filter
If DFfiltoption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
'Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) -
DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) -
DFdata(DFNumpnt - 2))
    For DFi = 1 To DFNumpnt
        DFTemp(DFi + 2) = DFdata(DFi)
    Next DFi
For DFi = 1 To DFNumpnt + 4
    DFprime(DFi) = DFTemp(DFi)
Next DFi
For DFi = 3 To DFNumpnt + 4

DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi - 1) + DFa2 * DFTemp(DFi - 2) + DFb1 * DFprime(DFi - 1) + DFb2 * DFprime(DFi - 2)
Next DFi
For DFi = 1 To DFNumpnt + 4
   DFTemp(DFi) = DFprime(DFi)
Next DFi
For DFi = DFNumpnt + 2 To 1 Step -1
   DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi + 1) + DFa2 * DFTemp(DFi + 2) + DFb1 * DFprime(DFi + 1) + DFb2 * DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
   DFnewdata(DFi) = DFprime(DFi + 2)
Next DFi
'*************Next three lines might need to be modified**************
'write the filtered data to the spreadsheet
For DFi = 1 To DFNumpnt
   'For K = 1 To 16
       Cells(DFi + 6, 7) = DFnewdata(DFi)
   'Next K

Next DFi
'******************************************************************************

Next col
For DFi = 1 To DFNumpnt
   Cells(DFi + 6, 29) = (DFi - 1) * (1 / 100)
Next DFi
End Sub
Sub filter2() 'trunk extension

For col = 18 To 18
    'Following are the variables needed to be defined outside
    'of the sub in order to get the sub "DFilter" to operate
    '************************Next three lines might need to be modified************************
    'Const DFNumpnt = 58 ' Number of points in the set of data being passed in
    DFNumpnt = 600
    Const DFcutoff = 1 ' Cutoff frequency for either a hi-pass or a low-pass
    Const DFSrate = 100 'Sampling rate of the original data
    '*******************************************************************************
    Const DFpi = 3.1415926
    Const DFfiltertype = "lp" ' can be either "lp" for low pass or "hp" for high pass
    Const DFtypef = "Butterworth"
    Dim DFti ' time interval (period) of the original data, 1/sampling rate
    Dim DFpcut
    Dim DFWC
    Dim DFk1, DFk2, DFk3
    Dim DFA0, DFA1, DFA2
    Dim DFb1, DFb2
    Dim DFFiltoption As String
    ReDim DFDdata(DFNumpnt) ' original data set need to be passed in to the sub
    ReDim DFDnewdata(DFNumpnt) ' Filtered data at the end of the sub
    ReDim DFTemp(1 To DFNumpnt + 4), DFDprime(1 To DFNumpnt + 4) As Single
    '************************Next three lines might need to be modified************************
    'get the data from the spreadsheet
    For DFi = 1 To DFNumpnt
        'For K = 1 To 16
        DFDdata(DFi) = Cells(DFi + 6, col)
        'Next K
        Next DFi
    Next DFi
    '*******************************************************************************
    'Fourth order, zero lag filter
    'correction to cutoff for high-pass filter
    DFFiltoption = DFFiltertype
    DFti = 1 / DFSrate
    If DFFiltoption = "hp" Then
        DFpcut = (1 / (2 * DFti)) - DFcutoff
    Else
        DFpcut = DFcutoff
    End If
    DFWC = Tan(DFPi * DFpcut * DFti)
    'Wc need to be corrected for the dual pass
    'Murphy and Robertson (1994),
    'J. of Applied Biomechanics, 10:374-381
    'And also, Robertson, Barden and Dowling
    'NACOB II, 1992
    If DFtypef = "Butterworth" Then
DFWC = DFWC / Sqr(Sqr(Sqr(2) - 1))
Else
    DFWC = DFWC / Sqr(Sqr(Sqr(2)) - 1)
End If
If DFtype = "Butterworth" Then
    DFk1 = Sqr(2) * DFWC
Else
    DFk1 = 2 * DFWC
End If
DFk2 = DFWC ^ 2
DFa0 = DFk2 / (1 + DFk1 + DFk2)
DFa1 = 2 * DFa0
DFa2 = DFa0
DFk3 = (2 * DFa0) / DFk2
DFb1 = (-2 * DFa0) + DFk3
DFb2 = 1 - (2 * DFa0) - DFk3
' correction to coefficients for high-pass filter
If DFfiloption = "hp" Then
    DFa1 = -DFa1
    DFb1 = -DFb1
End If
' Filter
DFTemp(1) = DFdata(1) + (DFdata(1) - DFdata(2))
DFTemp(2) = DFdata(1) + (DFdata(1) - DFdata(3))
DFTemp(DFNumpnt + 4) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFdata(DFNumpnt - 1))
DFTemp(DFNumpnt + 3) = DFdata(DFNumpnt) + (DFdata(DFNumpnt) - DFDa(DFNumpnt - 2))
For DFi = 1 To DFNumpnt
    DFTemp(DFi + 2) = DFdata(DFi)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFprime(DFi) = DFTemp(DFi)
Next DFi
For DFi = 3 To DFNumpnt + 4
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi - 1) + DFa2 * DFTemp(DFi - 2) + DFb1 * DFprime(DFi - 1) + DFb2 * DFprime(DFi - 2)
Next DFi
For DFi = 1 To DFNumpnt + 4
    DFTemp(DFi) = DFprime(DFi)
Next DFi
For DFi = DFNumpnt + 2 To 1 Step -1
    DFprime(DFi) = DFa0 * DFTemp(DFi) + DFa1 * DFTemp(DFi + 1) + DFa2 * DFTemp(DFi + 2) + DFb1 * DFprime(DFi + 1) + DFb2 * DFprime(DFi + 2)
Next DFi
For DFi = 1 To DFNumpnt
    DFnewdata(DFi) = DFprime(DFi + 2)
Next DFi
'Next three lines might need to be modified************
'write the filtered data to the spreadsheet
For DFi = 1 To DFNumpnt
    'For K = 1 To 16
    Cells(DFi + 6, 21) = DFnewdata(DFi)
    'Next K
    Next DFi

Next col
For DFi = 1 To DFNumpnt
    Cells(DFi + 6, 29) = (DFi - 1) * (1 / 100)
    Next DFi
End Sub
Sub energy() 'trunk flexion
For sn = 0 To 9
    sname = "0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    hysteresis1
    hysteresis2
Next sn
End Sub

Sub energy2() 'trunk extension
For sn = 0 To 9
    sname = "0" + Trim(Str(sn + 1))
    Sheets(sname).Select
    hysteresis3
    hysteresis4
Next sn
End Sub

Sub hysteresis1()
    n = Cells(4, 12)
    Pi = 3.1415926
    For I = 1 To n
        Cells(I + 6, 30) = Cells(I + 6, 7)
        Cells(I + 6, 31) = Cells(I + 6, 9)
    Next I
    For I = 1 To n
        If Cells(I + 6, 30) > 30 Then Cells(I + 6, 32) = ((Cells(I + 7, 30) - Cells(I + 6, 30)) * (Cells(I + 7, 31) - Cells(I + 6, 31)))
        If Cells(I + 6, 30) > 65 Then Cells(I + 6, 32) = 0
    Next I
End Sub
Sub hysteresis2()
  n = Cells(4, 12)
  Pi = 3.1415926

  For I = 1 To n
      Cells(I + 6, 35) = Cells(I + 6, 21)
      Cells(I + 6, 36) = Cells(I + 6, 23)
  Next I

  For I = 1 To n
      If Cells(I + 6, 35) > 30 Then Cells(I + 6, 37) = ((Cells(I + 7, 35) - Cells(I + 6, 35)) * (Cells(I + 7, 36) - Cells(I + 6, 36)))
        If Cells(I + 6, 35) > 65 Then Cells(I + 6, 37) = 0
      Next I
  End Sub

Sub hysteresis3() 'raw data
  n = Cells(4, 12)
  Pi = 3.1415926

  For I = 1 To n
      Cells(I + 6, 39) = Cells(I + 6, 7)
      Cells(I + 6, 40) = Cells(I + 6, 5)
  Next I

  For I = 1 To n
      Cells(I + 6, 41) = Abs((Cells(I + 7, 39) - Cells(I + 6, 39)) * (Cells(I + 7, 40) - Cells(I + 6, 40)))
        If Cells(I + 6, 39) < 0 Then Cells(I + 6, 41) = 0
      Next I
  End Sub

Sub hysteresis4() 'raw data
  n = Cells(4, 12)
  Pi = 3.1415926

  For I = 1 To n
      Cells(I + 6, 44) = Cells(I + 6, 21)
      Cells(I + 6, 45) = Cells(I + 6, 19)
  Next I

  For I = 1 To n
      Cells(I + 6, 46) = Abs((Cells(I + 7, 44) - Cells(I + 6, 44)) * (Cells(I + 7, 45) - Cells(I + 6, 45)))
        If Cells(I + 6, 44) < 0 Then Cells(I + 6, 46) = 0
      Next I
  End Sub
APPENDIX 4. LETTERS OF PERMISSION TO PUBLISH

December 1, 2005

Michael W. Olson
Southern Illinois University
Mailcode 4310
Carbondale, IL 62901

Our ref: OlsonThesisML12-05


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Date: March 10, 2005

Michael Olson
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Michael William Olson was born on 5 June 1974 in Janesville, Wisconsin. Michael was raised in Janesville and graduated from J. A. Craig High School in 1992. Following his high school graduation he attended the University of Wisconsin at Whitewater and graduated in 1997 with a Bachelor of Science degree in biology. Michael then attended the University of Wisconsin at La Crosse to pursue a Master of Science degree in exercise and sports science. During his time at La Crosse, he served as the graduate assistant for the men’s track and field team. After graduating from La Crosse, he worked as an assistant track coach for two years at Colby College in Waterville, Maine. Michael left Maine in 2001 to pursue his Doctor of Philosophy degree in kinesiology at Louisiana State University. He was the coordinator of the Biomechanics/Motor Behavior Lab during three years of his graduate studies in the Kinesiology Department. After completing his degree at Louisiana State University, Michael intends to continue his teaching and research responsibilities as an Assistant Professor in the Physical Education/Kinesiology Department at Southern Illinois University in Carbondale, Illinois.