PROLONGED LUMBAR TRUNK FLEXION DISTURBS PARASPINAL REFLEX BEHAVIOR

ELLEN ROGERS

Thesis presented to

The faculty of the Virginia Polytechnic Institute and State University

in partial fulfillment of the requirements for the degree of

Master of Science

in

Mechanical Engineering

Kevin P. Granata, Ph.D., Chair

Michael Madigan, Ph.D.

Maury Nussbaum, Ph.D.

February 16, 2005

Blacksburg, Virginia

Keywords: Low-Back, Reflex, Lumbar flexion, Stability

PROLONGED LUMBAR TRUNK FLEXION DISTURBS PARASPINAL REFLEX BEHAVIOR

Ellen Rogers

(ABSTRACT)

The neuromuscular response to prolonged lumbar flexion has recently been extensively studied in felines but has not been examined in humans. Animal studies suggest that prolonged lumbar flexion disturbs neuromuscular control of paraspinal muscles. This disturbance was linked to creep deformation of passive spinal tissues. Past research indicates that disturbance of paraspinal reflexes may limit spinal stability. The current study aimed to examine this behavior in humans. We hypothesized that prolonged lumbar flexion will disturb paraspinal reflex behavior in human subjects.

Reflex behavior was quantified following a fifteen minute period of static flexion. There was a trend suggesting an increase in reflex magnitude after flexion ($p = 0.055$). This trend was only significant in female subjects ($p < 0.003$). Increased reflex following flexion was associated with a transient period of EMG hyperexcitability similar to felines.

A second study was performed to quantify reflex behavior and creep deformation during flexion and recovery. Results indicated that creep occurred during prolonged flexion ($p < 0.001$). Reflexes were inhibited following flexion ($p < 0.03$). Both creep deformation and paraspinal reflex ($p > 0.05$) failed to exhibit significant recovery during the length of the test.

Inhibited paraspinal reflexes may contribute to spinal instability and risk of low back pain for workers using flexed postures, due to the inability of the neuromuscular system to coordinate an appropriate muscle response following an unexpected loading event. Future studies must examine appropriate work/rest intervals for workers using flexed postures to limit reflex disturbance from prolonged ligament strain.

ACKNOWLEDGEMENTS

The research described here was made possible through contributions from lab mates, family members and friends.

Dr. Granata

For teaching me, challenging me and having confidence in me. Thank you for being an excellent advisor.

Students of the Musculoskeletal Biomechanics Lab

Greg Slota, without whom nothing in this lab would be accomplished

Kevin Moorhouse, for development of analysis techniques instrumental to this research.

My family

My parents for their support through this process and throughout my life. You inspired me to go on for higher education.

Carolyn and Greg, for their support and friendship.

Patrick

I could not have done this without you.

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1 – INTRODUCTION

Trunk flexion postures are associated with increased risk of low back pain (LBP) (Kumar et al., 2001). Occupations requiring prolonged static or cyclic trunk flexion have been reported with high incidence rates of LBP (NIOSH, 1999; US Department of Labor, 1995). Recent animal studies have shown that prolonged exposure to lumbar flexion may contribute to disturbance of neuromuscular control (Solomonow et al, 1999), which may increase LBP risk.

Disturbance to neuromuscular control will limit spinal stability and therefore may contribute to LBP. Spinal stability may be defined as the ability of the trunk to return to equilibrium following a small perturbation. Spinal stability is maintained by three main mechanisms; passive tissues and bone provide intrinsic or passive stability, spinal muscles provide dynamic or active stability, and the neuromuscular system evaluates the requirements for stability and coordinates the muscle response (Panjabi, 2003). Impairment of one of these mechanisms, in particular poor muscular reflex response may cause instability and back pain. LBP patients have been shown to exhibit poor neuromuscular response and postural control compared to healthy subjects (Luoto et al., 1996; Radebold et al., 2000).

Lumbar flexion disturbs neuromuscular control in animal models (Solomonow et al., 1999). Inhibition of paraspinal reflexes in felines has been linked to elongation developed in passive tissues during flexion (Solomonow et al., 1999). During prolonged trunk flexion ligaments exhibit viscoelastic behavior and elongate over time (McGill and Brown, 1992). This behavior is known as creep deformation. Creep deformation has been well documented in humans and felines (McGill and Brown, 1992; Solomonow et al., 2003). Reflex inhibition following lumbar flexion in animals is hypothesized to result from mechanoreceptors in passive tissues becoming desensitized during tissue creep. Mechanoreceptors contribute to the initiation of muscle reflexes. Desensitization may therefore cause inhibition of muscle control and stability. Recovery of spinal reflexes in felines to normal levels was shown to take much longer than the time required to produce ligament elongation (Solomonow et al., 2003) indicating that prolonged trunk flexion may affect the stability of the spine for long periods of time even after normal posture is resumed.

Flexion induced disturbance to neuromuscular control has not been documented in humans. It is likely that human paraspinal reflexes will exhibit similar behavior as in felines, though this has yet to be shown. If humans demonstrate similar reflex disturbance following prolonged flexion there are potentially important implications for occupations requiring this type of work. The purpose of this study was to quantify changes in paraspinal reflex due to prolonged trunk flexion in humans. Trunk reflexes were measured using systems identification techniques that have been established in other joints (Zhang et al., 1999; Mirbagheri et al., 2000).

Other factors may contribute to changes in paraspinal reflex dynamics, such as gender and muscle pre-activation. Studies have indicated that muscle pre-activation causes increased muscle stiffness (Gardner-Morse and Stokes, 2001). Higher muscle stiffness may reduce the magnitude of reflexive muscle response (Stokes et al., 2000). Interactions between gender and prolonged flexion may influence changes in reflex behavior because females have been shown to exhibit more creep deformation than males over the same time period (Solomonow et al., 2003).

SPECIFIC AIMS

1. Measure changes in paraspinal reflex gain and delay due to prolonged trunk flexion.
2. Measure paraspinal reflex gain and delay as a function of gender and preload.
3. Evaluate passive tissue creep and paraspinal reflex behavior following recovery from prolonged ligament strain.

HYPOTHESES

1. Prolonged trunk flexion will cause inhibition of paraspinal reflexes.
2. A recovery period equal to that of the flexion loading will be insufficient to restore reflexes to original levels.

CHAPTER 2 – BACKGROUND

2.1 Effects of prolonged trunk flexion

Flexion-relaxation (FR) is a well documented phenomenon (Floyd and Silver, 1951; Kippers and Parker, 1984; McGill and Brown, 1992) that occurs during the motion to extreme trunk flexion from an upright position. At a trunk flexion angle of approximately 80º superficial erector spinae muscles suddenly deactivate, indicated by an abrupt onset of electrical silence (Kippers and Parker, 1984). During motion from full flexion to an upright posture, erector spinae muscles abruptly re-recruit at a similar angle of trunk flexion. FR has also been shown to occur during lifting (Dolan et al., 1994) and slumped sitting (Callaghan and Dunk, 2002). The angle at which the erector spinae deactivate can be changed with added external mass (Kippers and Parker, 1984; Gupta, 2001) and with lifting rate (Sarti et al., 2001).

One theory for the origin of flexion-relaxation that has been widely accepted since the discovery of this phenomenon is that the erector spinae muscles deactivate as a result of load sharing between muscles and passive tissues (Kippers and Parker, 1984). As the trunk approaches full flexion, the external moment becomes fully supported by passive tissues which have been brought into tension during flexion. Passive support may be contributed by ligaments, intervertebral discs, lumbodorsal fascia, and non-contractile tissue within erector spinae muscle (Dolan et al., 1994). Passive tissues have viscoelastic properties; as tissues are stretched they can support more load. This theory is supported in part by studies that show a change in FR onset angle with added load. Added external load changes the external moment and tissues must be stretched farther to replace muscle activity in supporting the greater external moment (Kippers and Parker, 1984).

Opposing evidence is given by Dolan et al. (1994) who separated the extensor moment during lifting into active or contractile components, and passive components which include the contributions from passive muscle tissue, ligaments, disc and fascia. Findings indicate that while the passive extensor moment increases with flexion angle, the passive contribution to the total extensor moment decreased with flexion angle. This seems to contradict the theory that passive support entirely replaces muscle activity in load bearing. One possible explanation for this result

given by Dolan et al. was that other muscles began to contribute more to the extensor moment at higher flexion angles. Indeed, Andersson et al. (1996) have shown that while superficial erector spinae muscle deactivate during extreme trunk flexion, deeper muscles, such as the quadratus lumborum and deep erector spinae become more active with increasing trunk angle. This suggests another explanation that FR is caused by a redistribution of muscle recruitment to deeper muscles that occurs during flexion. FR is likely caused in part by a combination of both increased passive support and deeper lumbar muscle activity.

Sustained or repetitive lumbar flexion can cause creep deformation in passive spinal tissues. Creep deformation occurs when there is an increase in lumbar flexion angle at a constant bending moment. Ligaments exhibit viscoelastic behavior and elongate over time. Creep is typically observed during prolonged stooping (i.e. gardening or assembly line working). Viscoelastic tissue creep has been shown in both humans (McGill and Brown, 1992) and animals (Solomonow et al., 2003). In humans, creep is observed during full flexion with an increase in trunk or lumbar flexion angle over time, which can easily be monitored with surface position markers placed on the spine. More invasive measures can be used on animals; specific spinal ligaments in felines have been isolated and stretched (Jackson et al., Spine 2001; Solomonow et al., 2003). Laboratory studies have shown that creep occurs exponentially (McGill and Brown, 1992). Furthermore following sustained flexion ligaments require time to return to their original lengths. The exponential time constant during recovery is greater than during flexion (McGill and Brown, 1992), indicating that passive tissues require longer to recover than to produce creep deformation.

Changes in reflex behavior in felines have been linked to tissue laxity and creep developed during spinal ligament stretch (Solomonow et al., 1999). It is hypothesized that laxity in passive spinal tissues causes desensitization of mechanoreceptors responsible for initiating reflexive muscular action. Therefore during prolonged lumbar flexion reflexes diminish and eventually are eliminated. Reduced or eliminated reflexes may have a detrimental effect on spinal stability; this issue will be discussed further in a following section.

Solomonow et al. have established a five phase ‘neurological disorder’ resulting from prolonged static or cyclic ligament elongation in felines (2003). During flexion, lumbar reflexes

and myoelectric activity decrease, presumably due to the desensitization of mechanoreceptors associated with creep deformation of passive spinal tissues. Superimposed on this trend are unpredictable muscle spasms. Static or cyclic load applied to ligaments causes microdamage in the tissue as well as changes to its mechanical properties (Carpenter et al., 1998; Soslowsky et al, 2000). Therefore it is suggested that these spasms occurred as a result of microdamage to the passive tissues of the lumbar spine (Solomonow et al., 2003). Muscle activity was recruited spasmodically to limit additional damage and pain in the viscoelastic tissues.

The five phase 'neurologic disorder' continued for an extended time following flexion. Recovery was characterized by three phases. Immediately following the stretch period, initial reflex hyper-excitability occurred, wherein lumbar myoelectric response became elevated relative to the inhibited response demonstrated during flexion. It was suggested that the increase in muscle activity was a feedback response designed to protect the viscoelastic tissues from further damage by limiting movement or compensating for lost tension in ligaments (Solomonow et al., 2003). This period of hyper-excitability was transient; muscle activity returned to inhibited levels where it exhibited a slow exponential recovery over a period of hours. The final phase of the 'neurological disorder' took place several hours following flexion. Delayed hyper-excitability occurred, indicating the possibility of inflammation in the ligaments (Solomonow et al., 2003).

No research on paraspinal reflexes during or after prolonged flexion has been conducted in humans. As viscoelastic tissue creep occurred in both humans and felines, it may be suggested that reflex behavior in humans will follow a pattern similar to that demonstrated in felines. Such a pattern of reflex behavior in humans could inhibit spinal stability and contribute to high levels of LBP reported in occupations requiring prolonged flexion. Insight into this phenomenon could be useful in LBP prevention and rehabilitation. To study this behavior in humans it is necessary to measure the myoelectric reflex response using electromyography.

2.2 Electromyography

When a muscle is stimulated by a nerve, a depolarization of the muscle membrane occurs and propagates along the length of the muscle fiber. This depolarization is called a muscle fiber

action potential. The superimposition of several muscle fiber action potentials constitutes a more complex motor unit action potential. The change in electric potential in or around the surrounding muscle due to depolarization of the cell membrane can be measured using differential electrodes parallel to the length of the fiber. This measurement is known as electromyography (EMG). Single muscle fiber activity can be recorded with a needle or fine wire inside the muscle. Surface EMG electrodes record the sum of motor unit potentials within the vicinity of the electrode and are most useful for recording superficial muscles and for non-invasive recording of overall recruitment patterns (Chaffin et al., 1999). Surface EMG appears qualitatively random due to the summation of many motor unit potentials over time. Appropriate electrode placement and proper processing of EMG is necessary for interpretation of myoelectric activity.

2.2.1 EMG Placement

To study the lower back, EMG should be recorded on the major muscle groups of the lumbar torso. Conventionally recorded muscle groups include the left and right erector spinae (ES), internal obliques (IO), rectus abdominus (RA) and external obliques (EO) (Granata et al., 1995). ES and IO are the main muscle groups responsible for generating extension moments; while RA and EO are the main muscle groups responsible for generating flexion moments. Specific placement of the electrodes on these muscles are as follows (Marras and Mirka, 1992): electrodes for the RA are placed 3 cm lateral and 2 cm superior to the umbilicus; ES 4 cm lateral to the L3 spinous process; posterior IO 8 cm lateral to the midline within the lumbar triangle at a 45°; and EO 10 cm lateral to the umbilicus with an orientation of 45° to vertical.

To study lumbar reflex behavior recording of myoelectric activity is required in the extension muscles, i.e. ES and IO. However the abdominal muscles should be recorded as well due to the phenomenon of co-contraction. Co-contraction occurs when both agonist muscles, which are responsible for moving a specific body segment, and antagonist muscles, which tend to oppose the desired motion, are used simultaneously. Co-contraction is a typical occurrence during human body movement (Chaffin et al., 1999), as well as during trunk movement (Granata and Lee, 2005). Therefore it is important to examine abdominal muscle recruitment even when the true interest lies in lumbar paraspinal muscle activity.

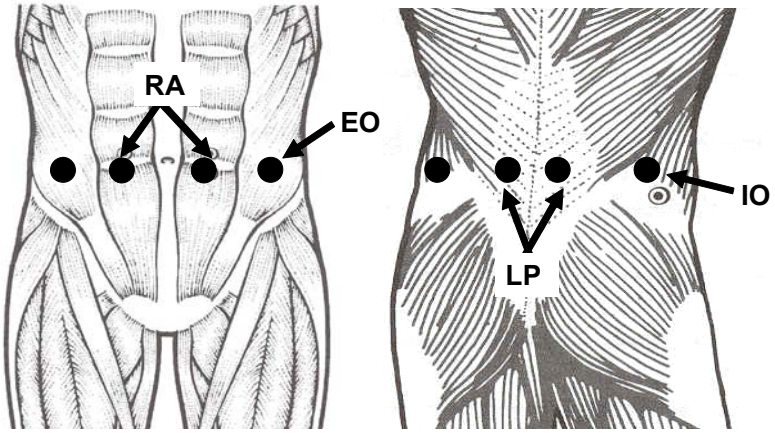

Figure 2.1. Schematic representation of the EMG placement of the four major trunk muscle groups (left and right rectus abdominus, external obliques, lumbar paraspinals, and internal obliques).

2.2.2 EMG Processing

Due to its seemingly random appearance, raw EMG must be appropriately processed in order to accurately represent muscle activation. The first step in EMG processing is to band-pass filter the signal, typically between 20 and 500 Hz (DeLuca, 1997). This removes low frequency signal below 20 Hz which can be affected by motion artifact, and high frequency noise. To prevent aliasing signals should therefore be sampled at 1000 Hz or greater. Band-stop filters can be used at particularly noisy frequencies, such as 60 Hz, to eliminate electromagnetic noise. To translate EMG into a single polarity, EMG is typically full-wave rectified. Subsequent low pass filtering serves to determine a linear envelope of the time history of the recorded activity (Potvin and Brown, 2004). Rectified and smoothed EMG signals are traditionally normalized to the maximum voluntary contraction (MVC) of the recorded muscle (DeLuca, 1997). This allows muscle forces to be expressed as a percentage of the theoretical force generating capacity. For this study, MVCs are isometric contractions in back extension, flexion, combined extension and torsion, and combined flexion and torsion. MVCs were recorded immediately prior to experimentation. EMG data recorded and processed in this fashion may subsequently be used to study reflex dynamics.

2.3 Quantification of Reflex Dynamics

2.3.1 Basic Reflex Dynamics

Spinal stability may be defined as the ability of the system to return to equilibrium following a small perturbation. Spinal stability is maintained by three main mechanisms; passive tissues and bone provide intrinsic or passive stability, spinal muscles provide dynamic or active stability, and the neuromuscular system evaluates the requirements for stability and coordinates the muscle response (Panjabi, 2003). Spinal musculature has been shown to be an intricate part in maintaining stability of the spine (Panjabi, 1992; McGill et al., 1986; Gardner-Morse and Stokes, 1998; Granata and Marras, 1995; Potvin et al., 1991) and in upright postures passive tissue contributions may be relatively small in comparison. To withstand perturbations, the trunk mainly relies on trunk stiffness (i.e. pre-activated trunk muscles) and reflexive muscular response (Stokes et al., 2000). Studies have indicated that applying a preload to the trunk increases muscular activation, making the trunk stiffer (Gardner-Morse and Stokes, 2001). Furthermore at high preloads reflexive muscular responses occur less frequently presumably due to the increased stiffness associated with high levels of muscle activation (Stokes et al., 2000). These studies imply that at low levels of trunk muscle activation reflex response plays an important role in returning the spinal system to equilibrium after a disturbance.

Maintaining stability requires a highly coordinated and dynamic effort of many muscle groups. This dynamic coordination is achieved through the neuromuscular system. Motor control response depends primarily on proprioceptive feedback from mechanoreceptors in the muscles and passive tissues. Muscle spindle receptors assess changes in muscle length and velocity and have direct reflex connections to the muscles controlling the movement of the joint (Kearney and Hunter, 1990; Simon, 1994). Passive tissues have similar pathways to muscle activation; Golgi, Pacinian and Ruffian mechanoreceptors in spinal ligaments assess changes in ligament length, stress, strain, or muscle force, and transmit signals to the central nervous system for the activation of musculature (Solomonow et al., 1998). When changes in spinal balance affect the stability of the spine the neuromuscular system is able to activate necessary muscles to restore and maintain stability (Panjabi 1992). Therefore a disturbance of the motor control system can impair reflexive muscular action responsible for restoring and maintaining stability. In fact, LBP patients have been shown to exhibit abnormal trunk muscle recruitment patterns

compared to healthy subjects in order to compensate for a loss of spinal stability (Van Dieen et al., 2003).

There is an inherent delay between position or force disturbance sensed by mechanoreceptors and reflex initiation. This delay, or latency, accounts for conduction time from the spindle and mechanoreceptors, through a multi-synaptic reflex loop and back to the muscle as well as propagation time of the muscle action potentials (Matthews, 1991). Furthermore the human stretch reflex can be separated into at least two components occurring at different times, short latency and long latency (Matthews, 1991). The short latency reflex is a monosynaptic spinal stretch reflex which occurs between 10-30 msec. The long latency reflex has a more complex and less understood pathway and generally occurs between 60-100 msec. A study of the trunk indicates that paraspinal reflexes occur with a delay of approximately 70 msec (Granata et al., 2004).

2.3.2 Spinal Reflex Elicitation using Pseudorandom Inputs

Reflex dynamics have been measured by relating changes in EMG activity to input position or force perturbations (Hunter and Kearney, 1983; Zhang et al., 1999; Granata et al., 2004). Sinusoids, sudden impacts (i.e. tendon tap) and stochastic inputs have been used to elicit reflexes. There may be advantages and disadvantages to each type of input. Sinusoidal inputs can be predictable; subjects may generate voluntary responses that will influence joint dynamics. Sinusoidal inputs may also inhibit neuromuscular control, similar to the effects of vibration (Hebert and Boucher, 1998). Sudden impact or quick-release inputs are an improvement over sinusoidal impulses in that the time duration is sufficiently short to eliminate voluntary response to a sustained input. However, since equilibrium before a sudden release is different than after the release, it is difficult to determine the effect of the disturbance on muscle activity when muscle activity at two different equilibrium conditions are compared. Linear estimates of joint dynamics are applicable only for small displacements about a prescribed operating point, i.e. equilibrium must be similar before and after the perturbation (Moorhouse and Granata, 2005). Furthermore responses to sudden impact inputs may be less repeatable than to other inputs.

Published studies demonstrate that improved measurement of joint dynamics are possible with high-bandwidth, pseudo-random binary (PRB) perturbation sequences about a baseline bias torque. Pseudorandom inputs are unpredictable and the results are repeatable (Kearney and Hunter, 1990). As reflex may be a function of joint velocity as well as change in joint position, use of position input may be more accurate than force input because nonlinearities in EMG response can be accounted for using position input. In the ankle, nonlinear analyses have accounted for approximately 70% of the variance in EMG signals in response to position PRB inputs, while linear analyses of force PRB inputs have accounted for up to 60% of variance (Hunter and Kearney, 1983). In terms of experimental methods, position input may be more difficult to implement because actuators used must have a higher dynamic response and larger force generating capacity to provide accurate position control (Kearney and Hunter, 1990). In this study, PRB force input was used to elicit reflex behavior in the paraspinal muscles. Future studies should use position control to examine nonlinearities in reflex behavior.

2.3.3 System Identification

Reflex dynamics in response to pseudorandom inputs have been quantified using linear systems identification techniques (Zhang et al., 1999; Mirbagheri et al., 2000). Using these techniques, it is necessary to model EMG response as a linear time invariant (LTI) system. A linear system has the useful properties of scalability and additivity. A system is scalable if multiplying the input by a constant results in the original output multiplied by the same constant. A system is additive (or superimposable) when input $x_1(t)$ yields output $y_1(t)$, input $x_2(t)$ yields output $y_2(t)$, and the input of the sum of $x_1(t) + x_2(t)$ yields an output of the sum of $y_1(t) + y_2(t)$. A linear system is time-invariant if a time shift of the input results in the same time shift in the output.

2.3.4 Convolution

Using the properties of LTI systems and the mathematical process of convolution, a LTI system can be fully described by its response to a delta function, also known as its impulse response function (IRF). The output y(t) is described by the impulse response function h(t) of the system convolved with (notated *) the applied input signal, x(t)

$$y(t) = \int_{\infty}^{\infty} h(t-\tau)x(\tau)d\tau = h(t) * x(t) \tag{2.1}$$

A LTI system can be described in its entirety by its IRF because once h(t) is known, the output of the system can be determined for any input. Using this technique an IRF can be calculated from force or position input and rectified EMG output.

2.3.5 Correlation Function Analysis

To study reflex dynamics using this technique, equation 2.1 must be represented in terms of correlation functions. This allows for simple matrix calculations of IRFs using complicated inputs. First the input and output of equation 2.1 can be shifted by r, using the time-invariance property (equation 2.2).

$$y(t+r) = \int_{\infty}^{\infty} h(t-\tau)x(\tau+r)d\tau \tag{2.2}$$

Next both sides are multiplied by x(r) and integrated over r.

$$x(r)y(t+r) = x(r)\int_{\infty}^{\infty} h(t-\tau)x(\tau+r)d\tau \tag{2.3}$$

$$\int_{-\infty}^{\infty} x(r)y(t+r)dr = \int_{-\infty}^{\infty} x(r)\int_{\infty}^{\infty} h(t-\tau)x(\tau+r)d\tau dr = \int_{-\infty}^{\infty}\int_{-\infty}^{\infty} h(t-\tau)x(r)x(\tau+r)d\tau dr \tag{2.4}$$

The integrals on the right side can be written as a convolution of the impulse response function and another integral (equation 2.5).

$$\int_{-\infty}^{\infty} x(r)y(t+r)dr = \int_{-\infty}^{\infty} h(t-\tau)\left(\int_{-\infty}^{\infty} x(r)x(\tau+r)d\tau\right)dr \tag{2.5}$$

Using the definition of correlation functions (equations 2.6 a,b), where C_{xy} is the cross-correlation function between input and output and C_{xx} is the auto-correlation function of the input, equation 2.5 can be simplified into equation 2.7.

$$C_{xy} = \int_{-\infty}^{\infty} x(\tau)y(t+\tau)d\tau \tag{2.6a}$$

$$C_{xx} = \int_{-\infty}^{\infty} x(\tau)x(t+\tau)d\tau \tag{2.6b}$$

$$C_{xy}(t) = \int_{-\infty}^{\infty} h(t-\tau) C_{xx}(\tau) d\tau \tag{2.7}$$

In discrete time equation 2.7 can be represented as equation 2.8.

$$C_{xy}(k) = \Delta T \sum_{i=-\infty}^{\infty} h(k-i) C_{xx}(i) \tag{2.8}$$

Equation 2.8 can also be represented with matrices and can be simply solved.

$$\begin{aligned} C_{xy} &= C_{xx} H \, \Delta T \\ H &= C_{xx}^{-1} C_{xy} \Delta T \end{aligned} \tag{2.9}$$

where:

C_{xy} is a vector whose i^{th} element is $C_{xy}(i)$

C_{xx} is a square matrix whose $(i, j)^{th}$ element is $C_{xx}(i-j)$

H is a vector whose i^{th} element is h(i)

For correlation analysis the mean of the signal should be zero in order to ensure accuracy of correlation function estimations, so both the applied input and the rectified EMG are demeaned (Bendat and Piersol, 2000). Published techniques illustrate that adding white noise to the input signal reduces the effect of system noise by decorrelating the data sampling artifact from the input signal (Ljung and Ljung, 1998). The amount of white noise added is found through trial and error. Too much added white noise can distort the signal while too little will be unable to reduce the effect of system noise.

2.3.6 IRF Quality

Following correlation analysis, the quality of the calculated IRF response can be determined. To measure the quality of the IRF representation of the reflex response, the computed IRF can be convolved with the original input sequence to produce an estimate of the EMG signal.

$$EMG_{predicted} = IRF * F(t) \tag{2.10}$$

Percent Variance Accounted For (VAF) is the difference between the physiologic signal and the estimated signal given in equation 2.11 (Hunter and Kearney, 1983).

$$VAF = 100\left(1 - \frac{\sum\left(EMG_{measured} - EMG_{predicted}\right)^2}{EMG_{measured}^{\ 2}}\right) \tag{2.11}$$

VAF equal to 100% indicates that the IRF predicts exactly the measured EMG signal from the input perturbations. These analyses techniques have been performed on the knee joint (Zhang et al., 1999) and the ankle joint, yielding VAF values between 50-60% (Hunter and Kearney, 1983). A much-simplified impulse response based approach was used by Granata et al. (2004) to quantify trunk reflexes in response to a sudden impact perturbation; however no estimate of VAF was determined. No other known research has used this approach to quantify trunk reflexes to PRB force input.

2.3.7 Quantification of IRF

Reflexes were quantified based on the peak amplitude of the transfer function between EMG response and force disturbance. Reflex gain, G_R, is determined from the peak amplitude of the IRF (Figure 2.2). Reflex gain represents the magnitude of muscle response immediately following a disturbance. Large values of G_R indicate a large reflex response; small values for G_R indicate minimal muscle activity following the disturbance. A reflex response was defined as a peak occurring two standard deviations or more above the mean IRF signal. This threshold was determined through visual inspection of pilot data. Reflex latency, or delay, is measured as the time at which the peak amplitude occurred. After 120 ms, any response is considered to be arbitrarily voluntary and therefore not quantified as a reflex (Matthews, 1991).

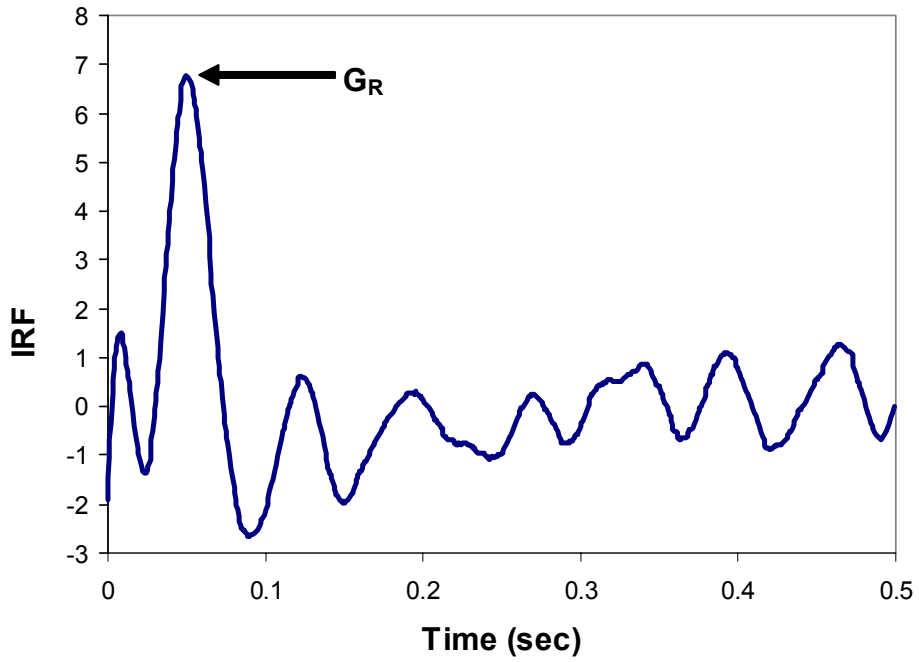

Figure 2.2. Visual depiction of impulse response function (IRF) relating EMG response to applied force perturbations. The peak amplitude of the IRF is referred to as reflex gain, G_R.

2.4 References

Andersson E.A., Oddsson L.I.E., Grundstrom H., Nilsson J., Thorensson A. EMG activities of the quadratus lumborum and reector spinae muscles during flexion-relaxation and other motor tasks. Clin.Biomech. 1996;11:392-400.

Bendat J.S., Piersol A.G. Random data: analysis and measurement procedures. 3 ed. New York: John Wiley & Sons, 2000.

Callaghan J., Dunk N. Examination of the flexion-relaxation phenomenon in erector spinae muscles during short duration slumped sitting. Clinical Biomechanics. 2002;17:353-360.

Carpenter J., Flangan C., Thomopoulos S., Yian E., Soslowsky L. The effect of overuse combined with intrinsic and extrinsic alterations in an animal model of rotator cuff tendonitis. Am. J. Sports Medicine. 1998;26:801-7.

Chaffin D.B., Andersson G.B.J., Martin B.J. (1999) Occupational Biomechanics, John Wiley and Sons, New York, NY.

DeLuca C. The use of surface electromyography in biomechanics. J Applied Biomechanics. 1997;13:135-163.

Dolan P., Earley M., Adams M.A. Bending and compressive stresses acting on the lumbar spine during lifting activities. J.Biomechanics. 1994;27:1237-1248.

Floyd W.F., Silver P.H.S. The function of the erector spinae muscles in certain movements and postures in man. J Physiology. 1955;28:199-210.

Gardner-Morse M.G., Stokes I. A. F. The effects of abdominal muscle coactivation on lumbar spine stability. Spine. 1998;23:86-92.

Gardner-Morse M.G., Stokes I.A.F. Trunk stiffness increases with steady-state effort. Journal of Biomechanics. 2001;34:457-63.

Granata K.P., Marras W.S. An EMG assisted model of biomechanical trunk loading during free-dynamic lifting. J.Biomechanics. 1995;28:1309-1317.

Granata K.P., Marras WS, Fathallah FA. A method for measuring external trunk loads during dynamic lifting exertions. J.Biomechanics. 1995;29:1219-22.

Granata K.P., Slota G., Bennett B.C. Paraspinal muscle reflex dynamics. J Biomech. 2004;37:241-7.

Granata K.P., Lee P. Co-contraction recruitment and spinal load during isometric trunk flexion. J Biomech. (In review).

Gupta A. Analyses of myoelectrical silence of erectors spinae. J Biomech. 2001;34:491-6.

Hebert J., Boucher J.P. Effect of manual segmental vibration on neuromuscular excitability. J Manipulative Physiol Ther. 1998; 21:528-33.

Hunter I.W., Kearney R.E. Two-sided linear filter identification. Med.Biol.Eng Comput. 1883;21:203-209.

Jackson M., Solomonow M., Zhou B., Baratta R. V., Harras M. Multifidus EMG and tension-relaxation recovery after prolonged static lumbar flexion. Spine. 2001;26:715-723.

Kearney R.E., Hunter I.W. System Identification of Human Joint Dynamics, Crit.Rev.Biomed.Eng. 1990;18:55-87.

Kippers V., Parker A.W. Posture related to myoelectric silence of erectores spinae during trunk flexion. Spine. 1984;9:740-745.

Kumar S. Theories of musculoskeletal injury causation. Ergonomics. 2001;44:17-47.

Ljung L., Ljung E.J. (1998) System Identification: Theory for the User, 2nd ed, Prentice Hall, Upper Saddle River, NJ.

Luoto S., Taimela S., Hurri A., Aalto H., Pyykko I., Alaranta H. Psychomotor speed and postural control in chronic low back pain patients: A controlled follow-up study. Spine. 1996;21:2621-2627.

Marras W.S., Mirka G.A. 1992, A comprehensive evaluation of trunk response to asymmetric trunk motion, Spine, 17, 318-326.

Matthews, P.B. The human stretch reflex and the motor cortex, Trends Neurosci. 1991;14:87-91.

McGill S.M., Norman R.W. Partitioning of the L4-5 dynamic moment into disc, ligamentous and muscular components during lifting. Spine. 1986; 11:666-678.

McGill S.M., Brown S. Creep response of the lumbar spine to prolonged full flexion, Clin Biomech. 1992;7:43-46.

Mirbagheri M.M., Barbeau H., Kearney, R.E. Intrinsic and reflex contributions to human ankle stiffness: Variation with activation level and position. Exp.Brain Res. 2000;135:423-436.

Moorhouse K.M., Granata K.P. Trunk stiffness and dynamics during active extension exertions. In: J.Biomechanics. (In Press).

Panjabi M.M. The stabilizing system of the spine. Part I Function, dysfunction, adaptation and enhancement. J.Spinal Disorders 1992;5:383-9.

Panjabi M.M. Clinical spinal instability and low back pain. J Electr.Kines. 2003;13:371-9.

Potvin J.R., McGill S.M., Norman R.W. Trunk muscle and lumbar ligament contributions to dynamic lifts with varying degrees of trunk flexion. Spine. 1991; 16:1099-107.

Potvin J.R., Brown A.H.M. Less is more: high pass filtering, to remove up to 99% of the surface EMG signal power, improves EMG-based biceps brachii muscle force estimates. J. Electr. & Kines. 2004;14:389-399.

Radebold A., Cholewicki, J., Panjabi, M.M., Patel, T.C. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine. 2000;25:947-954.

Sarti M., Lison J., Monfort M. Response of the flexion-relaxation relative to lumbar motion, load and speed. Spine. 2001;26:E421-6.

Simon S.R. (1994) Orthopaedic Basic Science, Am.Acad.Orthop.Surg., Rosemont, IL.

Solomonow M., Zhou B., Harras M., Lu Y., Baratta R.V. The ligamento-muscular stabilizing system of the spine. Spine. 1998;23:2552-2562.

Solomonow M., Zhou B., Baratta R.V., Lu Y., Harras M. Biomechanics of increased exposure to lumbar injury caused by cyclic loading: Part 1. Loss of reflexive muscular stabilization. Spine. 1999;24:2426-2434.

Solomonow M., Baratta R.V., Zhou B.H., Burger E., Zieske A., Gedalia A. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. J Electromyogr.Kinesiol. 2003;13:381-396.

Stokes I.A.F., Gardner-Morse M.G., Henry S.M., Badger G.J. Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature. Spine. 2000;25:1957-1964.

Survey of Occupational Injuries and Illnesses, National Institute of Occupational Safety and Health. 1999, Washington, DC.

Soslowsky L., Thomopoulos S., Tun S., Flangan C., Keefer C., Mastaw J., Carpenter J. Overuse activity injury in supraspinatus tendon in an animal model. Shoulder Elbow Surg. 2000;9:79-84.

Van Dieen J., Cholewicki J., Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine. 2003;28:834-841.

Workplace Injuries and Illnesses. Bureau of Labor Statistics, US Dept of Labor, Report USDL-95-509, 1995, Washington, DC.

Zhang L.Q., Rymer W.Z., Sliwa J.A. System identification of tendon reflex dynamics. IEEE Trans.Rehabil.Eng. 1999;7:193-203.

CHAPTER 3

EFFECTS OF STATIC FLEXION-RELAXATION ON PARASPINAL REFLEX BEHAVIOR

Published in: *Clinical Biomechanics* January 2005

Kevin P. Granata, Ph.D.

Ellen Rogers

Kevin Moorhouse, M.S.

Musculoskeletal Biomechanics Laboratories
Department of Engineering Science & Mechanics
School of Biomedical Engineering and Science
Virginia Polytechnic Institute & State University
219 Norris Hall (0219)
Blacksburg, VA 24061, *USA*

Abstract

Background. Static trunk flexion working postures and disturbed trunk muscle reflexes are related to increased risk of low back pain. Animal studies conclude these factors may be related; passive tissue strain in spinal ligaments causes subsequent short-term changes in reflex. Although studies have documented changes in the myoelectric onset angle of flexion-relaxation following prolonged static flexion and cyclic flexion we could find no published evidence related to the human reflex response of the trunk extensor muscles following a period of static flexion-relaxation loading.

Methods. Eighteen subjects maintained static lumbar flexion for fifteen minutes. Paraspinal muscle reflexes were elicited both before and after the flexion-relaxation protocol using pseudorandom stochastic force disturbances while recording EMG. Reflex gain was computed from the peak value of the impulse response function relating input force perturbation to EMG response using time-domain deconvolution analyses.

Findings. Reflexes showed a trend toward increased gain after the period of flexion-relaxation ($P<0.055$) and were increased with trunk extension exertion ($P<0.021$). Significant gender differences in the reflex gain were observed ($P<.01$).

Interpretations. Occupational activities requiring extended periods of trunk flexion contribute to changes in reflex behavior of the paraspinal muscles. Results suggest potential mechanisms by which flexed posture work may contribute to low back pain. Significant gender differences indicate risk analyses should consider personal factors when considering neuromuscular behavior.

Keywords: Low-Back; Reflex; Flexion-Relaxation

3.1 Introduction

Workers who undergo periods of static or cyclic trunk flexion have an increased risk of low back pain (Marras et al., 1993;Punnet et al., 1991). Biomechanical factors contributing to this risk may include excessive or cumulative spinal load (Kerr et al., 2001), ligament and disc strain (Dolan P. et al., 1994) and spinal instability (Omino and Hayashi, 1992). Active muscular recruitment and reflexes play a major role in both spinal and ligamentous load as well as spinal stability (Gardner-Morse and Stokes, 1998;Granata and Marras, 1995). However, neuromuscular activity may be disturbed by periods of static or cyclic strain in the spinal ligaments and discs (Jackson et al., 2001;Solomonow et al., 1999). A change in reflex response associated with flexed working postures may contribute to limitations in stabilizing control and risk of injury (Solomonow et al., 1998). The goal of this study was to quantify changes in reflex response associated with prolonged trunk flexion in healthy human subjects.

Flexed trunk postures can apply tensile strain to paraspinal muscles and passive viscoelastic tissues of the spine. Flexion-relaxation occurs during extreme lumbar flexion wherein strain of the passive tissues in the trunk and spine support the external flexion load thereby allowing deactivation of the trunk muscles (Kippers and Parker, 1984). Prolonged passive stretch of skeletal muscles influences gamma motor-neuron drive to reduce muscle spindle excitability. Studies in the ankle joint document reflex inhibition following static and cyclic stretch loading (Avela et al., 1999;Rosenbaum and Hennig, 1995). Flexion strain in the ligaments may also influence reflex behavior. Flexion loading of the ligamentous tissues of the spine causes inter-vertebral laxity; specifically tissue creep and stress-relaxation. These effects have been quantified in human cadaveric tissue (Twomey and Taylor, 1982), animal models (Solomonow et al., 2001), and in-vivo human measurements (McGill and Brown, 1992). Existence of reflex pathways have been observed originating from spinal ligaments to surrounding muscles; specifically the multifidus and longissimus (Kang et al., 2002). Thus, a perturbation force applied to the trunk causes transient tissue strain and excitation of large mechanoreceptors (Golgi, Pacinin, Ruffini fibers) in the spinal ligaments as well as type Ia and type II afferent muscle spindle sensors to initiate reflexive action of the paraspinal muscles (Simon, 1994). Biomechanical laxity of the spine from prolonged trunk flexion may influence neuro-sensor strain and reflex in response to force perturbations (Jackson et al, 2001).

Therefore, changes in reflex response following prolonged strain of the spinal ligaments may be related in part to ligament laxity (Solomonow et al, 1999). Although studies have documented changes in the myoelectric onset angle of flexion-relaxation following prolonged static flexion and cyclic flexion (Dickey et al., 2003;Solomonow et al., 2003a), we could find no published evidence related to the human reflex response of the trunk extensor muscles following prolonged passive stretch.

To date, studies linking reflex disturbances to prolonged spinal flexion are limited primarily to animal models (Claude et al., 2003). In surgical preparations a mechanical strain was applied to feline supraspinal ligaments and the electromyographic response was observed in adjacent paraspinal muscles (Solomonow et al, 1998). Following repeated ligament strain a brief period of reflex hyper-excitability was observed (Jackson et al, 2001). However, this period of hyper-excitability was short lived. Ligament laxity caused the mechanoreceptors to become desensitized thereby inhibiting electromyographic response of the paraspinal muscles for many hours (Gedalia et al., 1999). This laxity and disturbed reflex behavior may influence spinal stability (McGill and Brown, 1992). However, we could find no studies that validate these trends in humans. It is necessary to establish whether similar effects are observed from flexion-relaxation postures in humans.

To quantify changes in reflex response, systems identification techniques can be applied to determine the myoelectric response attributable to a force perturbation, i.e. reflex amplitude is represented in terms of the amplitude and temporal characteristics of the impulse response (Zhang et al., 1999). This measurement technique was applied to quantify the reflex response from measured electromyography (EMG) from the paraspinals before and after a period of static flexion-relaxation in healthy human subjects. Based on the available literature we hypothesized that prolonged flexion-relaxation in healthy subjects influences paraspinal reflex response behavior.

3.2 Methods

3.2.1 Subjects

Eighteen subjects, with no previous history of low back pain participated after signing informed consent approved by the institutional review board at Virginia Tech. Subjects included nine females with mean (standard deviation) height 164.8 (7.2) cm. and weight 61.1 (9.6) kg, and nine males with mean height 183.1 (7.4) cm. and mean weight 75.9 (9.2) kg.

3.2.2 Protocol

Muscle recruitment, reflex gain, and perturbation kinematics were quantified before and immediately following a fifteen minute period of static passive flexion loading of the spine. To impose spinal ligament strain subjects sat on the floor with their trunk in full lumbar flexion and legs extended with a small padded bolster under the knees to reduce hamstring stretch and to tilt the pelvis posteriorly. They relaxed in this flexed posture while observing EMG from the erector spinae displayed on an oscilloscope to assure voluntary de-recruitment of the erector spinae, i.e. flexion-relaxation. Immediately following the flexion-relaxation period neuromechanical measurements of muscle recruitment, reflex gain and perturbation kinematics were repeated.

To record muscle response and kinematic behavior subjects stood upright with their pelvis and legs strapped to a rigid support (Figure 3.1). A harness and cable system attached the subject to a servomotor (Pacific Scientific, Rockford, IL, USA) such that cable tension applied flexion loads at the T10 level of the trunk. The motor was programmed to provide three levels of isotonic load, 100 N, 135 N, and 170 N. Superimposed on the preload were force perturbations of ±75 N applied in a pseudo-random stochastic fashion (Figure 3.2a) with a flat bandwidth from 0-10 Hz. Pseudorandom stochastic inputs were chosen to avoid voluntary responses from predictable stimuli (Kearney and Hunter, 1990). The applied forces were measured by a force transducer attached to the motor sampled at 1000 Hz (Omega, Stamford, CT, USA). Three pseudorandom perturbation trials of ten seconds each were performed at each preload level presented in random order before and after the flexion-relaxation protocol. After flexion-relaxation, some recovery may have occurred during the data collection trials. Statistical order effects were minimized by the trial randomization. Furthermore, only three minutes were

required for data collection which was well below the documented recovery time constant, 9.4 minutes, reported by McGill and Brown (McGill and Brown, 1992).

EMG was recorded from bipolar surface EMG electrodes (Delsys, Boston, MA, USA) on the left and right rectus abdominus, internal oblique, external oblique, and erector spinae with placement as described by Marras (Marras and Mirka, 1992). EMG data were band-pass filtered in hardware between 20 and 450 Hz and sampled at 1000 Hz. The EMG signals were rectified and filtered using a 25 Hz, low-pass, seventh-order Butterworth filter in post-processing software (Figure 3.2b). Measured EMG data were normalized with respect to maximum voluntary exertion (MVE) in isometric trunk flexion, extension, combined torsion and extension, and combined torsion and flexion, recorded prior to the experiment. Trunk kinematics in response to the perturbation forces were recorded using surface mounted infrared motion sensors (Northern Digital, Waterloo, Ontario, Canada). Sensors were placed over the subject's spinous processes at S1, L5, T10 and C7 and sampled at 100 Hz Figure 3.2c). Kinematic data was rectified and filtered using a 75 Hz, low-pass, seventh-order Butterworth filter in post-processing software (Matlab, Natick, MA, USA).

3.2.3 Analysis

Reflex response was quantified in terms of latency and gain from the erector spinae EMG. A nonparametric impulse response function (IRF) was calculated from the pseudorandom force input and the rectified EMG output of the erector spinae muscles. Calculation of the nonparametric IRF was based on deconvolution techniques for a time-delayed linear systems response (Mirbagheri et al., 2000). For any linear time-invariant (LTI) system, the output y(t), i.e., rectified EMG, was described by the impulse response function h(t) of the system convolved with (notated *) the applied input signal, x(t), i.e. external trunk force,

$$y(t) = \int_{T_1}^{T_2} h(\tau)x(t-\tau)dt = h(t) * x(t) \tag{3.1}$$

where the $h(\tau)$ is considered trivial for time lags $\tau<T_1$ and $\tau>T_2$. Using this property, any LTI system can be described in its entirety by its IRF because once h(t) is known, the output of the system can be determined for any input. Accordingly, the discrete-time impulse response

function can be represented in terms of the input autocorrelation function and the input/output cross-correlation function:

$$c_{xy}(k) = \Delta t \sum_{j=M_1}^{M_2} h(j) c_{xx}(k-j) \tag{3.2}$$

where the input and output are sampled every Δt seconds, $M_1 = \frac{T_1}{\Delta t}$ and $M_2 = \frac{T_2}{\Delta t}$. This equation can then be written in matrix form as:

$$C_{xy} = \Delta t \, C_{xx} \, H \tag{3.3}$$

where C_{xy} is an $M_2 - M_1 + 1$ length vector whose i^{th} element is $c_{xy}(M_1 + i - 1)$, C_{xx} is an $M_2 - M_1 + 1$ square matrix whose i, j^{th} element is $c_{xx}(i - j)$ and H is an $M_2 - M_1 + 1$ length vector whose i^{th} element is $h(M_1 + i - 1)$. Using simple matrix inversion one can solve for the impulse response function, H, representing the EMG response (Figure 3.3). After $T_2 = 120$ msec any response is considered to be arbitrarily voluntary and therefore not quantified as a reflex (Matthews, 1991).

Both the applied trunk force and the rectified EMG were scaled to yield a mean value of zero prior to analysis in order to ensure accuracy of correlation function estimations. Published techniques illustrate that adding white noise to the input signal reduces the effect of system noise by decorrelating the data sampling artifact from the input signal (Ljung and Ljung, 1998). The magnitude of white noise added was 30% of one standard deviation of the input trunk force.

To measure the quality of the IRF representation of the reflex response, the computed IRF was convolved with the original pseudorandom force sequence to produce an estimate of the EMG signal. Root-mean-square (RMS) difference between the physiologic signal and the estimated signal was recorded as percent variance accounted for (VAF_{Reflex}) (Hunter and Kearney, 1983). VAF_{Reflex} equal to 100% indicates that the IRF predicts exactly the measured EMG signal from the input force perturbations. Reflex gain, G_R, was determined from the peak amplitude of the IRF and the latency, t_R, was the time at which the maximum peak occurred. For this analysis a reflex response was defined as a peak occurring two standard deviations or more above the mean IRF signal. If no peak was above this threshold the trial was discarded.

Since perturbation forces were applied in a random time sequence the trunk movement had random qualitative appearance (Figure 3.2c). To quantify movement amplitude kinematic gain, G_K, was estimated from the peak of the IRF determined from the pseudorandom force input and the position output recorded from the infrared motion sensors (Figure 3.4). G_K represents the dynamic trunk flexion-extension movement in response to the force perturbations(Moorhouse and Granata, 2004). Analogous to the reflex data, VAF_K was computed to estimate the quality of the kinematic measurement.

Secondary analyses included quantification of baseline trunk muscle recruitment and pelvic angle. To quantify trunk muscle recruitment the RMS level of the normalized, rectified and filtered EMG signal from each muscle was quantified during the period of constant isotonic load, i.e. 250 msec mean value recorded immediately before the initiation of the pseudorandom force perturbations. Although subjects maintained an upright trunk posture lumbar lordosis was not controlled. Pelvic angle was quantified as an analog to lordosis and measured from the angle between the two kinematic sensors placed at S1 and L5. This was recorded during the period of constant isotonic load prior to initiation of the pseudorandom force perturbations of each trial.

3.2.4 Statistical Analysis

Statistical repeated measures analyses (ANOVA) were performed to determine the effect of flexion-relaxation, gender and preload on baseline EMG activity, G_R, t_R, VAF_{Reflex}, G_K, and VAF_K. The independent variable of gender was analyzed as a between subject variable while flexion-relaxation effect and preload were analyzed as within subject variables. Significance was determined at the level of $\alpha<0.05$. Tukey honest significant difference post-hoc analyses were used to compare differences among significant treatments. To quantify representative changes in antagonistic co-activation and lumbar lordosis the RMS EMG activity from the rectus abdominis and pelvic angle were similarly tested for influence of gender and flexion-relaxation.

3.3 Results

Reflexes were observed in 81.4% of the trials. There was no significant difference in rate of reflex occurrence for gender, flexion-relaxation effect or preload. VAF_{Reflex} had an average

value of 48%. There were no significant main effects of gender, flexion-relaxation, or preload on VAF_{Reflex}. However, the effect of preload approached significance ($P<0.056$). This phenomenon is attributed to the fact that at higher preload levels EMG response is greater, resulting in a higher signal to noise ratio. At lower preloads the lower signal to noise ratio negatively affects the accuracy of the impulse response. VAF_K had an average value of 93%. There were no significant effects of flexion-relaxation, preload level, or gender on VAF_K.

Analyses revealed that reflex gains, G_R, were influenced significantly by flexion-relaxation, gender and preload as well as a significant gender-by-preload interaction (Tables 3.1 and 3.2). Three way interactions yielded no significance effects and therefore were not reported. Females had larger reflex G_R than males ($P<0.01$). Post-hoc analyses of the gender-by-flexion-relaxation interaction revealed that G_R was greater in females than in males after the flexion-relaxation protocol ($P<0.0002$) but the gender difference failed to reach statistical significance before the period of flexion-relaxation ($P<0.19$).

The main effect of flexion-relaxation in reflex gain was slightly outside the range of significance ($P=0.055$) with a trend suggesting an increase in G_R after flexion. Post-hoc analyses indicated a significant increase in G_R after flexion-relaxation ($P<0.003$) in females. Males had no significant change for the number of subjects tested.

Reflex gain increased with preload. A significant difference in G_R occurred between the lowest and highest preload settings ($P<0.02$), with reflex gain, G_R, being greater at higher preloads. There was also a significant gender difference in G_R at each preload level, with G_R being greater in women than men. There was no significant difference in reflex latency, t_R, due to preload, gender, or flexion-relaxation protocol.

There was a significant main effect of flexion-relaxation in kinematic gain with G_K being reduced after flexion-relaxation ($P<0.003$). There was no significant difference in G_K between males and females. However, gender-by-flexion-relaxation interactions revealed a significant effect due to flexion-relaxation in females ($P<0.011$). There was no significant effect of flexion-relaxation in males for G_K ($P<0.72$). There was a significant main effect of preload on G_K, with the highest G_K occurring at the lowest preload.

Effects of gender and flexion-relaxation on baseline muscle activity and pelvic angle were evaluated (Table 3.3). Rectus abdominis coactivation was greater in females than in males both before and after flexion-relaxation ($P<0.001$). In females the baseline rectus abdominis coactivation showed no significant change due to flexion-relaxation. In males, there was a trend toward increased coactivation following flexion-relaxation ($P<0.070$). Pelvic angle increased following flexion-relaxation ($P<0.002$). A significant gender-by-flexion-relaxation interaction revealed that the change was significant in males ($P<0.024$), but not in female participants ($P<0.23$). There was no main effect of gender in pelvic angle.

3.4 Discussion

Four primary effects were observed in the results. First, trends indicated higher reflex gain following a period of static flexion-relaxation, specifically in females. This indicates greater EMG response per unit of perturbation force, i.e. hyper-excitable paraspinal reflex response following 15 minutes of flexion-relaxation. Second, reflex gain increased with trunk flexion preload. Third, females exhibited larger reflex gains than males. Finally, kinematic gain decreased following flexion-relaxation indicating less trunk movement in response to a force perturbation.

Matthews (Matthews, 1991) showed that the human stretch reflex consists of at least two different components including a short latency spinal reflex and a long latency transcortical reflex. The short latency reflex, occurring between 10-30 msec was observed in some trials (Figure 3.3) but the amplitude was smaller than the peak G_R and therefore not included in the statistical analyses. Peak amplitude of the long latency reflex was represented by the reflex gain, G_R, of the IRF and typically occurs around 60 msec (Matthews, 1991). This accounts for conduction time from the spindle and mechanoreceptors, through a multi-synaptic reflex loop and back to the muscle as well as propogation time of the muscle action potentials. Magnusson et al. (Magnusson et al., 1996) noted that sudden flexion loading and unloading of the trunk yielded mean reflex delays of 100 msec and 50 msec respectively. Thus, experimental protocol may be an important factor when examining reflex latency. In the current study the perturbation loading was stochastic and included both flexion loading and flexion unloading with mean reflex latency $t_R = 67$ msec. Reflex latency, t_R, showed no significant effects for gender, flexion-

relaxation or preload level. However, the response rate was fast enough that it may contribute to control of the trunk in response to external force disturbances.

Trends toward increased reflex gain, G_R, were observed following 15 minutes of static flexion-relaxation posture (P=0.055). Passive stretch of skeletal muscles can reduce muscle spindle excitability thereby inhibiting reflex amplitude (Avela et al, 1999;Rosenbaum and Hennig, 1995). Conversely, animal models demonstrate that passive stretch of spinal ligaments can cause brief hyper-excitability of the paraspinal muscle response (Solomonow et al., 2003b). Our results suggest similar behavior is observed in humans. The force sequences before and after the flexion-relaxation protocol were similar in amplitude and signal character. However, this does not guarantee similar movement dynamics. Kinematic response to the force perturbations, G_K, was reduced following 15 minutes of static flexion-relaxation. Hence, the force disturbance caused less trunk movement after the flexion-relaxation protocol than before. This was contrary to our expectations because published data suggest reduction in stiffness following prolonged flexion-relaxation (McGill and Brown, 1992;Solomonow et al, 2003a) suggesting increases in G_K should be expected. An increase in baseline muscle contraction could partially explain the reduced G_K values after flexion-relaxation. However, no changes in baseline EMG levels were observed in the data. It should be noted that a change in G_K was significant only in the female participants. Similarly, the increase in reflex gain, G_R, was significant only in the female participants. Thus, we believe that the decrease in movement following flexion-relaxation may be attributed to the increase in reflex gain. Recognizing that the natural kinematic frequency of the trunk is approximately 1 Hz (Figure 3.4) the reflex response can contribute as feedback control to limit movement following an external force disturbance. This suggests that paraspinal reflexes may play a significant role in the control of trunk muscle stiffness and associated spinal stability. In animal models brief periods of hyper-excitable reflexes after flexion-relaxation was followed by prolonged periods of reflex depression that may limit spinal stability (Claude et al, 2003;Jackson et al, 2001;Solomonow et al, 1999). Further research is necessary in human subjects to determine whether reflex inhibition is observed following the period of hyperexcitability associated with prolonged flexion relaxation.

Reflex gain increased with trunk extension effort. Motor neurons that initiate the reflex action are influenced by muscle tone and may not be excited if the muscle is completely relaxed (Matthews, 1986). Therefore, research shows that reflex excitation increases with low-level preactivation of muscles whereas at high exertion levels muscle stiffness is increased thereby causing reduced displacement and reflex response (Bennett et al., 1994;Matthews, 1986). The saturation point between enhanced reflex excitability and reduced response is generally considered to be approximately 50% of maximum voluntary contraction. During trunk extension exertions Stokes et al. (Stokes et al., 2000) showed that muscle responses occurred less frequently at high preloads than at low exertion levels. In the current study, mean distance from the L5/S1 junction to the level of T10 where the force perturbation was applied was 14.9 cm indicating that the three preloads were equivalent to external flexion moments of 14.9 N-m, 20.1 N-m, and 25.3 N-m. These are less than 20% of the maximum extension moment strength reported by Parnianpour (Parnianpour et al., 1991). Because the effort levels were below 50% MVC, one should expect increased reflex gain with increased preload. Thus, results were consistent with the neurophysiologic literature, i.e. for the three preload levels tested reflex gain increases significantly from the lowest to the highest level.

Reflex gain was greater in females than males. Since force perturbations of 75 N were applied to both males and females alike, one might expect that the smaller trunk inertia of the women would result in greater movement and subsequently larger G_K. This was not observed. However, we have noted elsewhere that trunk stiffness is greater in females than in males (Moorhouse and Granata, 2004). Increased stiffness combined with smaller inertia causes high natural frequency of motion indicating faster movement in females. The reflex response is strongly influenced by tissue strain rate (Simon, 1994). An estimate of the measured EMG response was achieved by convolving the reflex IRF with the measured input force signal for comparison with the actual EMG signal. The variance-accounted-for (VAF) by the IRF explained approximately half of the EMG signal in the current study. This demonstrates the ability to identify reflex behavior from force perturbations. It also highlights a limitation of the study, i.e. further efforts must attempt to reduce the variability of the reflex gain estimate. Mirbaghari et al. (Mirbagheri et al, 2000) concluded that VAF of the reflex response may exceed 95% if one uses kinematic velocity as the reference disturbance rather that the external force signal examined in the current study. Hence, future research must quantify the paraspinal muscle

reflex response with respect to trunk kinematics. Nonetheless, if the force disturbance produced faster initial kinematic disturbances in the female participants then greater reflex gain should be expected. This has been observed in the knee joint and may explain the gender-specific behavior observed in our data (Shultz et al., 2004). We believe this velocity-dependent contribution to increased reflex gain in women helped to limit movement amplitude following the external force disturbances and explains why G_K was similar in men and women. Further study is necessary to understand gender specific neuromuscular control behaviors that may influence spinal stability.

Several effects remain unexplained. Why was the change in reflex gain following flexion-relaxation observed only in females? As noted above, we believe a gender difference in trunk stiffness may contribute to reflex gain but this fails to explain why flexion-relaxation caused a change in neither G_R nor G_K in male subjects. It is possible that mechanical and neuromuscular response to flexion-relaxation postures is gender specific. Published studies indicate that females developed more spinal laxity than males during static flexion-relaxation protocols (McGill and Brown, 1992; Solomonow et al, 2003a). Nonetheless, those studies report significant increases in trunk laxity and associated tissue creep in males. Hence, we expected change in reflex in the males as well as in females. Trunk muscle co-contraction may contribute to gender effects. Our results agree with previous measurements wherein gender differences in antagonistic coactivity of the trunk muscle have been observed (Granata et al., 2001). There was no change in baseline EMG following flexion-relaxation in either males or females (Table 3.3) but the male participants demonstrated a trend toward greater antagonistic coactivation after flexion-relaxation than before ($P<.07$). Spinal posture may also influence reflex gain by modulating the muscles and ligament strain lengths. Although all subjects maintained an upright trunk posture both before and after the flexion-relaxation protocol, pelvic tilt angle increased following flexion-relaxation in male subjects suggesting greater lumbar lordosis. No change in posture was observed in the female subjects. The combined effects of co-contraction and spinal curvature may influence gender-specific response to flexion-relaxation postures. Gender differences in reflex response to the flexion-relaxation protocol may suggest complex neuromotor and biomechanical mechanisms, and may indicate potential gender differences to injury risk from static flexion work postures.

In conclusion, paraspinal reflexes demonstrated a trend toward increased reflex gain immediately following 15 minutes of static flexion-relaxation posture. These results agree with animal models wherein a hyper-excitable reflex response is observed following prolonged ligament stretch (Solomonow et al., 2003c). It is noteworthy that those feline models demonstrate that the period of hyper-excitability is brief and followed by a prolonged reflex depression lasting up to 7 hours. The current study did not record the recovery of the reflex gain so it remains to be demonstrated whether reflex depression is observed in humans following flexion-relaxation. These changes in reflex response may influence stability control and risk of low-back pain. Patients with low-back pain demonstrate reduced and slowed reflexes compared to asymptomatic control subjects (Luoto et al., 1996;Radebold et al., 2000) but it remains unclear whether this is a compensatory consequence of low-back pain (Van Dieen et al., 2003) or a contributing cause. Nonetheless, it has been proposed that changes in paraspinal muscle response following flexed posture work may contribute to LBP risk (Holm et al., 2002). Results suggest that occupational activities that require prolonged trunk flexion may contribute to changes in reflex behavior of the paraspinal muscles. Results also indicate that risk analyses must consider personal factors including gender when considering neuromuscular behavior that influence spinal load and stability.

3.5 Acknowledgement

This research was supported in part by a grant R01 AR46111 from NIAMS of the National Institutes of Health. We wish to thank G. Slota and G. Kauffman for their assistance in data collection and formatting.

3.6 References

Avela, J., Kyrolainen, H., & Komi, P. V. 1999, Altered reflex sensitivity after repeated and prolonged passive muscle stretching, J.Appl.Physiol., 86, 1283-1291.

Bennett, D. J., Gorassini, M., & Prochazka, A. 1994, Catching a ball: contributions of intrinsic muscle stiffness, reflexes, and higher order responses, Can.J Physiol Pharmacol., 72, 525-534.

Claude, L. N., Solomonow, M., Zhou, B. H., Baratta, E. R. V., & Ping, M. 2003, Neuromuscular dysfunction elicited by cyclic lumbar flexion, Muscle & Nerve, 27, 348-358.

Dickey, J. P., McNorton, S., & Potvin, J. R. 2003, Repeated spinal flexion modulates the flexion–relaxation phenomenon, Clin.Biomech., 18, 783-789.

Dolan P., Earley M., & Adams M.A. 1994, Bending and compressive stresses acting on the lumbar spine during lifting activities, J.Biomechanics, 27, 1237-1248.

Gardner-Morse, M. G. & Stokes, I. A. F. 1998, The effects of abdominal muscle coactivation on lumbar spine stability, Spine, 23, 86-92.

Gedalia, U., Solomonow, M., Zhou, B. H., Baratta, R. V., Lu, Y., & Harris, M. 1999, Biomechanics of increased exposure to lumbar injury caused by cyclic loading - Part 2. Recovery of reflexive muscular stability with rest, Spine, 24, 2461-2467.

Granata, K. P. & Marras, W. S. 1995, An EMG assisted model of biomechanical trunk loading during free-dynamic lifting, J.Biomechanics, 28, 1309-1317.

Granata, K. P., Orishimo, K., & Sanford, A. H. 2001, Trunk Muscle Coactivation in Preparation for Sudden Load, J.Electromyo.Kinesiol., 11, 247-254.

Holm, S. H., Indahl, A., & Solomonow, M. 2002, Sensorimotor control of the spine, J.Electromyogr.Kinesiol., 12, 219-234.

Hunter, I. W. & Kearney, R. E. 1983, Two-sided linear filter identification, Med.Biol.Eng Comput., 21, 203-209.

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

Kang, Y. M., Choi, W. S., & Pickar, J. G. 2002, Electrophysiologic evidence for an intersegmental reflex pathway between lumbar paraspinal tissues, Spine, 27, E56-E63.

Kearney, R. E. & Hunter, I. W. 1990, System Identification of Human Joint Dynamics, Crit.Rev.Biomed.Eng., 18, 55-87.

Kerr, M., Frank, J., Harry, S., Norman, R. W., Wells, R., Neumann, P., & Claire, B. 2001, Biomechanical and psychophysical risk factors for low-back pain at work, Am J.Pub.Health, 91, 1069-1075.

Kippers, V. & Parker, A. W. 1984, Posture related to myoelectric silence of erectores spinae during trunk flexion, Spine, 9, 740-745.

Ljung, L. & Ljung, E. J. 1998, *System Identification: Theory for the User*, 2 edn, Prentice Hall, Upper Saddle River, NJ.

Luoto, S., Taimela, S., Hurri, A., Aalto, H., Pyykko, I., & Alaranta, H. 1996, Psychomotor speed and postural control in chronic low back pain patients. A controlled follow-up study, Spine, 21, 2621-2627.

Magnusson, M. L., Aleksiev, A., Wilder, D. G., Pope, M. H., Spratt, K., Lee, S. H., Goel, V. K., & Weinstein, J. N. 1996, Unexpected load and asymmetric posture as etiologic factors in low back pain., Eur.Spine J., 5, 23-35.

Marras, W. S., Lavender, S. A., Leurgans, S. E., & et.al. 1993, The role of dynamic three-dimensional trunk motion in occupationally-related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury, Spine, 18, 617-628.

Marras, W. S. & Mirka, G. A. 1992, A comprehensive evaluation of trunk response to asymmetric trunk motion, Spine, 17, 318-326.

Matthews, P. B. 1991, The human stretch reflex and the motor cortex, Trends Neurosci., 14, 87-91.

Matthews, P. B. C. 1986, Observations on the automatic compensation of reflex gain on varying the pre-existing level of motor discharge in man, J.Physiol., 374, 73-90.

McGill, S. M. & Brown, S. 1992, Creep response of the lumbar spine to prolonged full flexion, Clin Biomech., 7, 43-46.

Mirbagheri, M. M., Barbeau, H., & Kearney, R. E. 2000, Intrinsic and reflex contributions to human ankle stiffness: Variation with activation level and position, Exp.Brain Res., 135, 423-436.

Moorhouse, K. M. & Granata, K. P. Trunk stiffness and dynamics during active extension exertions. J.Biomechanics . 2004.
Ref Type: In Press

Omino, K. & Hayashi, Y. 1992, Preparation of dynamic posture and occurrence of low back pain, Ergonomics, 35, 693-707.

Parnianpour, M., Campello, M., & Sheikhzadeh, A. 1991, The effect of posture on triaxial trunk strength in different directions: Its biomechanical consideration with respect to incidence of low-back problems in construction industry, Intl.J.Ind.Ergon., 8, 279-287.

Punnet, L., Fine, L. J., Keyserling, W. M., Herrin, G. D., & Chaffin, D. B. 1991, Back disorders and non-neutral trunk postures of automobile assembly workers, Scand.J.Work Envir.Health., 17, 337-346.

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

Rosenbaum, D. & Hennig, E. M. 1995, The influence of stretching and warm-up exercises on Achilles tendon reflex activity, J.Sports Sci., 13, 481-490.

Shultz, S. J., Carcia, C. R., & Perrin, D. H. 2004, Knee joint laxity affects muscle activation patterns in the healthy knee, J Electromyogr.Kinesiol., 14, 475-483.

Simon, S. R. 1994, *Orthopaedic Basic Science* Am.Acad.Orthop.Surg., Rosemont, IL.

Solomonow, M., Baratta, R. V., Banks, A., Freudenberger, C., & Zhou, B. H. 2003a, Flexion-relaxation response to static lumbar flexion in males and females, Clin.Biomech.(Bristol., Avon.), 18, 273-279.

Solomonow, M., Baratta, R. V., Zhou, B. H., Burger, E., Zieske, A., & Gedalia, A. 2003b, Muscular dysfunction elicited by creep of lumbar viscoelastic tissue, J Electromyogr.Kinesiol., 13, 381-396.

Solomonow, M., Eversull, E., He, Z. B., Baratta, R. V., & Zhu, M. P. 2001, Neuromuscular neutral zones associated with viscoelastic hysteresis during cyclic lumbar flexion, Spine, 26, E314-E324.

Solomonow, M., Zhou, B., Baratta, R. V., Lu, Y., & Harras, M. 1999, Biomechanics of Increased Exposure to Lumbar Injury Caused by Cyclic Loading: Part 1. Loss of Reflexive Muscular Stabilization, Spine, 24, 2426-2434.

Solomonow, M., Zhou, B., Harras, M., Lu, Y., & Baratta, R. V. 1998, The ligamento-muscular stabilizing system of the spine, Spine, 23, 2552-2562.

Solomonow, M., Zhou, B. H., Baratta, R. V., & Burger, E. 2003c, Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion, Clin.Biomech., 18, 890-898.

Stokes, I. A. F., Gardner-Morse, M. G., Henry, S. M., & Badger, G. J. 2000, Decrease in trunk muscular response to perturbation with preactivation of lumbar spinal musculature, Spine, 25, 1957-1964.

Twomey, L. T. & Taylor, J. R. 1982, Flexion creep deformation and hysteresis in the lumbar vertebral column, Spine, 7, 116-122.

Van Dieen, J. H., Cholewicki, J., & Radebold, A. 2003, Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine, Spine, 28, 834-841.

Zhang, L. Q., Rymer, W. Z., & Sliwa, J. A. 1999, System identification of tendon reflex dynamics, IEEE Trans.Rehabil.Eng., 7, 193-203.

3.7 Figures and Tables

Figure 3.1. Experimental setup. Subject connected to servomotor for data collection.

Figure 3.2. Force input, erector spinae EMG output and trunk movement in a female subject after the flexion protocol.

Figure 3.3. Impulse Response Function (IRF) for the erector spinae muscle in a female subject after the flexion protocol. The peak reflex gain and rise time are labeled.

Figure 3.4. Impulse Response Function (IRF) relating input force perturbation to trunk movement in a female subject. Kinematic gain, G_K is labeled.

Table 3.1. Effects of isotonic trunk extension preload. Mean (standard deviation) of peak reflex gain, latency, and kinematic gain. Results show significant main effects for gender, preload and two-way interactions on reflex gain, G_R, and kinematic gain, G_K.

Table 3.2. Effects of flexion-relaxation (FR). Mean (standard deviation) of peak reflex gain, latency, and kinematic gain. Interactions between variables of gender, flexion-relaxation effect, and preload are shown. Significances are indicated by superscripts identified in footnotes.

Table 3.3. Mean (standard deviation) of baseline rectus abdominis activity and pelvic angle.

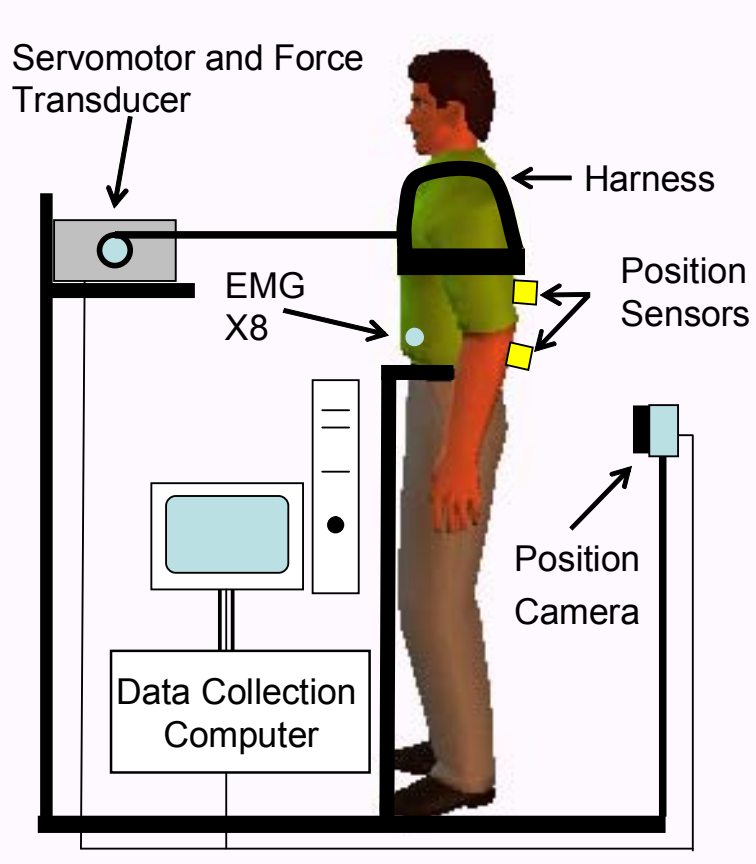

Figure 3.1. Experimental setup. Subject connected to servomotor for data collection.

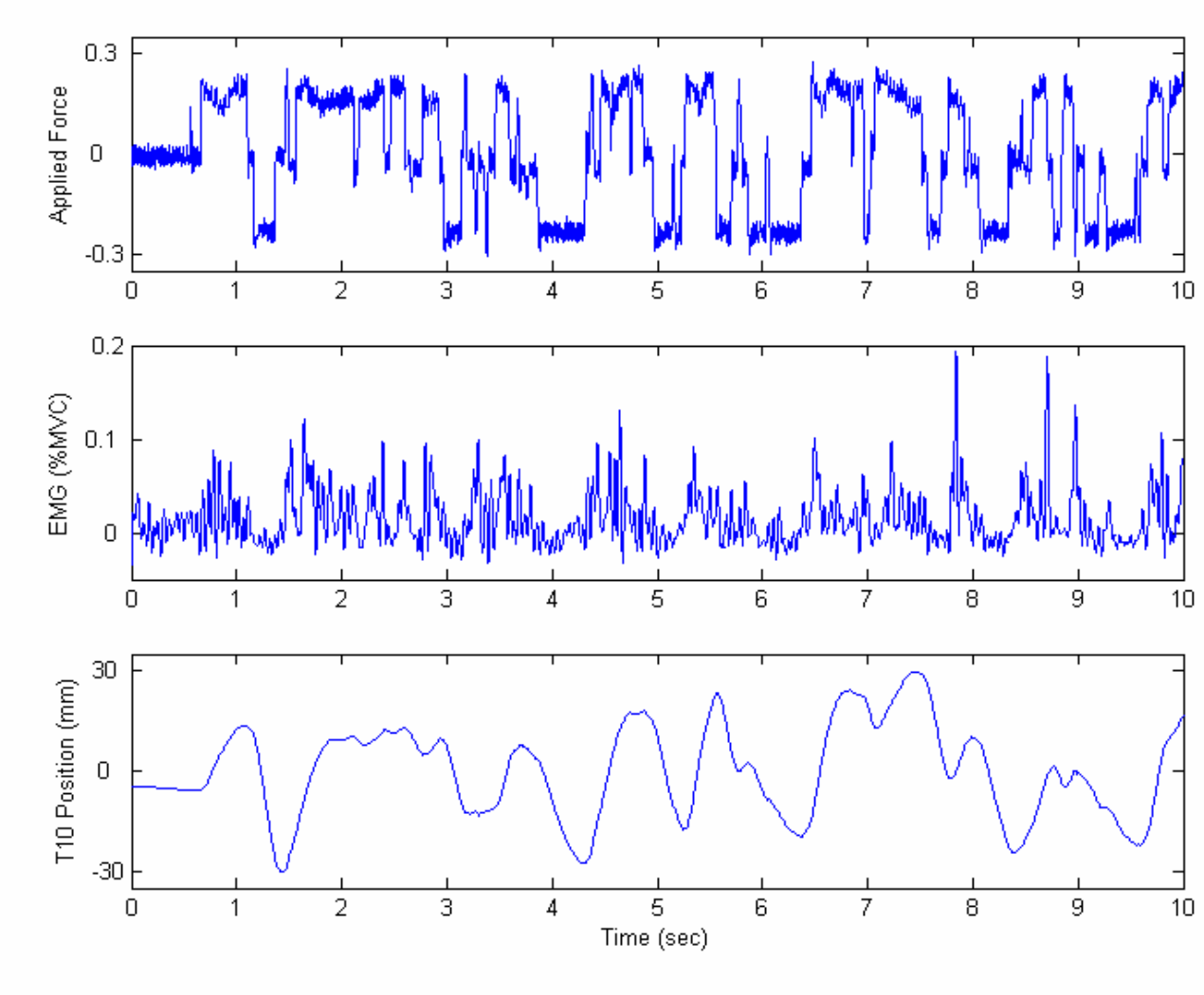

Figure 3.2. Force input, erector spinae EMG output and trunk movement in a female subject after the flexion protocol.

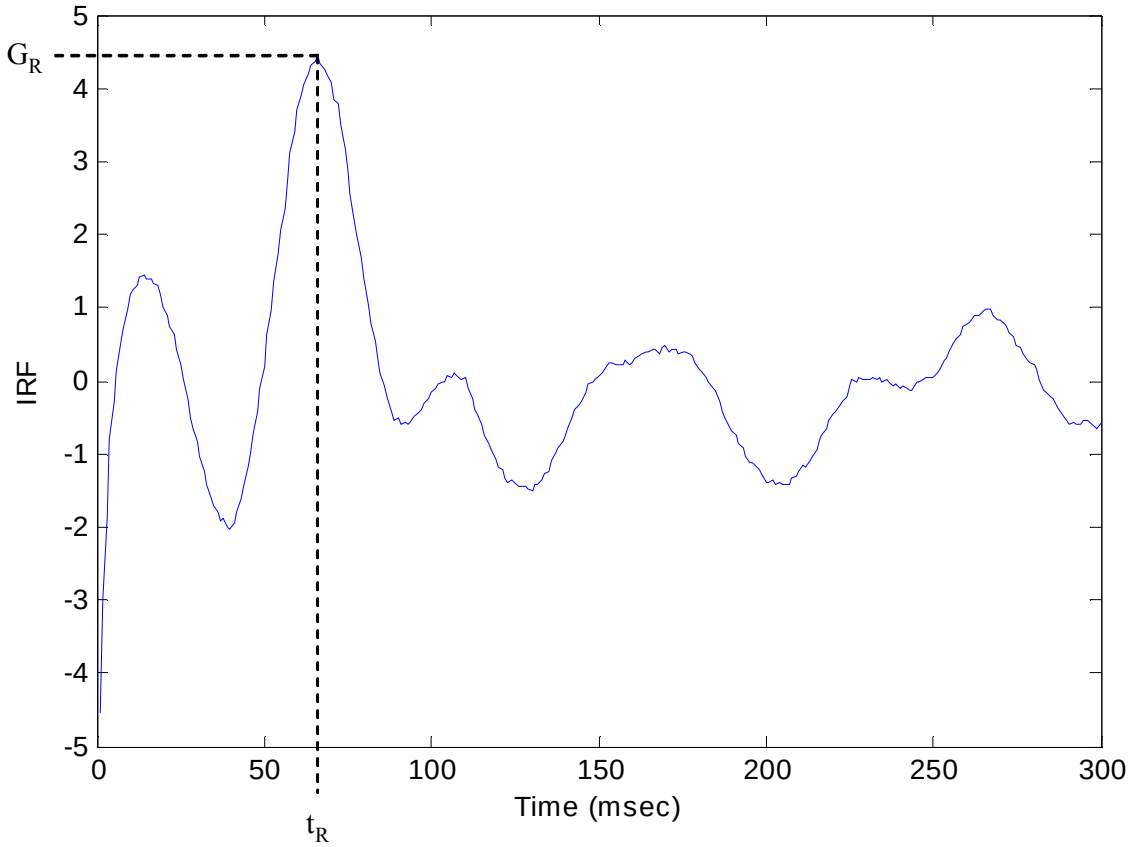

Figure 3.3. Impulse Response Function (IRF) for the erector spinae muscle in a female subject after the flexion protocol. The peak reflex gain and rise time are labeled.

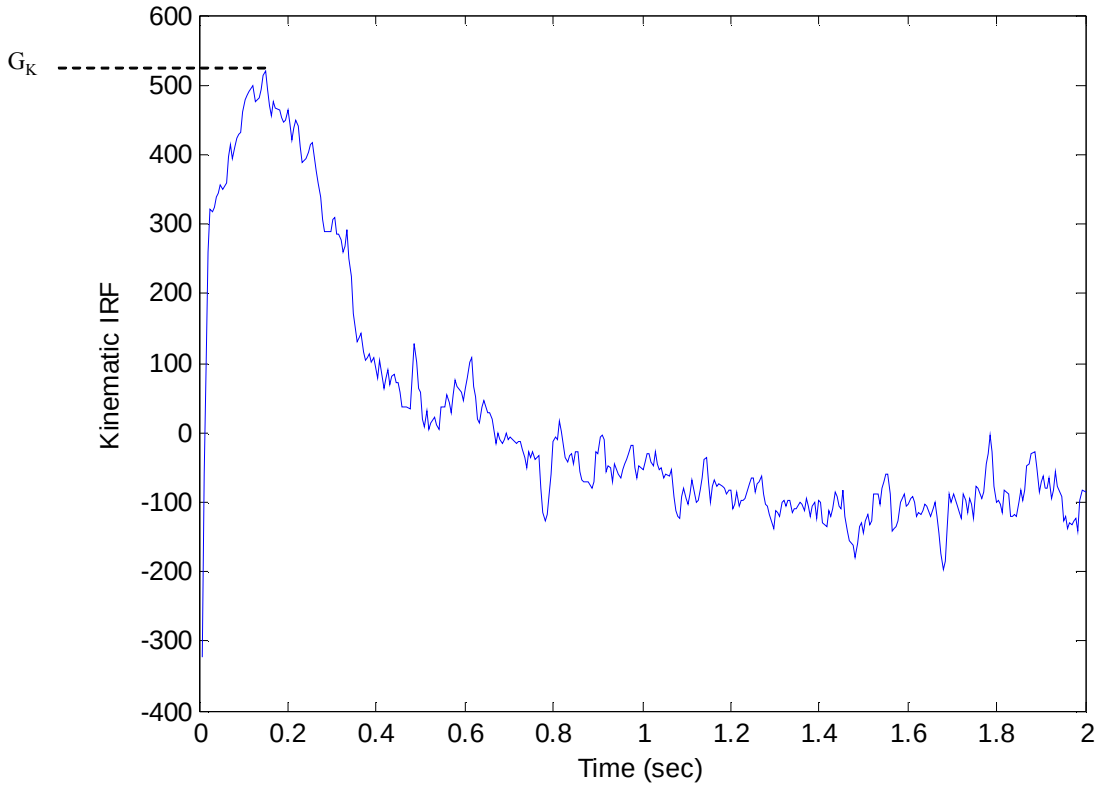

Figure 3.4. Impulse Response Function (IRF) relating input force perturbation to trunk movement in a female subject. Kinematic gain, G_K is labeled.

Table 3.1. Effects of isotonic trunk extension preload. Mean (standard deviation) of peak reflex gain, latency, and kinematic gain. Results show significant main effects for gender, preload and two-way interactions on reflex gain, G_R, and kinematic gain, G_K.

		Men	Women	Average
Reflex Gain G_R (% / N)	**100 N preload**	0.987 (0.375) [3]	1.476 (0.846) [3]	1.224 (0.688) [2]
	135 N preload	1.032 (0.465) [3]	1.467 (0.735) [3]	1.259 (0.640)
	170 N preload	1.112 (0.418) [3]	1.644 (0.834) [3]	1.370 (0.726) [2]
	Average	1.054 (0.420) [1]	1.529 (0.802) [1]	1.285 (0.677)
Reflex Latency t_R (msec)	**100 N preload**	70.70 (17.71)	68.36 (12.07)	69.56 (15.17)
	135 N preload	68.46 (15.93)	65.45 (7.59)	67.00 (12.59)
	170 N preload	66.71 (13.85)	63.62 (9.17)	65.21 (11.83)
	Average	68.62 (15.84)	65.81 (9.89)	67.26 (13.33)
Kinem. Gain G_K (mm / N)	**100 N preload**	590.4 (90.83) [4]	582.4 (85.70) [4]	598.5 (87.8) [2]
	135 N preload	557.5 (74.49)	558.6 (73.34) [4]	558.0 (72.9) [2]
	170 N preload	521.6 (87.88) [4]	511.6 (89.34) [4]	517.2 (87.4) [2]
	Average	556.5 (87.93)	559.6 (90.62)	557.9 (88.73)

[1] significant main effect for gender $P<0.05$

[2] significant main effect for preload $P<0.05$

[3] significance effect of gender within preload level $P<0.05$

[4] significance effect of preload within gender group $P<0.05$

Table 3.2. Effects of flexion-relaxation (FR). Mean (standard deviation) of peak reflex gain, latency, and kinematic gain. Interactions between variables of gender, flexion-relaxation effect, and preload are shown. Significances are indicated by superscripts identified in footnotes.

		Men	Women	Average
Reflex Gain G_R (% / N)	**Before FR**	1.070 (0.469)	1.333 (0.635) [4]	1.198 (0.569) [2]
	After FR	1.038 (0.369) [5]	1.724 (0.905) [4 5]	1.371 (0.763) [2]
	Average	1.054 (0.420) [1]	1.529 (0.802) [1]	1.285 (0.677)
Reflex Latency t_R (msec)	**Before FR**	69.39 (15.83)	65.36 (8.28)	67.43 (12.84)
	After FR	67.86 (15.97)	66.26 (11.33)	67.08 (13.87)
	Average	68.62 (15.84)	65.81 (9.89)	67.26 (13.33)
Kinem. Gain G_K (mm / N)	**Before FR**	562.1 (95.00)	581.1 (96.19) [4]	570.6 (95.10) [3]
	After FR	550.9 (81.47)	538.1 (81.01) [4]	545.2 (80.75) [3]
	Average	556.5 (87.93)	559.6 (90.62)	557.9 (88.73)

FR = Flexion-Relaxation

[1] significant main effect for gender $P<0.01$

[2] main effect for flexion-relaxation approaching significant $p = 0.055$

[3] main effect for flexion-relaxation significant $P< 0.01$

[4] significance effect of flexion-relaxation within gender group $P<0.05$

[5] significance effect of gender within flexion-relaxation condition $P<0.05$

Table 3.3. Mean (standard deviation) of baseline rectus abdominis activity and pelvic angle.

		Men	Women	Average
Rectus Abd EMG (%MVC)	Before FR	0.0336 (0.0603) [2]	0.0752 (0.1684) [2]	0.0544 (0.1276)
	After FR	0.0467 (0.0888) [2]	0.0709 (0.1691) [2]	0.0588 (0.1351)
	Average	0.0402 (0.0758)	0.0731 (0.1681)	0.0566 (0.1311)
Pelvic Angle (deg)	Before FR	12.49 (7.65) [3]	13.97 (6.51)	13.10 (7.03) [1]
	After FR	14.42 (8.08) [3]	15.51 (5.95)	14.87 (7.09) [1]
	Average	13.45 (7.72)	14.74 (6.05)	13.98 (7.01)

FR = Flexion-Relaxation

[1] significant main effect for flexion-relaxation $P<0.05$

[2] significant effect of gender within flexion-relaxation condition $P<0.05$

[3] significance effect of flexion-relaxation within gender group $P<0.05$

CHAPTER 4

DISTURBED PARASPINAL REFLEX FOLLOWING PROLONGED FLEXION-RELAXATION AND RECOVERY

Submitted to: *SPINE* January 2005

Ellen L. Rogers

Kevin P. Granata, Ph.D.

Musculoskeletal Biomechanics Laboratories
Department of Engineering Science and Mechanics
School of Biomedical Engineering
Virginia Polytechnic Institute and State University
219 Norris Hall (0219)
Blacksburg, VA 24061

Keywords: Flexion-relaxation, Reflex, Low-Back, Spine, EMG, Stability

Key-Points

1. Reflexes in the paraspinal muscles and posture of lumbar flexion-relaxation were recorded in human subjects before and immediately after 16 minutes of cyclic flexion-relaxation tasks and after a recovery period.

2. Trunk angle measured during flexion-relaxation postures was significantly greater following cyclic flexion-relaxation tasks indicating creep deformation of passive supporting structures in the trunk.

3. Reflexes were diminished following static flexion-relaxation tasks and failed to recover to baseline levels during 16 minutes of recovery

4. Results suggest that neuromuscular control of spinal stability may be limited following prolonged flexion-relaxation.

Mini-Abstract

Laxity in passive tissues of the spine may contribute to desensitization of mechanoreceptors. Paraspinal reflexes were inhibited following flexion-relaxation with no statistical recovery within 16 minutes of the task.

Abstract

Study Design. Repeated measures experimental study of the effect of flexion-relaxation, recovery, and gender on paraspinal reflex dynamics.

Objective. To determine the effect of prolonged flexion-relaxation and recovery time on reflex behavior in human subjects.

Summary of Background Data. Prolonged spinal flexion has been shown to disturb the paraspinal reflex activity in both animals and humans. Laxity in passive tissues of the spine from flexion strain may contribute to desensitization of mechanoreceptors. Animal studies indicate that recovery of reflexes may take up to several hours. Little is known about human paraspinal reflex behavior following flexion tasks or the recovery of reflex behavior following the flexion tasks.

Methods. Twenty-five subjects performed static flexion-relaxation (FR) tasks. Paraspinal muscle reflexes were recorded before and immediately after FR and after a recovery period. Reflexes were quantified from systems identification analyses of EMG response in relation to pseudorandom force disturbances applied to the trunk.

Results. Trunk angle measured during FR postures was significantly greater following static FR tasks ($p < 0.001$) indicating creep deformation of passive supporting structures in the trunk. Reflex response was diminished following FR ($p < 0.029$) and failed to recover to baseline levels during sixteen minutes of recovery.

Conclusion. Reduced reflex may indicate that the spine is less stable following prolonged FR and therefore susceptible to injury. The absence of recovery in reflex after a substantial time indicates that elevated LBP risk from FR may persist after the end of the flexion task.

Keywords. Flexion-relaxation, Reflex, Low-Back, Spine, EMG, Stability

4.1 Introduction

Trunk flexion postures are a well recognizing risk factor for low-back pain (LBP)[1;2]. Recent investigations have begun to link prolonged and/or cyclic trunk flexion to impaired neuromuscular function. Specifically, animal models demonstrate that the reflex response in the paraspinal muscles is disturbed following spine flexion [3]. This flexion-induced neuromuscular disturbance in those animals was attributed to creep deformation of the passive tissues in the spine [4]. Spinal creep deformation is also observed in humans following static flexion postures[5]. However, we are aware of no studies in humans to investigate whether paraspinal reflexes are disturbed in association with spinal creep deformation from flexed torso postures in humans.

Studies using a feline model show that extreme static or cyclic lumbar flexion causes neuromuscular dysfunction in the form of reduced muscle activity and reflexes. Paraspinal electromyographic (EMG) activity in these animals decreased significantly throughout twenty minutes of static and cyclic flexion [3]. During recovery the animals demonstrated initial transient hyper-excitability followed by depressed reflex response for several hours with slow recovery to normal. Measurements on humans similarly demonstrated hyper-excitable paraspinal reflex response immediately following static flexion [6]. Whether this behavior in humans is transient and followed by reduced reflex response remains to be demonstrated. Other joints have been shown to exhibit reduced reflex behavior in humans following passive flexion [7;8]. No investigation of recovery time is available for human paraspinal reflexes.

One of the underlying mechanisms of reduced paraspinal reflexes associated with prolonged spinal flexion is linked to the function of the viscoelastic tissues in the spine. Mechanoreceptors in the spinal ligaments reflexively activate the paraspinal musculature [9-12]. This reflex regulates continuous spinal movement and acts to control stability to prevent damage from unexpected perturbations and extreme loads. During prolonged static and cyclic lumbar flexion, passive tissues in the spine may provide resistance against flexion load allowing the trunk muscles to become deactivated [13;14]. This phenomenon, known as flexion-relaxation produces tissue laxity and creep, indicated by an increase in relative trunk angle over time [5;15;16]. Laxity in the viscoelastic tissues developed during extreme flexion may cause mechanoreceptors in the ligaments to become desensitized, reducing their ability to monitor vertebral movements

and initiate reflexive muscular action [4]. Slow recovery of reflexes in a feline model may be due to the slow recovery of viscoelastic tissue laxity [17]. Studies indicate that recovery from passive tissue creep in humans takes more time than is required to produce creep deformation, i.e. recovery is slower than deformation [5]. Thus, human paraspinal reflexes should be expected to exhibit a similar pattern during recovery to that of the feline model.

Our previous studies observed modified paraspinal reflex following prolonged static flexion relaxation (sFR) [6] but did not record whether this effect was associated with postural creep deformation nor did it attempt to record recovery from the reflex disturbance. The goal of the current effort was to determine the effect of prolonged FR and recovery time on paraspinal reflex behavior in human subjects. We hypothesize that 1) prolonged FR will result in reduced paraspinal reflex response and 2) after a period of recovery equal in time to that of the FR, viscoelastic tissue creep and reflex response will not have fully recovered. We believe this will indicate that prolonged FR disturbs the neuromuscular system for a significant amount of time following flexion.

4.2 Materials and Methods

4.2.1 Subjects

Twenty-five subjects with no previous history of low back pain participated after signing informed consent approved by the institutional review board at Virginia Tech. Subjects had a mean (standard deviation) age of 21.4 (2.9) years. Subjects included 12 males with mean height 183.9 (5.6) cm. and mean weight 81.6 (11.3) kg, and 13 females with mean height 166.1 (6.9) cm. and mean weight 63.5 (8.6) kg.

4.2.2 Equipment

To test our hypotheses lumbar paraspinal reflexes were examined before, during and after prolonged flexion-relaxation; as well as during and after a recovery period. To record trunk muscle response and kinematic behavior subjects were seated without trunk support in an upright posture (Figure 4.1a). Their pelvis was immobilized by a restraining belt. A harness and cable system attached the subject to a servomotor such that cable tension applied flexion loads at the

T10 level of the trunk. A servomotor (Pacific Scientific, Rockford, IL) was used to elicit paraspinal reflexes according to protocol described in our previous work [6]. The motor provided a constant isotonic preload of 135 N that the subjects were instructed to resist by maintaining their upright seated posture. Pseudorandom stochastic force perturbations of ±75 N were superimposed on the preload (Figure 4.2a). The applied forces were measured by a force transducer attached to the motor (Omega, Stamford, CT) and EMG response to the force perturbations recorded from surface electrodes. For the subsequent protocol description, a reflex trial consists of two ten-second force perturbation trials of the servomotor during which reflexes were elicited and recorded.

Three surface-mounted, six degree of freedom (DOF) electromagnetic position sensors (Motion Star System, Ascension Technology Corp., Burlington, VT) were placed on the subject at S1, T10, and the manubrium, and sampled at 100 Hz. Kinematic data were rectified and filtered using a 25 Hz, low-pass, 7th order Butterworth filter in post-processing software (Matlab, Natick, MA).

Eight pre-gelled active bipolar surface EMG electrodes were placed on the subject at the left and right rectus abdomini, internal obliques, external obliques, and lumbar paraspinal muscles with placement as described by Marras [18]. Data was collected using silver bar electrodes with interelectrode distance of 10 mm integrated into the active differential preamplifier (Delsys DE 2.1, Boston MA). A reference electrode was placed on the prominent bony section of the left tibia. EMG signals were amplified by 1000 with a frequency band-pass of 20 – 450 Hz, CMRR of 92 dB. EMG signals were recorded at 1000 Hz. EMG data were band-stop filtered at 60 Hz and 100 Hz in post-processing software to eliminate electromagnetic interference from the Motion Star System. EMG was rectified and low pass filtered at 25 Hz using a 7th order Butterworth filter. Both force and EMG signals were treated with the same filters to avoid phase shift discrepancy from processing. Rectified and filtered EMG was subsequently normalized to levels recorded during maximum voluntary exertions performed in isometric trunk flexion, extension, combined torsion and extension, and combined torsion and flexion.

4.2.3 Experimental Protocol

Experimental protocol was divided into two phases, static flexion-relaxation (sFR) and recovery, each containing four cycles. Paraspinal reflexes were recorded prior to the beginning of sFR as described above. To impose spinal ligament stretch, subjects remained within the pelvic restraint which acted to prevent anterior tilt of the pelvis and initiated a full flexion posture by leaning forward (Figure 4.1b) as described by McGill [5] and Solomonow [19]. Feet were positioned so that the knees were slightly bent and the amount of stretch occurring in the hamstrings was reduced. This posture was held for four minutes. Flexion-relaxation occurred while subjects were seated in full flexion, monitored by measured erector spinae EMG. The four periods of static flexion are referred to as sFR to indicate that subjects maintained full static flexion during which flexion-relaxation occurred.

During each cycle of sFR both EMG and kinematic data were recorded for five seconds at the beginning of every minute. This allowed us to measure the progression of trunk angle and to monitor that erector-spinae EMG was in fact de-recruited to assure FR. At the end of the four minute sFR period subjects returned to an upright posture and a reflex trial was recorded. This cycle was repeated four times for a total of sixteen minutes of sFR. Both the FR posture and reflex data collection were performed in a seated posture to facilitate transition time from FR to reflex measurement. Less than one minute was required between sFR cycles to record the reflex data and return them to the sFR posture.

Immediately following the sFR phase subjects entered the Recovery phase which included four four-minute cycles, each separated by a reflex trial. During each four minute recovery cycle, subjects sat in an upright and relaxed posture. At the beginning of each minute they were instructed to resume the flexed posture for approximately ten seconds so that kinematic data could be collected and recovery-progression of trunk angle could be monitored.

Although a reflex trial was recorded every four minutes and trunk angle every minute, only the three most relevant positions in time were analyzed statistically: time T_0 was the baseline measurement recorded before the first sFR cycle; time T_{FR} represents the measurement time immediately following the fourth and final cycle of sFR; time T_R was immediately following the fourth and final cycle of recovery.

4.2.4 Data Analysis

Reflex response was quantified in terms of latency and gain from the erector spinae EMG in the method of our previous work [6]. A nonparametric impulse response function (IRF) was calculated from the pseudorandom force input and the rectified EMG output of the erector spinae muscles. Calculation of the nonparametric IRF was based on deconvolution techniques for a time-delayed linear systems response [20]. For any linear time-invariant (LTI) system, the output y(t), i.e., rectified EMG, was described by the impulse response function h(t) of the system convolved with (notated *) the applied input signal, x(t), i.e. external trunk force,

$$y(t) = \int_{T_1}^{T_2} h(\tau)x(t-\tau)dt = h(t) * x(t) \tag{4.1}$$

where the $h(\tau)$ is considered trivial for time lags $\tau<T_1$ and $\tau>T_2$. Using this property, any LTI system can be described in its entirety by its IRF because once h(t) is known, the output of the system can be determined for any input. Accordingly, the discrete-time impulse response function can be represented in terms of the input autocorrelation function and the input/output cross-correlation function:

$$c_{xy}(k) = \Delta t \sum_{j=M_1}^{M_2} h(j)c_{xx}(k-j) \tag{4.2}$$

where the input and output are sampled every Δt seconds, $M_1 = \frac{T_1}{\Delta t}$ and $M_2 = \frac{T_2}{\Delta t}$. This equation can then be written in matrix form as:

$$C_{xy} = \Delta t\, C_{xx}\, H \tag{4.3}$$

where C_{xy} is an $M_2 - M_1 + 1$ length vector whose i^{th} element is $c_{xy}(M_1 + i - 1)$, C_{xx} is an $M_2 - M_1 + 1$ square matrix whose i, j^{th} element is $c_{xx}(i - j)$ and H is an $M_2 - M_1 + 1$ length vector whose i^{th} element is $h(M_1 + i - 1)$. Using simple matrix inversion one can solve for the impulse response function, H, representing the EMG response (Figure 4.3).

Reflex gain, G_R, represents the amplitude of the transfer function relating the paraspinal EMG response to the perturbation force (Figure 4.3). Reflex gain characterizes the magnitude of muscle reflex response scaled respect to the time-dependent amplitude of the force disturbance. High G_R indicates a large reflex response, small G_R indicates minimal muscle activity following

the disturbance. G_R was quantified based on the peak of the IRF computed from the applied input force and rectified EMG output of paraspinal muscles. Peak amplitude of the IRF must be greater than two standard deviations above the mean of the IRF in order to be considered a reflex. A second criterion required that G_R must occur between 20 and 120 msec following the perturbation. This ensured that the reflex response was not confounded by electromechanical delay or by voluntary contribution to EMG response.

The dynamic trunk flexion-extension movement in response to the force perturbations was analyzed using an approach similar to that of the reflex gain. Movement was quantified by an IRF, using the applied force as the input and trunk movement (i.e. linear position of T10 sensor) as the output response [21]. The kinematic gain, G_K, was estimated from the peak of the kinematic IRF (Figure 4.4).

To measure the quality of the IRF representation of both the reflex and kinematic responses the computed IRF was convolved with the original pseudorandom force sequence to produce an estimate of the output signal. RMS difference between the physiologic signal and the estimated signal was recorded as percent variance accounted for (VAF). VAF equal to 100% indicates that the IRF exactly predicts the measured signal from the input force perturbations.

To obtain a measure of ligament elongation or laxity, two trunk angle measurements were recorded. The sagittal trunk angle, θ_T, recorded the overall angle of the trunk in the sagittal plane, determined from the difference in angle between the markers at S1 and the manubrium. The second measure was lordosis angle, θ_L, which was a relative angle of the curvature of the spine from S1 to T10. Lordosis angle was computed from the difference in the sagittal angle of the position sensors located at S1 and T10. Both trunk angle and lordosis angle were recorded while the subject was fully flexed. For comparison between subjects, trunk angle was taken as the difference from the first flexion angle recorded.

To quantify trunk muscle co-recruitment the RMS level of the rectified, filtered, and normalized EMG signal from each muscle was quantified during the 250 msec period of constant isotonic load, immediately before the initiation of the pseudorandom force perturbations.

4.2.5 Statistical Analysis

Statistical repeated measures analysis, ANOVA, was performed to determine the effect of gender, sFR and recovery on G_R, and G_K, trunk muscle recruitment, and trunk angle. The independent variable of gender was analyzed as a between subject variable, while the effects of sFR and recovery were analyzed as a within subject variable. Significance was determined at the level of $\alpha < 0.05$. Tukey honest-significant difference post-hoc analyses were used to compare differences among significant treatments.

4.3 Results

Analyses revealed that the sagittal trunk angle, θ_T, increased significantly from T_0 to T_{FR} ($p < 0.001$) and from T_0 to T_R ($p < 0.001$), indicating tissue elongation occurred during the flexion protocol (Table 4.1). However there was no significant change in θ_T from T_{FR} to T_R, possibly suggesting that the Recovery phase of the protocol was insufficient to allow laxity in the passive tissues to recover. There was no significant difference in main effect of gender on θ_T.

Lordosis angle, θ_L, results also revealed increases due to sFR from T_0 to T_{FR} ($p < 0.001$) and from T_0 to T_R, ($p < 0.001$). There was no significant change from T_{FR} to T_R, further indicating that passive tissues were unable to recover in this protocol. There was no significant main effect of gender on θ_L. Post-hoc analyses of a significant gender-by-time interactions indicated that in males there was a significant change in θ_L from T_0 to T_{FR} ($p < 0.02$) but not from T_0 to T_R ($p < 0.08$). However, in females there was a significant increase in θ_L from T_0 to T_{FR} ($p < 0.001$) and T_0 to T_R ($p < 0.001$). There was no significant change from T_{FR} to T_R in males or females.

Minute-by-minute changes in θ_T and θ_L were analyzed during the flexion period to determine whether recovery took place during the minute of upright sitting required for reflex trials. θ_T showed no significant recovery following the minute of upright sitting. θ_L however did exhibit significant recovery from the end of the four-minute flexion periods to the beginning of the following flexion periods ($p < 0.008$). This recovery was significant only in females ($p < 0.022$).

Reflexes were observed in 84% of trials with the remaining trials failing to satisfy the criteria for defining G_R. Reflex variance accounted for, VAF_{reflex}, had an average value of 47.4%. This average value of VAF_{reflex} is consistent with previous results in our laboratory [6].

The main effect of reflex gain, G_R, due to sFR was significant. G_R decreased significantly from T_0 to T_{FR} ($p < 0.029$). There was a significant difference in G_R from T_0 to T_R, but no significant difference in G_R between T_{FR} and T_R indicating that reflex gain failed to recover within the sixteen minute recovery period. There was no significant main effect of gender on G_R. Post-hoc analyses yielded significant differences in G_R between males and females at T_0. At T_0, females had significantly higher G_R than males ($p < 0.018$), but there was no difference between genders at T_{FR} or T_R. In males, neither sFR nor recovery was a significant factor on G_R. However, in females reflex gain decreased significantly from T_0 to T_{FR} ($p < 0.047$) and from T_0 to T_R ($p < 0.001$). There was no significant difference in G_R between T_{FR} and T_R in either gender.

Movement analyses yielded an average value of kinematic variance accounted for, VAF_K, of 91.5%. Kinematic gain, G_K, showed a trend suggesting a decrease from T_0 to T_{FR} ($p < 0.062$) but no significant change from T_0 to T_R or T_{FR} to T_R. There was a no significant main effect or interactions with gender in G_K.

The effects of gender, sFR and recovery on trunk muscle recruitment were evaluated. No muscle groups showed significant gender differences in baseline EMG. Right and left external oblique muscles showed significant decrease in baseline EMG activation level from T_0 to T_{FR}. There was no significant change from T_0 to T_R or from T_{FR} to T_R. No other muscle group showed significant differences in EMG activation level due to sFR or recovery.

4.4 Discussion

Results show that prolonged sFR influenced the function of the neuromuscular system in humans. Lumbar paraspinal reflex gain decreased significantly after sixteen minutes of flexion-relaxation, which was consistent with our hypothesis. Reduced reflexes may suggest that the spine was less stable following prolonged FR and therefore more susceptible to LBP and injury [22]. Research indicates that LBP patients to exhibit reduced responsive control and delayed

muscle responses compared to healthy subjects [23;24]. The decrease in reflex gain observed in the current study may be linked to the significant increase in spinal tissue laxity associated with sFR. An increase in trunk angle indicates that passive tissue creep occurred and laxity in these tissues may have contributed to desensitization of neurosensors [25]. However, the results of this investigation were inconsistent with our previous results wherein reflex behavior was recorded from subjects following fifteen minutes of continuous sFR [6]. In that study we found a trend toward increased reflex gain immediately following prolonged sFR, which we concluded was consistent with the hyperexcitable EMG response found in animal research [25]. There are several possible explanations as to why our previous results differ from what we found here. Solomonow et al. [3] investigated muscle activity during both static and cyclic loading. In the case of static loading, hyperexcitability was present immediately following the end of loading and EMG was typically elevated from its baseline level. In the case of cyclic loading the EMG magnitude following the loading was typically less than baseline. This could be due to the intermittent rest periods associated with cyclic loading, although further investigation into this issue is necessary. In the current study subjects underwent sixteen minutes of sFR but the four-minute cycles were separated by approximately one minute of seated upright active exertion to record reflexes (Figure 4.1). Therefore, our protocol may be considered cyclic rather than static flexion and it may be reasonable to expect different results than static FR.

One major difference between this study and our previous investigation is subject position during reflex trials, i.e. upright standing versus upright sitting. It is possible that this difference may contribute to the discrepancy in our results regarding hyper-excitable versus depressed reflex, though it is unclear precisely what effect this difference has on reflex response. In the seated posture hip movement in response to force perturbations was less than in the standing posture, which could enhance accuracy of results. Furthermore, lengths of the paraspinal muscles are different in standing versus sitting postures which may affect reflex gain values. One major drawback of our previous attempt to measure the effect of sFR on reflex response was the absence of trunk angle measurements during flexion. In the current study the creep deformation of the trunk posture was documented to demonstrate that reflex change after sFR may be associated with the viscoelastic creep. Further research is necessary to determine whether this neuromechanical relationship is causative.

The second noteworthy result of this investigation was that reflex gain failed to change during the recovery phase. This indicates that no appreciable recovery of paraspinal reflexes took place. This outcome may be due to the fact that neither lordosis nor sagittal trunk angle recovered within the experimental session, indicating that creep in the viscoelastic tissues may not have recovered sufficiently to restore sensitivity to the mechanoreceptors for reflex initiation. A ten second period of flexion was performed each minute throughout the recovery phase to measure trunk angle and laxity recovery. This repeated ligament strain may have been too frequent to allow recovery of passive tissues. Nevertheless, feline models showed depressed reflexes several hours after prolonged flexion [26]. Hence, reflex disturbance may require longer to recover than sixteen minutes following sixteen minutes of sFR. Our results agree with the literature in that the recovery process for tissue creep and reflex response may be slow.

Of further interest is the progression of reflex gain and trunk angle throughout the entire protocol (Figure 4.5). Reflex gain was measured every four minutes during FR and every four minutes during recovery for a total of nine measurements. Trunk angle was measured every minute. While only the main times of interest were statistically analyzed for changes in reflex the patterns of change in lordosis angle and reflex are interesting to note. Following a cycle of sFR and one minute of sitting in an upright posture, i.e. during reflex trials, θ_L exhibited significant recovery. Any recovery achieved was transient because by the second minute of the ensuing sFR cycle θ_L returned to elevated levels and continued increasing during the remaining sFR. This trend is interesting to note in conjunction with results from animal models showing that recovery may be transient [4]. A ten minute rest after fifty minutes of cyclic flexion loading of the feline lumbar spine allowed between 20% and 30% recovery of reflexive muscle activity. However all recovery was completely lost after only twenty seconds of additional loading following the rest period. These results underscore the importance of adequate rest periods following torso flexion loading. One minute of rest after four minutes of flexion appeared to be inadequate for recovery of lumbar creep deformation and reflex gain. The importance of frequent and long rest periods during prolonged flexion is clear but the amount of rest necessary to prevent significant reflex reduction and its influence on spinal stability has yet to be determined.

Lordosis angle results were consistent with McGill [5] who showed that recovery of passive tissue creep takes longer than the time in which the creep occurred. During the recovery cycles, lordosis angle remained approximately constant, presumably due to the repeated bouts of flexion performed for data collection. However, θ_L clearly did not increase further, indicating that this duty cycle of flexion was not enough to cause further tissue creep. Further investigation into the disturbance of paraspinal reflexes as a result of various flexion duty cycles is necessary.

There was a trend suggesting reduced kinematic gain immediately following FR ($p < 0.062$). This is consistent with our previous results wherein a significant decrease in kinematic gain was observed following 15 minutes of sFR. Decreased kinematic gain implies that trunk displacement decreased and therefore trunk stiffness was increased following sFR. Active spinal musculature has been shown to increase trunk stiffness [21]. In order to reduce trunk displacement, spinal muscles should exhibit increased activation levels following sFR [27]. However, of the eight muscles examined, only the external obliques showed a significant change in baseline activity after sFR, and this was actually a reduction in activation after sFR. The other major muscle groups of the trunk showed no significant change after sFR so increased trunk stiffness cannot be attributed to changes in co-contraction. Andersson et al. [28] showed that while superficial erector spinae muscles deactivate during FR, deep lumbar muscles, such as the quadratus lumborum and deep erector spinae, become more active with increasing trunk flexion angle. Measurement of these muscle groups would require indwelling EMG electrodes, and therefore were not recorded in our study. It is possible that the activity of these muscles contributed to the reduced trunk displacement and apparent increase in stiffness following sFR.

Results of this study must be viewed in light of certain methodological limitations. The mean VAF_{reflex} of 47% agrees with published VAF values using similar linear EMG analyses [29]. However, nonlinear EMG analyses have yielded higher VAF values in other joints, so these methods should be investigated in the future. An inherent limitation may arise from our protocol due to the fact that subjects were seated during reflex trials. Loads imposed on laborers may be different from those in our experimental settings. The seated posture was used in this study to limit the transition time between sFR periods and reflex measurements. Trials discarded for not meeting the determined reflex gain criteria may indicate a limitation of the statistical analysis

reported. Inclusion of these trials may have increased variability of results. Muscle fatigue may also influence the results, but since the paraspinal muscles were de-recruited during the sFR postures fatigue is highly unlikely. Despite these limitations, our results appear to correspond with literature concerning reflexes in a feline model.

Results confirm in humans that prolonged static flexion results in passive tissue creep along with a neuromuscular disorder and that extended rest may be required for full recovery. Recovery of passive tissue creep and lumbar paraspinal reflexes required time longer than measured in this study which was equal to the time spent in a flexed posture. Inhibited paraspinal reflexes may contribute to risk of LBP or injury for workers using flexed postures, due to the inability of the neuromuscular system to coordinate an appropriate muscle response following an unexpected loading event. General recommendations for occupational situations include limiting exposure to flexed posture work and providing long rest breaks. Further research is required for the recommendation of specific guidelines.

4.5 Acknowledgement

This research was supported by grants R01 AR46111 from NIAMS of the National Institutes of Health and R01 OH07352-01 from NIOSH of the Centers of Disease Control. We wish to thank G. Slota and G. Kauffman for their assistance in data collection and processing.

4.6 References

1. Punnett L and Wegman DH. Work-related musculoskeletal disorders: the epidemiologic evidence and the debate. J Electr.Kines. 2004;14:13-23.

2. Marras WS, Lavender SA, Leurgans SE, and et.al. The role of dynamic three-dimensional trunk motion in occupationally-related low back disorders: The effects of workplace factors, trunk position and trunk motion characteristics on risk of injury. Spine 1993;18:617-28.

3. Solomonow M, Baratta RV, Zhou BH, Burger E, Zieske A, and Gedalia A. Muscular dysfunction elicited by creep of lumbar viscoelastic tissue. J Electr.Kines. 2003;13:381-96.

4. Solomonow M, Zhou B, Baratta RV, Lu Y, and Harras M. Biomechanics of Increased Exposure to Lumbar Injury Caused by Cyclic Loading: Part 1. Loss of Reflexive Muscular Stabilization. Spine 1999;24:2426-34.

5. McGill SM and Brown S. Creep response of the lumbar spine to prolonged full flexion. Clin Biomech. 1992;7:43-6.

6. Granata KP, Rogers E, and Moorhouse KM. Effects of static flexion-relaxation on paraspinal reflex dynamics. J.Biomech. 2004;24:16-24.

7. Rosenbaum D and Hennig EM. The influence of stretching and warm-up exercises on Achilles tendon reflex activity. J.Sports Sci. 1995;13:481-90.

8. Avela J, Kyrolainen H, and Komi PV. Altered reflex sensitivity after repeated and prolonged passive muscle stretching. J.Appl.Physiol. 1999;86:1283-91.

9. Stubbs M, Harris M, Solomonow M, Zhou B, Lu Y, and Baratta RV. Ligamento-muscular protective reflex in the lumbar spine of the feline. J Electr.Kines. 1998;8:197-204.

10. Panjabi MM. Clinical spinal instability and low back pain. J Electr.Kines. 2003;13:371-9.

11. Solomonow M, Zhou B, Harras M, Lu Y, and Baratta RV. The ligamento-muscular stabilizing system of the spine. Spine 1998;23:2552-62.

12. Kang YM, Choi WS, and Pickar JG. Electrophysiologic evidence for an intersegmental reflex pathway between lumbar paraspinal tissues. Spine 2002;27:E56-E63.

13. Schultz AB, Haderspeck-Grib K, Sinkora G, and Warwick DN. Quantitative studies of the flexion-relaxation phenomenon in the back muscles. J Orthop.Res. 1985;3:189-97.

14. Kippers V and Parker AW. Posture related to myoelectric silence of erectores spinae during trunk flexion. Spine 1984;9:740-5.

15. Dickey JP, McNorton S, and Potvin JR. Repeated spinal flexion modulates the flexion–relaxation phenomenon. Clin.Biomech. 2003;18:783-9.

16. Olson MW, Li L, and Solomonow M. Flexion-relaxation response to cyclic lumbar flexion. Clin.Biomech.(Bristol., Avon.) 2004;19:769-76.

17. Gedalia U, Solomonow M, Zhou BH, Baratta RV, Lu Y, and Harris M. Biomechanics of increased exposure to lumbar injury caused by cyclic loading - Part 2. Recovery of reflexive muscular stability with rest. Spine 1999;24:2461-7.

18. Marras WS and Mirka GA. A comprehensive evaluation of trunk response to asymmetric trunk motion. Spine 1992;17:318-26.

19. Solomonow M, Baratta RV, Banks A, Freudenberger C, and Zhou BH. Flexion-relaxation response to static lumbar flexion in males and females. Clin.Biomech.(Bristol., Avon.) 2003;18:273-9.

20. Mirbagheri MM, Barbeau H, and Kearney RE. Intrinsic and reflex contributions to human ankle stiffness: Variation with activation level and position. Exp.Brain Res. 2000;135:423-36.

21. Moorhouse KM, Granata K P. Trunk stiffness and dynamics during active extension exertions. In: J.Biomechanics. (In Press).

22. Panjabi M.M. The stabilizing system of the spine. Part I Function, dysfunction, adaptation and enhancement. J.Spinal Disorders 1992;5:383-9.

23. Radebold A, Cholewicki J, Panjabi MM, and Patel TC. Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine 2000;25:947-54.

24. Radebold A, Cholewicki J, Polzhofer GA, and Green TP. Impaired postural control of the lumbar spine is associated with delayed muscle response times in patients with chronic idiopathic low back pain. Spine 2001;26:724-30.

25. Solomonow M, Zhou BH, Baratta RV, and Burger E. Biomechanics and electromyography of a cumulative lumbar disorder: response to static flexion. Clin.Biomech. 2003;18:890-8.

26. Solomonow M, Zhou BH, Baratta RV, Lu Y, Zhu MP, and Harris M. Biexponential recovery model of lumbar viscoelastic laxity and reflexive muscular activity after prolonged cyclic loading. Clinical Biomechanics 2000;15:167-75.

27. Cholewicki J, Juluru K, and McGill SM. Intra-abdominal pressure mechanism for stabilizing the lumbar spine. J Biomech. 1999;32:13-7.

28. Andersson E.A., Oddsson L.I.E., Grundstrom H., Nilsson J., and Thorensson A. EMG activities of the quadratus lumborum and reector spinae muscles during flexion-relaxation and other motor tasks. Clin.Biomech. 1996;11:392-400.

29. Kearney RE and Hunter IW. System identification of human triceps surae stretch reflex dynamics. Exp.Brain Res. 1983;51:117-27.

4.7 Figures and Tables

Figure 4.1. a) Subjects were seated in an upright posture for both reflex measurement and recovery. To quantify paraspinal reflex gain a cable and harness system connected the subject to the servomotor, which applied pseudorandom force perturbations to elicit reflexes. b) To achieve passive spinal flexion the subjects flexed forward into static flexion-relaxation (sFR). This posture was held for four cycles of four minutes each, separated by one minute of upright sitting. Reflexes were measured before and immediately after each sFR cycle.

Figure 4.2. a) Applied force including preload and pseudorandom force perturbations. b) Rectified lumbar paraspinal muscle EMG data for a typical reflex trial.

Figure 4.3. Typical Impulse Response Function (IRF) computed from applied trunk force (input) and rectified EMG (output). Reflex Gain, G_R was competed from the peak of the IRF Data at times greater than 120 msec were assumed arbitrarily voluntary and therefore appears as noise in the IRF.

Figure 4.4. Typical Impulse Response Function computed from input applied trunk force and output T10 displacement. Kinematic Gain, G_K is labeled.

Figure 4.5. Progression of a) lordosis angle and b) reflex gain. In the lordosis angle plot, the four minute cycles are depicted by connecting lines, whereas the minute in between cycles is represented by a disconnect. Significant recovery of lordosis angle was observed during the minute between trials, but the recovery was incomplete. Reflex gain was analyzed at time T_0 (baseline measurement recorded before the first sFR cycle), T_{FR} (immediately following the fourth and final cycle of sFR) and T_R (immediately following the fourth and final cycle of recovery) and revealed a significant decline following the sFR trials.

Table 4.1. Mean (and standard deviation) of trunk angle, reflex gain, and kinematic gain. Reflex gain and kinematic gain were recorded in upright postures, while trunk and lordosis angles were recorded in fully flexed postures. Significant effects are labeled with superscripts.

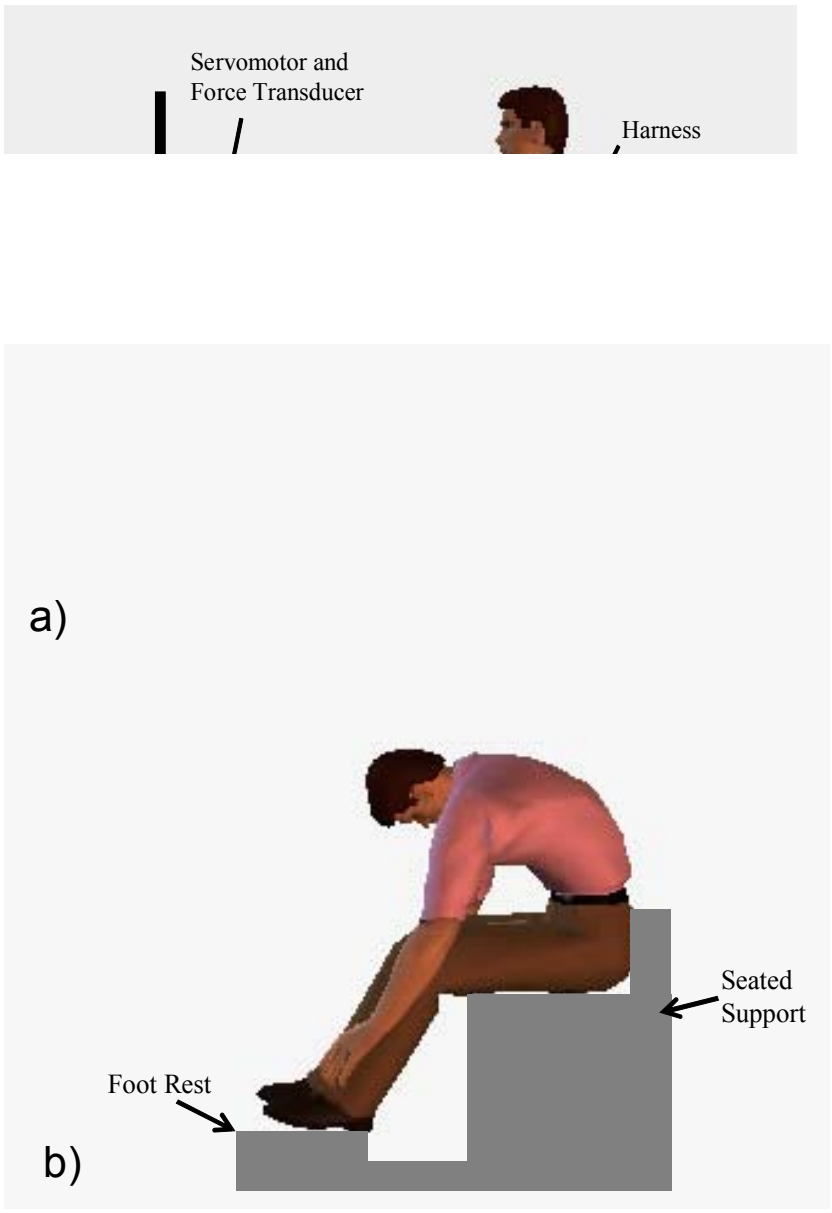

Figure 4.1. a) Subjects were seated in an upright posture for both reflex measurement and recovery. To quantify paraspinal reflex gain a cable and harness system connected the subject to the servomotor, which applied pseudorandom force perturbations to elicit reflexes. b) To achieve passive spinal flexion the subjects flexed forward into static flexion-relaxation (sFR). This posture was held for four cycles of four minutes each, separated by one minute of upright sitting. Reflexes were measured before and immediately after each sFR cycle.

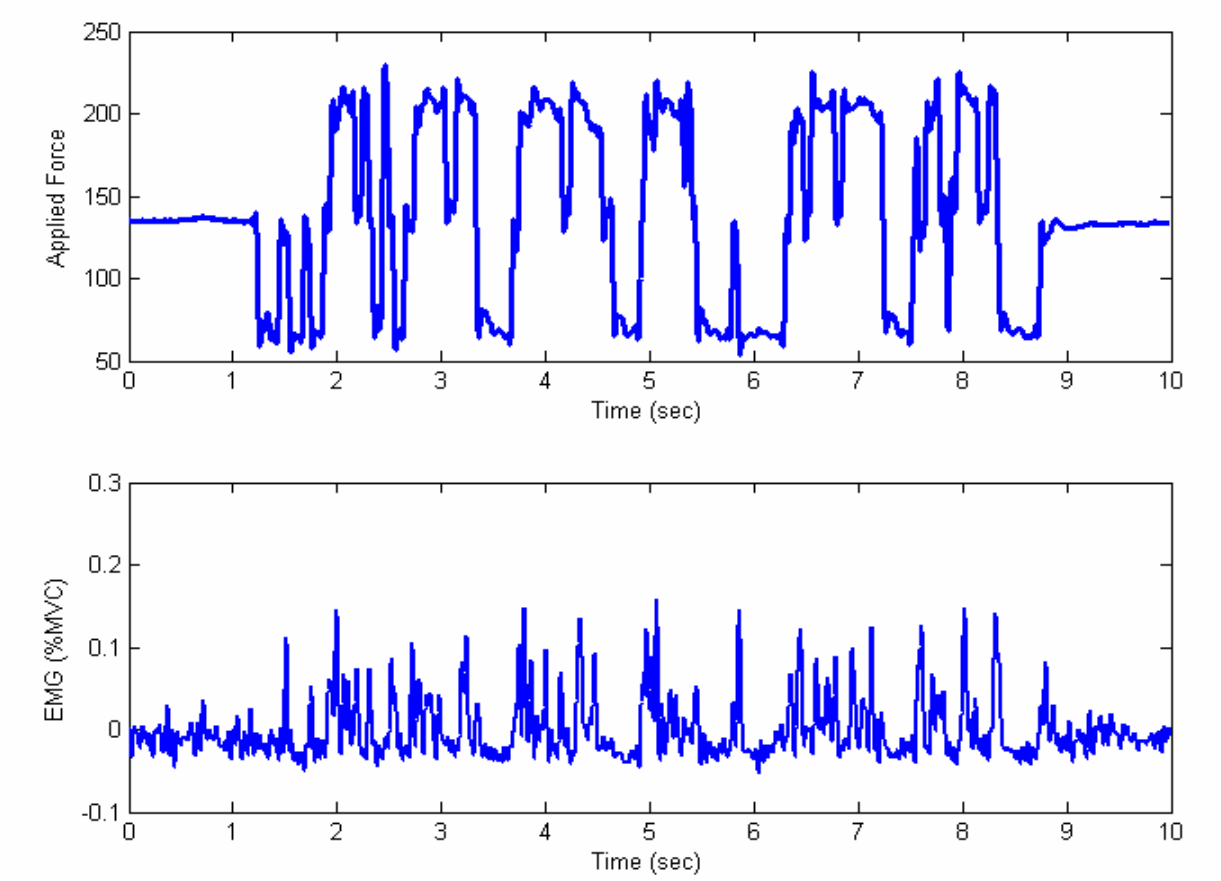

Figure 4.2. a) Applied force including preload and pseudorandom force perturbations. b) Rectified lumbar paraspinal muscle EMG data for a typical reflex trial.

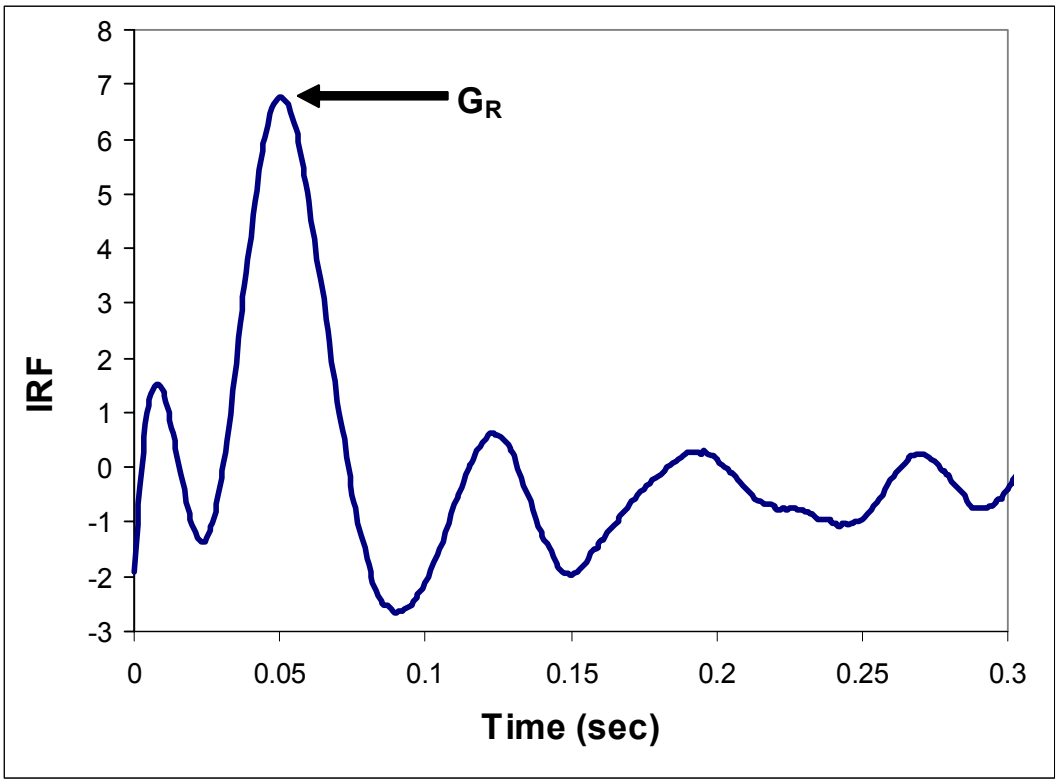

Figure 4.3. Typical Impulse Response Function (IRF) computed from applied trunk force (input) and rectified EMG (output). Reflex Gain, G_R was competed from the peak of the IRF Data at times greater than 120 msec were assumed arbitrarily voluntary and therefore appears as noise in the IRF.

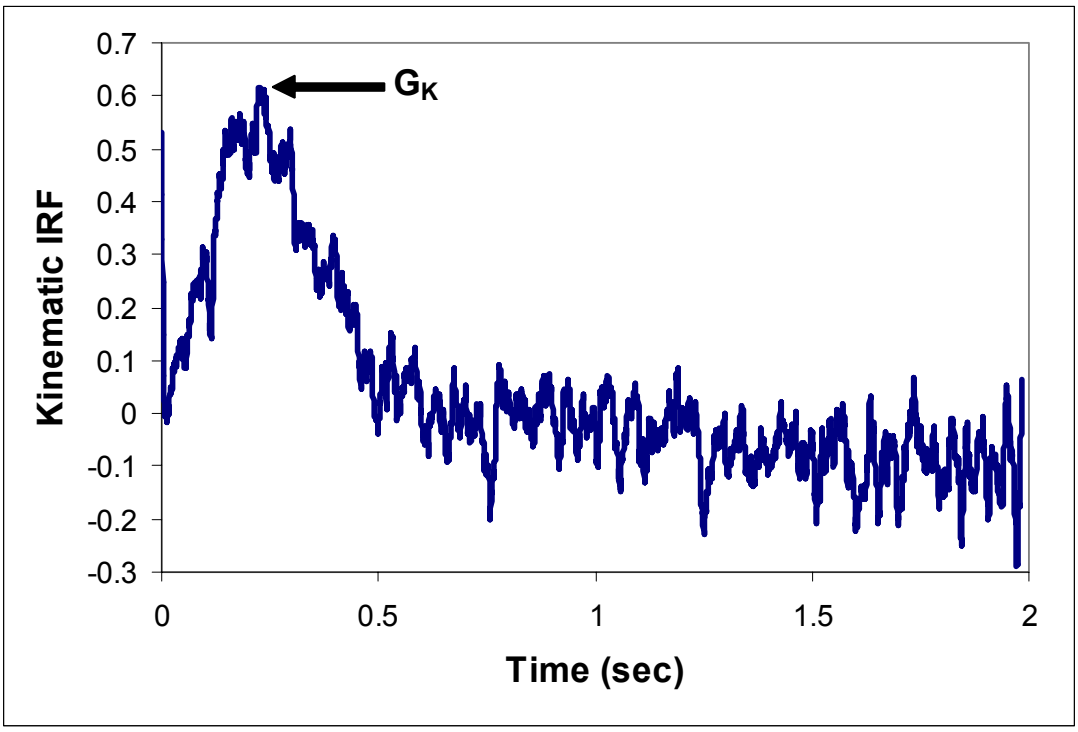

Figure 4.4. Typical Impulse Response Function computed from input applied trunk force and output T10 displacement. Kinematic Gain, G_K is labeled.

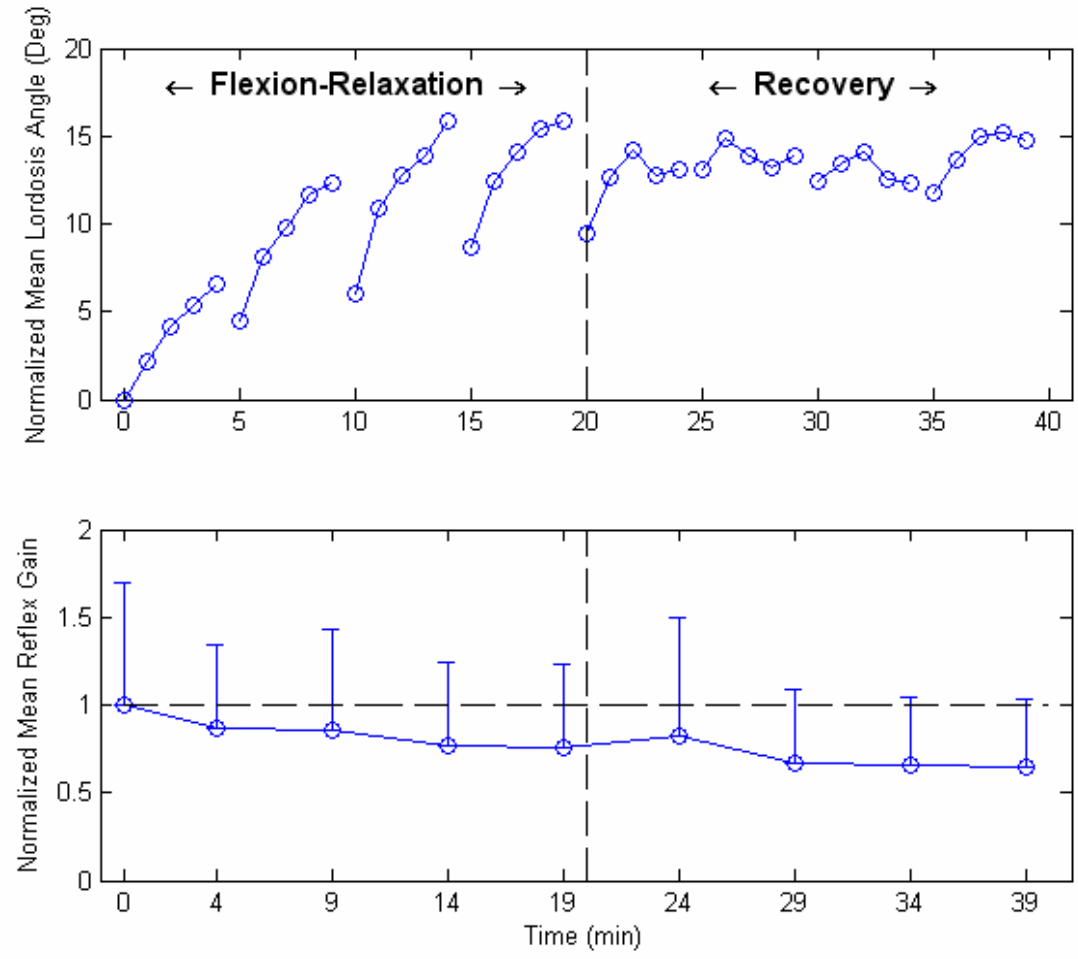

Figure 4.5. Progression of a) lordosis angle and b) reflex gain. In the lordosis angle plot, the four minute cycles are depicted by connecting lines, whereas the minute in between cycles is represented by a disconnect. Significant recovery of lordosis angle was observed during the minute between trials, but the recovery was incomplete. Reflex gain was analyzed at time T_0 (baseline measurement recorded before the first sFR cycle), T_{FR} (immediately following the fourth and final cycle of sFR) and T_R (immediately following the fourth and final cycle of recovery) and revealed a significant decline following the sFR trials.

Table 4.1. Mean (and standard deviation) of trunk angle, reflex gain, and kinematic gain. Reflex gain and kinematic gain were recorded in upright postures, while trunk and lordosis angles were recorded in fully flexed postures. Significant effects are labeled with superscripts.

Table 1.

θ_T (deg)	Men	Women	Combined
T_0	0 (0)	0 (0)	0 (0)
T_{FR}	4.40 (4.99) [2]	6.11 (4.04) [2]	5.33 (4.47) [1]
T_R	5.65 (5.87) [2]	8.24 (5.28) [2]	7.06 (5.58) [1]
Combined	3.35 (4.95)	4.28 (5.14)	4.13 (5.07)

θ_L (deg)	Men	Women	Combined
T_0	0 (0)	0 (0)	0 (0)
T_{FR}	17.45 (16.16) [2]	22.25 (11.99) [2]	20.33 (13.61) [1]
T_R	14.20 (15.30)	23.70 (17.66) [2]	19.9 (17.02) [1]
Combined	10.55 (14.51)	15.32 (16.26)	13.41 (15.63)

G_R	Men	Women	Combined
T_0	4.09 (2.21) [3]	6.03 (4.51) [3]	5.06 (3.64)
T_{FR}	3.65 (2.71)	4.31 (2.20) [2]	3.98 (2.46) [1]
T_R	2.83 (1.57)	3.68 (2.37) [2]	3.25 (2.03) [1]
Combined	3.52 (2.24)	4.67 (3.30)	4.10 (2.86)

G_K	Men	Women	Combined
T_0	0.566 (0.097)	0.611 (0.104)	0.589 (0.097)
T_{FR}	0.492 (0.073)	0.581 (0.091)	0.539 (0.093)
T_R	0.543 (0.145)	0.586 (0.073)	0.565 (0.112)
Combined	0.533 (0.107)	0.592 (0.088)	0.565 (0.101)

[1] significant effect of flexion-relaxation $p<0.05$

[2] significance due to FR within gender group $p<0.05$

[3] significance due to gender within time group $p<0.05$

CONCLUSIONS

Paraspinal reflexes following prolonged lumbar flexion-relaxation were examined in this study. Results of this study have demonstrated that paraspinal reflexes are disturbed due to prolonged flexion and that extended recovery is required to return reflexes to original levels. Reflex disturbance was attributed to creep deformation of passive spinal tissues.

Maintaining stability requires a highly coordinated and dynamic effort of many muscle groups which is achieved by the neuromuscular system. Disturbance of the motor control system can impair reflexive muscular action responsible for maintaining or restoring stability following an unexpected loading event. Therefore an inhibition of paraspinal reflexes relating to prolonged flexion-relaxation may be detrimental to stability. Though this study has provided insight into the mechanisms of stability, spinal stability was not quantified. To the author's knowledge there are no existing measures of spinal stability. The intent of this study was to show that paraspinal reflexes were disturbed by prolonged flexion. To relate these results to the motivation of the study, disturbance of paraspinal reflexes has been associated in a general way to spinal instability and low back pain. Results of the current study should be viewed in light of this limitation.

Prolonged trunk flexion has been associated with risk incidence rates of low back pain in the workplace. Results of this study indicate that inhibition of neuromuscular control may be related to low back pain following flexed posture work. Inhibition of neuromuscular control may therefore be at least partially responsible for high incidence rates of LBP in these occupations. Further studies should investigate this phenomenon further along with observed but as yet unexplained gender differences in reflex response to flexion-relaxation.

Appendix A – IRB approval

Virginia Tech
VIRGINIA POLYTECHNIC INSTITUTE AND STATE UNIVERSITY

Institutional Review Board

Dr. David M. Moore
IRB (Human Subjects)Chair
Assistant Vice President for Research Compliance
CVM Phase II- Duckpond Dr., Blacksburg, VA 24061-0442
Office: 540/231-4991; FAX: 540/231-6033
email: moored@vt.edu

DATE: December 22, 2004

MEMORANDUM

TO: Kevin P. Granata Engineering Science & Mechanics 0219

FROM: David Moore

SUBJECT: **IRB Expedited Continuation**: "Musculoskeletal Biomechanics of Movement and Control " IRB # 04-635 ref 03-632

This memo is regarding the above referenced protocol which was previously granted expedited approval by the IRB on January 21, 2004. The proposed research is eligible for expedited review according to the specifications authorized by 45 CFR 46.110 and 21 CFR 56.110. Pursuant to your request of last week, as Chair of the Virginia Tech Institutional Review Board, I have granted approval for extension of the study for a period of 12 months, effective as of January 21, 2005.

Approval of your research by the IRB provides the appropriate review as required by federal and state laws regarding human subject research. It is your responsibility to report to the IRB any adverse reactions that can be attributed to this study.

To continue the project past the 12-month approval period, a continuing review application must be submitted (30) days prior to the anniversary of the original approval date and a summary of the project to date must be provided. Our office will send you a reminder of this (60) days prior to the anniversary date.

Virginia Tech has an approved Federal Wide Assurance (FWA00000572, exp. 7/20/07) on file with OHRP, and its IRB Registration Number is IRB00000667.

Appendix B – Subject Consent Form

Title of Project: Musculoskeletal Biomechanics of Movement and Control
Investigators: E.L. Rogers, K.P. Granata

Purpose of this Research
To understand musculoskeletal injury and improve clinical diagnoses of injury it is necessary to understand how muscles control force and movement. The purpose of this study is to measure the relation between human movement, force generation and muscle activity. We are also interested in observing how gender, fatigue and physical conditioning influence these parameters. Throughout the course of this project more than 200 subject volunteers will participate including healthy individuals from the age of 18 to 55.

Procedures
We will tape adhesive markers and sensors on your skin around your trunk, legs and arms. These sensors are EMG electrodes that measure the activity of your muscles and position sensors to measure how you move. After some preliminary warm up stretches, we may ask you to push and/or pull as hard as you can against a resistance. We may then ask you to hold or lift a weight or weighted-box and to bend forward and back. We may also ask you to do some fatiguing exertions such as holding or lifting a heavy weight or pushing/pulling against a bar or cable for several minutes. We may also apply a quick but small force to record reflexes. You may be requested to return for repeated testing. Between test sessions you may be asked to participate in specified physical conditioning as per the American College of Sports Medicine recommended guidelines

Risks
The risks of this study are minor. They include a potential skin irritation to the adhesives used in the tape and electrode markers. You may also feel some temporary muscle soreness such as might occur after exercising. Subjects participating in physical conditioning may experience muscle soreness and/or musculoskeletal injury associated with inherent risks of cardiovascular, strength training and therapeutic exercise. To minimize these risks you will be asked to warm-up before the tasks and tell us if you are aware of any history of skin-reaction to tape, history of musculoskeletal injury, cardiovascular limitations.

Benefits
By participating in this study, you will help to increase our understanding musculoskeletal control of movement and musculoskeletal injury mechanisms. We hope to make this research experience interesting and enjoyable for you where you may learn experimental procedures in biomechanical sciences. We do not guarantee or promise that you will receive any of these benefits and no promise of benefits has been made to encourage your participation.

Anonymity and Confidentiality
Experimental data collected from your participation will be coded and matched to this consent form so only members of the research team can determine your identity. Your identity will not be divulged to unauthorized people or agencies. Digital video recorded during the experimental trials will be used to track the movement of the sensors by means of computer analyses and is insufficient video quality to observe individual participant identifying characteristics. Secondary VHS-style video may be recorded to validate the digital motion data. This camera angle is placed to avoid facial or other identifying characteristics. Sometimes it is necessary for an investigator to break confidentiality if a significant health or safety concern is perceived or the participant is believed to be a threat to himself/herself or others.

Compensation
Participants required to return for multiple test sessions or participate in physical conditioning for this protocol will receive payment per the number of test sessions as well as a bonus for full completion of the multi-session research protocol. Subjects participating in experiments as part of course or laboratory procedures will receive

appropriate credit for analysis of specified data as described in the course syllabus but not for personal performance during the experimental session. If course credit is involved and the subject chooses not to participate alternative means for earning equivalent credit will be established with the course instructor.

Freedom to Withdraw

You are free to withdraw from a study at any time without penalty. If you choose to withdraw, you will be compensated for the portion of the time of the study (if financial compensation is involved). If you choose to withdraw, you will not be penalized by reduction in points or grade in a course (if course credit is involved). You are free not to answer any questions or respond to experimental situations that they choose without penalty.

There may be circumstances under which the investigator may determine that you should not continue as a subject. You will be compensated for the portion of the project completed.

Approval of Research

This research project has been approved, as required, by the Institutional Review Board for Research Involving Human Subjects at Virginia Polytechnic Institute and State University, by the Department of Engineering science and Mechanics.

21 January 2003 — IRB Approval Date

20 January 2004 — Approval Expiration Date

Subject's Responsibilities

I voluntarily agree to participate in this study. I have the following responsibilities:

- Inform the investigators of all medical conditions that may influence performance or risk
- Comply to the best of my ability with the experimental and safety instructions
- Inform the investigator of any physical and mental discomfort resulting from the experimental protocol

Subject's Permission

I have read and understand the Informed Consent and conditions of this project. I have had all my questions answered. I hereby acknowledge the above and give my voluntary consent:

Subject Name (Print):_____

Subject signature:

Date

_____ Date_____

Witness (Optional except for certain classes of subjects)

Should I have any pertinent questions about this research or its conduct, and research subjects' rights, and whom to contact in the event of a research-related injury to the subject, I may contact:

Investigator(s): Ellen Rogers E-mail: elrogers@vt.edu Phone 231-2022

Faculty Advisor: K.P. Granata E-mail: Granata@vt.edu Phone 231-7039

_____ _____

Departmental Reviewer/Department Head — Telephone/e-mail

David M. Moore
Chair, IRB
Office of Research Compliance
Research & Graduate Studies

Subjects must be given a complete copy (or duplicate original) of the signed Informed Consent

Appendix C – Data Collection Forms

Data Collection Form for Study 1

EFFECTS OF STATIC FLEXION-RELAXATION ON PARASPINAL REFLEX BEHAVIOR

Subject #: 101

Max Exertions: s101max1…6

Before Flexion

Trial	Preload (N)	Comments	Actual File #
1	135		1
2	135		2
3	100		3
4	100		4
5	170		5
6	170		6
7	170		7
8	100		8
9	135		9

After Flexion

10	170		10
11	100		11
12	100		12
13	170		13
14	100		14
15	135		15
16	170		16
17	135		17
18	135		18

Data Collection Form for Study 2

DISTURBED PARASPINAL REFLEX FOLLOWING PROLONGED FLEXION-RELAXATION AND RECOVERY

Subject #: 201

Max Exertions: s201max1...6

Flexion

Protocol	Filename	Comments
Motor 0	s201str0	
	s201str1	
stretch 0	s201str2	
	s201str3	
	s201str4	
	s201str5	
	s201str6	
Motor 1	s201str7	
	s201str8	
stretch 1	s201str9	
	s201str10	
	s201str11	
	s201str12	
	s201str13	
Motor 2	s201str14	
	s201str15	
stretch 2	s201str16	
	s201str17	
	s201str18	
	s201str19	
	s201str20	
Motor 3	s201str21	
	s201str22	
stretch 3	s201str23	
	s201str24	
	s201str25	
	s201str26	
	s201str27	
Motor 4	s201str28	
	s201str29	

Recovery

Protocol	Filename	Comments
stretch 0	s201rec0	
	s201rec1	
	s201rec2	
	s201rec3	
	s201rec4	
Motor 1	s201rec5	
	s201rec6	
stretch 1	s201rec7	
	s201rec8	
	s201rec9	
	s201rec10	
	s201rec11	
Motor 2	s201rec12	
	s201rec13	
stretch 2	s201rec14	
	s201rec15	
	s201rec16	
	s201rec17	
	s201rec18	
Motor 3	s201rec19	
	s201rec20	
stretch 3	s201rec21	
	s201rec22	
	s201rec23	
	s201rec24	
	s201rec25	
Motor 4	s201rec26	
	s201rec27	

Appendix D – Raw Data

Raw Data – Study 1

Muscles: 8 = Left erector spinae
4 = Right erector spinae

Gender: 0 = Male
1 = Female

Reflex Gain			**Before Stretch**			**After Stretch**		
Muscle	**Gender**	**Subject**	**Preload (N)**			**Preload (N)**		
			100	**135**	**170**	**100**	**135**	**170**
8	0	5	2.30	2.30	2.51	0.60	0.85	0.72
8	0	5	1.43	2.68	1.58			
8	0	5	2.06	2.14	1.84			
4	0	5	1.21	2.95	2.04	1.93	1.24	1.54
4	0	5	1.39	2.30	1.43	1.01	2.07	1.70
4	0	5	1.09	1.66	1.56		1.53	1.60
8	0	6	0.75	1.09	1.04	1.31	0.78	0.97
8	0	6	0.74	1.02	1.16	1.16		1.61
8	0	6		0.57		0.80		1.45
4	0	6	1.12	1.58	1.08	1.16	0.94	1.04
4	0	6	0.96	1.23	1.03	1.32	1.29	1.91
4	0	6		1.09	1.20	0.81	1.50	1.51
8	0	8	0.52	0.72	1.07	0.72	1.01	0.62
8	0	8	0.45	0.52	0.58	0.67	1.19	
8	0	8	0.60	0.47	0.77	0.72	1.23	
4	0	8	0.46	0.51	0.71	0.67	0.66	0.49
4	0	8	0.53	0.60	0.58	0.53	0.59	0.63
4	0	8	0.54	0.54			0.63	0.65
8	0	10	1.28	0.92	1.33	1.02	0.70	1.03
8	0	10	1.57	2.13	1.33	0.80	1.28	1.29
8	0	10	2.05	2.10	0.89		0.97	
4	0	10	0.83	0.88	0.99	0.83	1.19	0.76
4	0	10	1.05	0.87	0.79		0.79	0.98
4	0	10	0.86	1.02			0.87	0.55
8	1	11	1.63	1.58	2.23	2.31	2.17	2.12
8	1	11	1.99	1.59	2.99	2.05	3.24	4.28
8	1	11	2.25		1.43	3.69	2.60	2.49
4	1	11	0.66	0.73	1.98	4.18	1.61	2.20
4	1	11	0.67	0.77	1.61	3.58	1.97	2.04
4	1	11			0.95		2.02	1.64

8	1	12	1.20	0.82	1.21	2.06		2.46
8	1	12	1.99	0.80	0.97		3.40	1.58
8	1	12	1.23	0.94				1.52
4	1	12	0.33	0.35	0.52	0.50	0.57	0.49
4	1	12	0.50				0.85	
4	1	12	0.40					
8	1	13	0.90	1.08	1.45	0.95	1.21	1.24
8	1	13	0.95	0.88	1.13	1.30	1.07	1.38
8	1	13	1.15	1.64	1.45	1.01		
4	1	13	0.61	0.77	0.57		0.73	
4	1	13	0.66	0.52	0.74	1.22	0.88	0.82
4	1	13	0.92	0.52		1.24		1.23
8	0	17	0.62	0.66	0.62	0.67	1.20	1.08
8	0	17		0.61	0.92	0.47	1.07	0.98
8	0	17	0.78	0.79	0.72		1.18	
4	0	17	0.96	1.76	0.92	1.20	1.37	1.19
4	0	17	2.04	1.04	1.71	1.34	2.12	0.97
4	0	17	1.57	0.97	1.59	0.99	0.74	
8	1	18	1.38	1.34	2.04	2.24	2.65	1.77
8	1	18	1.32	0.88	1.39	0.75	1.51	
8	1	18				0.78		
4	1	18	1.05	1.10	1.83	1.28	1.46	1.09
4	1	18	1.01	1.49	1.30	1.29	1.47	1.68
4	1	18		1.40	1.58	1.41	2.04	1.18
8	0	19	0.27	0.41	0.23	0.39	1.54	2.42
8	0	19	0.29	0.19	0.35	1.21		
8	0	19	0.31					
4	0	19	0.66	0.38	0.98	0.52	0.76	0.58
4	0	19	0.89	0.33	0.96	0.80	0.26	0.65
4	0	19				0.62	0.58	
8	1	20	0.74	0.66	0.39	0.49	0.98	0.39
8	1	20	0.31					
8	1	20	0.43					
4	1	20	0.60	0.70	0.47	0.53	0.34	0.33
4	1	20	0.29		0.50		0.50	0.40
4	1	20					0.48	
8	0	21	1.04	1.12	1.17	1.14	0.97	1.42
8	0	21	0.92	1.34	1.10	0.92	0.97	1.06
8	0	21	1.52		1.03			0.97

4	0	21	0.87	0.92	0.97	1.46	0.56	1.19
4	0	21	0.87	0.94	0.79	0.72	0.79	0.53
4	0	21						
8	1	22	1.41	1.00	2.05	1.56	2.71	3.46
8	1	22		1.28	1.50	1.68	1.02	1.89
8	1	22		1.02		1.68		2.96
4	1	22	2.03	1.36	2.04	1.44	1.46	1.80
4	1	22		1.51	1.34	1.64	1.40	1.17
4	1	22						
8	1	23	2.22	1.61		3.39	2.70	2.79
8	1	23	1.84		2.88	3.43	2.37	3.07
8	1	23	2.12	2.91	2.46	3.48	2.45	2.41
4	1	23	1.42	1.39	2.13	1.61	1.25	3.00
4	1	23	0.74	1.61	1.73	1.57	1.89	2.89
4	1	23	1.24	1.89	1.83	1.44	1.72	2.76
8	1	24	2.28	2.33	2.16	1.49	1.08	1.95
8	1	24	1.61	2.01	2.26	1.82	1.59	
8	1	24	1.89	2.47		1.14		
4	1	24		1.30	1.19	1.61	1.72	1.05
4	1	24	1.20	1.10	1.79	1.26	0.86	1.63
4	1	24	0.96	0.92	1.00	1.00	1.54	1.86
8	0	28	1.37	0.81	1.22	0.70	1.24	0.56
8	0	28	1.34	0.98	1.44	0.64	0.81	1.15
8	0	28	1.12	0.93	1.02		0.81	0.89
4	0	28	1.71	1.05	1.47	1.01	1.26	1.27
4	0	28	1.92	1.06	1.57	0.78	0.85	0.93
4	0	28	1.53	1.02	1.45		0.69	
8	0	29	0.75	0.97	1.03	0.91	1.22	1.14
8	0	29	1.24	1.07	1.04	1.35	0.57	1.38
8	0	29	0.65		1.19	1.31		
4	0	29	0.93	0.99	0.90	1.25	1.68	1.49
4	0	29		0.92	1.74	1.48		1.57
4	0	29		0.99	1.07	1.86		
8	1	30	1.77	1.57	2.77	3.78	2.72	3.03
8	1	30	2.09		2.49	3.08	2.55	4.35
8	1	30		2.86	2.76	2.41	3.25	3.56
4	1	30		3.46	1.84			6.98
4	1	30		3.49	2.57			
4	1	30						

Reflex Delay

Muscle	Gender	Subject	Before Stretch Preload (N)			After Stretch Preload (N)		
			100	135	170	100	135	170
8	0	5	85	75	71	69	67	58
8	0	5	80	79	77			
8	0	5	75	78	82			
4	0	5	65	75	69	72	65	75
4	0	5	71	71	79	74	72	77
4	0	5	71	76	69		76	72
8	0	6	102	56	56	58	117	61
8	0	6	110	50		61		66
8	0	6				54		31
4	0	6	58	64	61	59	63	109
4	0	6	123	55	67	56	62	73
4	0	6		63	54	114	54	23
8	0	8	62	108	69	67	57	65
8	0	8	111	66	60	58	51	
8	0	8	68	106	66	53	60	
4	0	8	60	64	67	57	62	60
4	0	8	56	55	64	57	54	64
4	0	8	53	54				65
8	0	10	80	69	62	75	75	73
8	0	10	76	74	59	65	64	70
8	0	10	72	76	74		65	
4	0	10	75	77	71	78	77	64
4	0	10	73	72	78		68	67
4	0	10	79	78			76	75
8	1	11	58	62	65	60	59	62
8	1	11	61	97	60	58	68	60
8	1	11	59		49	56	70	69
4	1	11	67	54	65	56	54	65
4	1	11	55	81	56	57	53	66
4	1	11			64		63	62
8	1	12	53	54	59	109	68	49
8	1	12	59	67	51		55	107
8	1	12	48	60				62
4	1	12	110	65	54	108	61	54
4	1	12	61				58	
4	1	12	49					

8	1	13	65	60	69	80	67	59
8	1	13	63	72	66	64	69	68
8	1	13	67	66	65	71		
4	1	13	62	60	65		60	
4	1	13	60	65	60	55	62	52
4	1	13	64	68		63		61
8	0	17	77	78	82	82	78	84
8	0	17	79	83	77	76	80	79
8	0	17	80	79	82		82	
4	0	17	77	79	80	78	79	81
4	0	17	77	80	77	79	76	87
4	0	17		80	78	85	77	
8	1	18	65	65	68	69	69	69
8	1	18	73	58	72	73	67	
8	1	18				69		
4	1	18	68	72	73	67	70	66
4	1	18	72	68	74	74	70	74
4	1	18	71	73	70	77	71	68
8	0	19	20	21	48	42	84	28
8	0	19	42	38	57	32		
8	0	19	41					
4	0	19	56	65	57	58	54	99
4	0	19	24	71	113	43	60	112
4	0	19				60	64	
8	1	20	57	54	41	69	60	42
8	1	20	66					
8	1	20	75					
4	1	20	54	59	88	59	60	42
4	1	20	70		76		59	57
4	1	20					46	
8	0	21	88	69	63	51	118	53
8	0	21	56	69	97	64	50	55
8	0	21	115		63			66
4	0	21	104	62	77	102	81	95
4	0	21	101	87	88	104	116	61
4	0	21						
8	1	22	80	78	78	65	71	75
8	1	22		80	73	82	71	69
8	1	22		87		75		76

4	1	22	73	85	67	71	54	72
4	1	22		79	74	44	83	80
4	1	22						
8	1	23	75	72		78	67	66
8	1	23	79		67	69	67	68
8	1	23	65	65	65	74	67	77
4	1	23	64	68	63	78	84	67
4	1	23	65	76	66	73	73	69
4	1	23	70	65	71	74	84	73
8	1	24	66	59	64	63	60	60
8	1	24	66	64	59	63	64	
8	1	24	54	68		59		
4	1	24		63	61	68	61	70
4	1	24	68	65	60	68	67	71
4	1	24	64	64	60	63	69	67
8	0	28	65	48	58	61	67	72
8	0	28	58	61	59	65	55	59
8	0	28	55	60	55		62	62
4	0	28	67	56	54	56	61	61
4	0	28	60	47	58	65	60	64
4	0	28	55	58	57		65	
8	0	29	60	45	96	55	55	47
8	0	29	45	54	51	53	58	49
8	0	29	98		50	53		
4	0	29	105	53	59	105	52	46
4	0	29		54	53	53		49
4	0	29		56	51	54		
8	1	30	60	46	58	58	58	59
8	1	30	59		62	55	59	59
8	1	30		56	59	70	60	60
4	1	30		63	59			
4	1	30		54	52			
4	1	30						

Raw Data – Study 2

Muscles: 10 = Left erector spinae
6 = Right erector spinae
Gender: 0 = Male
1 = Female

Reflex Gain

Gender	Subject	Muscle	T0	T1	T2	T3	T4	T5	T6	T7	T8
0	201	6	3.90	4.56	6.11	7.13	4.93	7.39	5.21	5.77	5.17
0	201	10	6.92	4.68	6.40	5.25	3.62	4.04	4.74	5.75	4.75
0	202	6	2.42	2.60	4.31	3.91	1.91	2.64	1.85	3.92	3.89
0	202	10	2.89	3.65	4.16	3.38	2.28	0.52	0.39	0.33	0.73
0	203	6	3.18		0.91	1.12	1.00	1.30	1.45	1.61	1.53
0	203	10	4.10	4.11	3.98	7.00	7.85	7.21	4.07	4.22	3.55
1	205	6	5.59	6.57	5.18	5.02	5.32	4.07	3.63	5.21	6.68
1	205	10	3.95	3.47	5.47	5.37	3.70	3.12	3.57	5.43	6.06
0	206	6	2.81	4.48	4.09			3.31	1.75		0.71
0	206	10	3.02	4.44	5.52	2.32	2.63	2.59	1.26	0.94	1.69
0	207	6	2.28	2.53	2.23	1.95	1.43		2.69	2.07	2.20
0	207	10	5.14	5.61	6.10	4.95	5.05	3.61	4.43	3.14	3.33
0	208	6	2.18	3.84	3.11	1.72	2.30	2.97	3.94	2.36	2.08
0	208	10	2.31	2.27	2.03	2.01	1.97	1.97	1.94		1.40
1	209	6	0.40	0.71				2.34			
1	209	10	0.83			0.14	0.48	0.58		1.28	1.81
1	210	6	3.73	5.60		4.02		4.67	8.21	2.01	
1	210	10	2.88	5.31	1.98	2.52	3.63	2.35	2.22	1.71	1.36
0	213	6	2.32	2.27	2.22	1.88	2.26	2.35	1.96	2.06	2.44
0	213	10	2.81	5.15	4.26	4.59	3.68	5.97	3.72	5.07	7.39
0	214	6	5.22	6.26	6.78	6.83	7.23		6.61		
0	214	10	9.41	10.99	16.30	12.07	12.22		7.19		4.47
1	215	6	2.72	0.97	2.38	2.01	2.41	2.23	3.31	2.64	2.20
1	215	10	7.21	2.79							8.61
1	216	6	6.84	6.02	7.95	6.66	8.38	5.98	5.51	5.06	5.69
1	216	10	6.74	4.15	6.29	6.19	5.79	5.00	4.16	3.95	4.07
0	217	6	3.36	2.67	2.16	1.87	2.20	2.60	3.88	3.17	1.62
0	217	10	5.81	3.75	4.06	4.00	3.54	5.04	7.22	4.44	3.60
1	218	6	5.79	4.19	5.31	3.51	2.31	1.42	1.70	1.92	1.11
1	218	10	4.42	2.99	4.74	4.10	3.50	2.33	0.72	0.92	1.48
1	220	6									
1	220	10	0.59	2.65	2.60	2.05	1.84	1.86	1.89	2.04	1.70
1	221	6	17.10	5.46	3.75	2.61	5.09	3.13	5.49	7.75	6.38
1	221	10	12.73	11.68	10.50	4.82	8.08	7.33	8.30	7.22	7.65
1	223	6	3.44	8.97	5.62	5.01	4.80	2.44		2.53	

1	223	10	4.13	4.00	1.96	1.82	1.54	1.11	0.66	0.40	0.63
0	224	6	8.51	8.91	4.12	9.51	5.00	10.18	7.60	4.86	6.00
0	224	10	6.67	7.15	6.24	3.90	6.21	4.91	4.41		
1	227	6	3.01	3.94	2.73	3.33	4.60	3.89	2.34	3.83	3.80
1	227	10	14.46	9.00	8.71	6.57	6.75	15.11	4.58	4.40	5.61
1	228	6	6.24	2.83	2.80	2.92	2.73	2.52	2.32	2.51	4.02
1	228	10	7.35	7.18	3.93	6.38	5.59	14.44	6.80	8.19	7.61
0	229	6		2.86	2.54	3.04	2.22	2.26	2.97		
0	229	10	3.03	2.43	1.76	1.63	2.23	1.40	1.61	1.92	1.70
1	230	6	8.16	14.21	7.61	8.68	14.77	8.57	6.90	13.54	9.01
1	230	10	11.03		13.38		19.69	23.40	13.99		15.50

Reflex Delay

Gender	Subject	Muscle	T0	T1	T2	T3	T4	T5	T6	T7	T8
0	201	6	59	55	57	52	53	54	54	59	58
0	201	10	52	60	52	53	55	55	59	57	49
0	202	6	57	23	55	57	56	56	54	52	59
0	202	10	51	54	57	54	47	48	51	44	58
0	203	6	44		51	53	47	51	50	59	59
0	203	10	51	70	61	64	61	59	64	59	62
1	205	6	60	56	52	63	56	59	61	51	45
1	205	10	55	54	55	57	45	91	56	71	33
0	206	6	62	51	47			62	62		56
0	206	10	61	62	58	59	60	72	69	67	71
0	207	6	63	55	53	56	53		54	55	57
0	207	10	61	63	57	56	56	55	58	61	60
0	208	6	55	51	51	53	33	26	39	43	55
0	208	10	68	58	62	62	63	63	63		56
1	209	6	42	49				51			
1	209	10	26			51	33	66		61	68
1	210	6	60	64		27		32	20	95	0
1	210	10	94	66	52	67	60	102	60	60	66
0	213	6	49	47	55	49	56	56	52	49	44
0	213	10	56	56	56	54	57	55	60	58	56
0	214	6	65	65	71	65	69		65		
0	214	10	69	66	65	62	69		62		17
1	215	6	67	65	30	36	35	34	41	41	46
1	215	10	55	52							56
1	216	6	45	50	50	55	57	56	62	53	54
1	216	10	51	55	52	53	55	52	55	59	48
0	217	6	40	40	47	46	38	47	51	40	51
0	217	10	52	46	51	49	52	51	49	50	51
1	218	6	66	63	61	65	65	67	26	70	67
1	218	10	56	55	54	53	62	52	44	49	51
1	220	6									
1	220	10	57	58	53	68	55	58	65	98	68
1	221	6	55	57	63	33	54	35	57	62	59
1	221	10	58	56	57	60	57	56	57	63	54
1	223	6	47	60	58	58	64	57		47	
1	223	10	60	62	61	59	62	62	61	58	61

0	224	6	53	50	49	47	60	52	50	49	54
0	224	10	57.5	54	57	55	50	54	57		
1	227	6	66	64	62	61	64	63	59	62	68
1	227	10	59	56	52	60	62	29	53	59	62
1	228	6	52	48	48	50	53	47	47	52	44
1	228	10	56	50	52	50	49	49	59	39	51
0	229	6		44	51	51	47	45	46		
0	229	10	47	47	53	48	49	48	49	49	49
1	230	6	73	38	36	32	44	52	29	39	67
1	230	10	57		21		31	62	43		58

Vita

Ellen Louise Rogers

Ellen L. Rogers was born in Lansing, MI on August 17, 1981 to James and Valerie Rogers. Ellen attended Wilde Lake High School in Columbia, MD, graduating in May, 1999. Next she attended Lafayette College, where she graduated summa cum laude with a B.S. degree in Mechanical Engineering in May, 2003. Ellen went on to graduate school at Virginia Polytechnic and State University (Virginia Tech), finishing her M.S. in Mechanical Engineering in May, 2005. Working as a graduate research assistant in the Musculoskeletal Biomechanics Laboratory at Virginia Tech, Ellen's research focused on studying changes in low-back reflexes following prolonged flexion. Ellen's research has been presented at a national ASB conference and published in the journal Clinical Biomechanics. During her free time, Ellen enjoys swimming with the Blacksburg Masters swim team, reading, and participating in outdoor sports such as hiking, biking, running and skiing.