

BIOMECHANICAL EFFECTS OF CYCLIC LOADING ON THE LOWER BACK

A Thesis

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By

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*Dedicated to my father, the late Rodney “Rocky” Claude Sr. and my loving mother
Deborah Claude for supporting me in every pursuit throughout life.*

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ABSTRACT

More than 30 million people in the United States may have low back pain at any time, and 10 million of them have chronic symptoms. Epidemiological studies indicate that along with axial compressive loads, other factors including repetitive twisting or lateral bending and lifting are significant risk factors for low-back disorders. Literature repeatedly confirms that cyclic occupational functions expose workers to a 10-fold increase in episodes of low back injury and pain. This study examined the biomechanical effects of cyclical loading on the lower back. Twenty *in vivo* feline preparations were subjected to passive cyclic loading at 20 N (n=6), 40 N (n=7), and 60 N (n=7) for 20 minutes continuously, followed by 7 hours of rest. The skin over the lumbar spine was dissected from the thoracic level to the sacral level and reflected laterally to expose the dorsolumbar fascia. Six pairs of stainless steel fine wire electromyography (EMG) electrodes were inserted into the multifidus muscles of the L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 on the right side. An “S” shaped stainless steel hook was inserted around the middle of the supraspinous ligament of the L-4/L5 motion segment and connected to the vertical actuator of a Bionic 858 Material Testing System. The load was applied by the MTS actuator with a computer controlled loading system operated in a load control mode; the resulting electrical activity was recorded and analyzed. Results showed that continual cyclical loading on the supraspinous ligament and lumbar spine resulted in creep or laxity within the viscoelastic structures of the spine. The creep then caused desensitization of the mechanoreceptors, located within the ligament. The initial response, due to a decrease in mechanoreceptor sensitivity, was an exponential decrease of electrical activity during the 20-minute loading period for 20N,

40N, and 60N. The greatest percentage of recovery was observed 10 minutes immediately following the loading period for 20N, 40N, and 60N. The electrical activity for all loads increased near the end of recovery. Full recovery of reflexive muscular activity was never observed during any loading period.

CHAPTER 1-INTRODUCTION

More than 30 million people in the United States may have low back pain at any time, and 10 million of them have chronic symptoms (Panjabi, 1996). From 1993 to 1999, the U.S department of Labor, Bureau of Labor Statistics reported that four out of ten injuries and illnesses resulting in time away from work were sprains and strains, most often involving back pain (BLS, 1995). According to the National Institute of Occupational Safety and Health (NIOSH), for injury and illness cases involving days away from work, approximately 706,000 cases resulted from overexertion and repetitive motion (Keyserling, 2000). Also, approximately 530,000 lost work-time cases were associated with manual materials handling activities such as lifting, pushing, pulling and carrying and over 60% of these cases involved back pain (Keyserling, 2000).

Although our society is increasingly post-industrial, with less heavy labor and more automation and robotics, disability due to by low back pain has steadily risen. Fortunately, most back-pain patients will recover substantially even with severe pain. However, the recurrences are common and the majority of patients experience them. Contributing biomechanical factors to low-back pain are exposure to repetitive, static and vibratory activities (Kumar, 2001). This study focuses on the impact of cyclic lumbar loading on the development of low back pain in the lumbar spine.

The spinal stabilizing system can be divided into three subsystems: the spinal column, spinal muscles surrounding the spinal column, and the control unit. White et al. (1978) defines spinal stability as the spine's ability to maintain its patterns of displacement under physiologic loads so that there is no initial or additional neurologic deficit, no major deformity, and no incapacitating pain. To relay a clear understanding of

spinal stability, Panjabi used the “ball in a bowl” as an analogy of the load displacement curve, with the shape of the bowl indicating spinal stability (Panjabi, 1996). A deeper bowl represents a more stable spine while a more shallow bowl represents a less stable spine. Panjabi hypothesized that for someone without spine injury there is a normal neutral zone and range of motion and in turn, no spinal pain. He defined the neutral zone as that part of the range of motion where there is minimal resistance to intervertebral motion. In this instance, the bowl is not too deep or too shallow. However, when an injury occurs to an anatomical structure, the neutral zone of the spinal column increases and the ball moves freely over a larger distance. According to the analogy, this bowl would be more shallow. As a consequence, pain results from this combination. The spinal stabilizing system may then react by actively limiting the neutral zone via activation of the muscles.

The isolated spine cannot withstand a large amount of force before it buckles. Cholewicki et al. (1991) reported that the isolated thoracolumbar spine buckles under compressive loads exceeding 20N and the lumbar part of the spine buckles under approximately 90N. In vivo a spine may experience compressive loads ranging from about 6000N (McGill et al., 1986) for more demanding everyday tasks and up to 18000N during competitive power lifting (Cholewicki et al., 1991). The musculature significantly increases the spine’s ability to remain stable under large loads. The activation of both the agonist and antagonist muscles seems to stiffen spinal segments, as necessary, to maintain stability under external or internal loading (Granata et al., 1995). Spinal compression causes spinal instability and is traditionally assumed the principal biomechanical mechanism associated with occupationally related low back disorders

(Granata et al., 1999). Due to the fact that NIOSH lifting guides base safe and hazardous tasks on static estimates of compressive loads, research examining the risk of low-back pain often focuses on axial compressive loads associated with occupational tasks. However, epidemiological studies indicate that other factors, including repetitive twisting or lateral bending and lifting are significant risk factors for low-back disorders. Literature repeatedly confirms that cyclic occupational functions expose workers to a 10-fold increase in episodes of low back injury and pain (McGill et al., 1986). The injury often occurs after the work is completed while they are performing simple, unloaded movements. Though the onset of low back pain is sometimes associated with sudden injury, it is probably the result of cumulative damage of the spinal components often associated with chronic loading. Pain arises from any neurally innervated structure. All paraspinal muscles and all non-muscle paraspinal tissues are neurally innervated (Kang et al., 2001).

In the past, ligamentous structures were considered to be the primary restraints of most of the major joints (Hirokawa et al., 1991). However, literature has repeatedly shown musculature to be the major stabilizing force of the spine (White et al., 1978; Gedalia et al., 1999). Ligaments are endowed with sensory receptors and research has shown that ligament loading leads to protective contraction of the multifidus muscle (Solomonow et al., 1998). Research has also shown that laxity within these structures may cause desensitization of the sensory receptors within. Previous work demonstrated that laxity is induced due to both passive cyclic (Solomonow et al., 1999; Gedalia et al., 1999) and static (Williams et al., 2000; Jackson et al., 2001) constant displacement. In some of these studies, both the loading and recovery periods were examined. The rest

period required for full recovery of the muscle activity has not yet been determined after undergoing passive static or cyclic loading. McGill and Brown (1992) demonstrated that the laxity induced in viscoelastic tissues is exponential and the recovery is much longer than the loading interval. This thesis will 1) Determine the behavior of reflexive muscular activity after various magnitudes of cyclic loading under load control and seven hours of rest, 2) Assess the development of creep in the spine's viscoelastic structures, and 3) Develop a model for both the loading period and following rest. The goal of this research is to provide deeper insight into the biomechanical behavior of the lumbar spine during cyclic loading within the physiological range.

CHAPTER 2- BACKGROUND AND LITERATURE REVIEW

Low back pain is an extreme socioeconomic problem and industry is flooded with workers who perform manual lifting tasks which lead to low back pain. In 1999, Solomonow et al. demonstrated that long durations of cyclical loading may expose the spine to injury. Each of the preparations in the study underwent cyclical loading for 50 minutes, 10 minutes of rest, and a final 50 minutes of cyclic loading to assess recovery of the muscular activity. The results show that creep was induced in the ligaments, discs and capsules of the spine. The supraspinous ligament creep was denoted by an elongation of the ligament, while the intervertebrae disc creep was denoted by a decrease in the disc fluidity and deformation in the collagenous structure of the disc. Not only does this creep occur under long duration but, Solomonow, M, Baratta, R., et al. (in press) demonstrated creep development during 10 minutes of static lumbar flexion in humans. Spasms, which indicate damage in the viscoelastic tissues, were present in the EMG of both the male and females in this study. The muscles compensated for the decrease in the ability of the viscoelastic tissues to generate the passive forces and aid in spinal stability. The muscle activity increased proportionately with respect to creep in the viscoelastic structures. Chu et al. (2003) showed a similar phenomena within the anterior cruciate ligament of the human. This study, which examined knee flexion and extension at various degrees, showed that a neuromuscular disorder may develop from ligament creep. This disorder consisted of spasms, increased electromyography and force of the agonist muscle and less help from the antagonist muscle.

In 1998, Solomonow et al. studied the link between the mechanoreceptors located within the supraspinous ligament and multifidus muscle contraction. They reported

electromyographic activity of the multifidus muscles when mechanoreceptors in the supraspinous ligament of the cat were stimulated. From this, they determined that there is a ligamento-muscular reflex arc from mechanoreceptors in the ligaments to muscles that, on activation, develop forces that stabilize the spine. As stated earlier, creep within the viscoelastic structures cause desensitization of the mechanoreceptors, which serve the major purpose of signaling the Central Nervous System that the spine needs support. The relevance of the Solomonow et al. (1999) study was to show that the mechanoreceptors response to the Central Nervous System declined exponentially as the structures continuously underwent creep. Before fatigue occurred within the multifidus, the muscle activity decreased exponentially and the spine was exposed to injury and instability.

Therefore, it is fair to say that the reflex muscular activation by receptors within the ligaments, capsule and disc bears a major responsibility for maintaining ongoing spine stability. Overall, the musculature has been most noted for generating forces which maintain spinal stability under diverse conditions (Panjabi, 1996). Although the muscles are the major stabilizing forces of the spine, the ligament's role is very important. The spine's stability is maintained by forces generated by passive viscoelastic structures (ligaments, discs, capsules) and by active forces generated from muscular contractions (Panjabi, 1996; Granata et al., 1995).

2.1 Anatomy of Lower Back

●Human Vertebral Column

The vertebral column or spinal column of the human is formed by a series of 32 bones called vertebrae. These vertebrae each belong to one of five groups of the spinal column. The first group is the cervical spine which consists of eight vertebrae, next is the

thoracic spine which has twelve vertebrae, then the lumbar spine which has five vertebrae, then five in the sacral and, lastly, one to two in the coccygeal. The first three groups of vertebrae are known as the movable vertebrae and the last two groups are the fixed vertebrae.

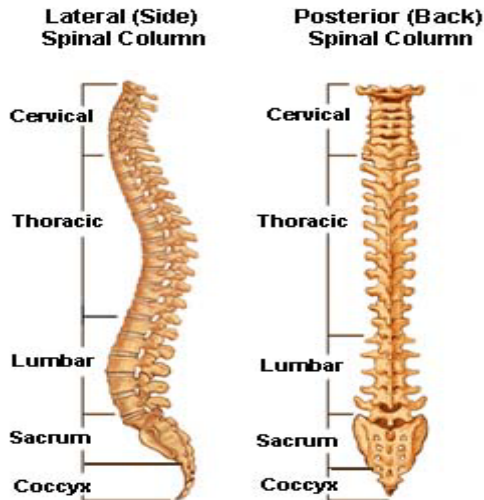


Figure 1. Spinal Column of Human

- Intervertebral Discs

The intervertebral discs exist between two adjacent moveable vertebrae. They contribute to the spinal column's stability because they are strongly bound to the vertebrae while still allowing considerable movement between the adjoining bones. The discs allow extension and flexion of the vertebral column. They make up about one fourth of the spinal column length and they serve as shock absorbers which protect the vertebrae, brain, and other structures. The outer layer of the discs is composed of fibrocartilage, while the inner core is composed of the highly elastic gelatinous substance called nucleus pulposus.

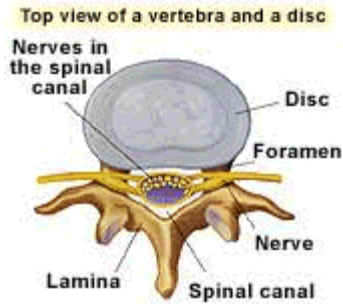


Figure 2. Top view of a vertebra and a disc

- Fascia

This is the term commonly used to refer to all fibrous connective tissue not specifically organized as tendons or ligaments. It is usually in the form of a membranous sheet and it varies in thickness according to its functional demands.

- Ligaments and Tendons

The principal constituent forming both ligaments and tendons is a dense regular connective tissue. These structures are pliable and have the capability to withstand great tensional stress in one direction. The ligaments connect bone to bone and their purpose is to strengthen the joints and in turn, restrain abnormal movements. However, they do allow freedom of movement of the joint in its normal range of motion. The tendons connect muscles to bones and have various lengths and thicknesses.

- Supraspinous Ligament

The supraspinous ligament is a strong fibrous cord which connects the spinous processes from the fifth cervical vertebra to the sacrum. It is thicker and broader in the lumbar region than in the thoracic region. The superficial fibers of this ligament extend over three or four vertebra; the deeper fibers pass between two or three vertebra; and the

deepest connect the spinous processes of two neighboring vertebra and fuse in with the interspinal ligament.

- Multifidus Muscle

The Multifidus muscle acts to extend the vertebral column as well as rotating it to the opposite side. It exists on both sides of the spinous processes of the vertebrae from the sacrum to the axis. It lies deeper in the lumbar and thoracic region than in the cervical region.

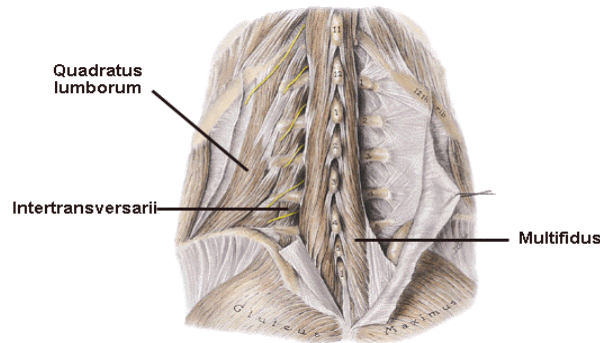


Figure 3. Multifidus muscle along the five lumbar vertebrae in a human

2.2 Basic Functions of Muscles

The structural unit of contraction is the muscle fiber. It ranges from a few millimeters to 30 cm and a diameter of 10 to 100 micrometers and upon contraction it will shorten up to 57% of its resting length (Basmajian et al., 1985). However, the muscle fiber must get its fuel from somewhere to perform these actions. The motor unit is what allows the muscle fiber to contract. It is the single smallest controllable functional unit, consisting of a single α motor neuron, the axon which runs down the

motor nerve and its terminal branches, and all of the muscle fibers that these branches innervate. The ultimate response is relayed back to the spinal cord.

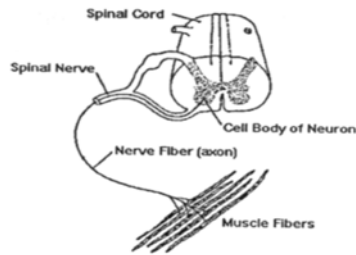


Figure 4. The Motor Unit

Under normal conditions, an action potential travels down the motor neuron axon and activates all of the muscle fibers of the motor unit (Paton et al., 1967). The motor unit obeys the all or none law, meaning that all of the muscle fibers connected to that motor unit contract or none contract. Muscle contraction generates ion movement across the muscle cell membrane which produces an electromagnetic field. This electromagnetic field, which is known as the muscle fiber action potential, can be recorded using surface, needle or wire electrodes. The summation of each individual muscle fiber action potentials is the motor unit action potential (MUAP).

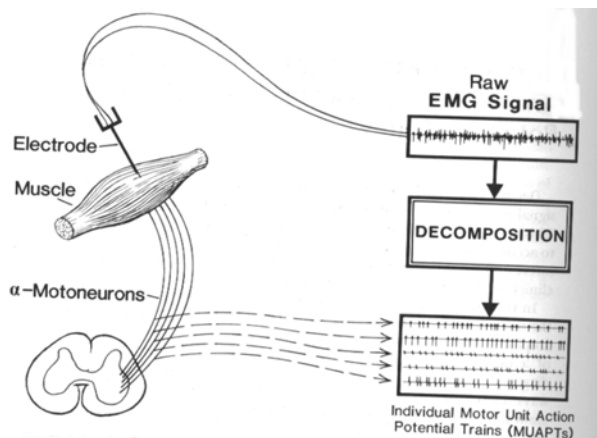


Figure 5. Collection process of Motor Unit Action Potential (MUAP)

Motor units fire randomly, all having their own amplitudes, duration and waveform. The muscle fibers from many motor units are designated a given recording area and the electromyographic (EMG) signal is the sum of all of the detected signals.

2.3 Recording Techniques

The EMG collection system consists of electrodes, amplifiers, filters, and an acquisition device. An electrode is “a device through which an electrical current enters or leaves an electrolyte; i.e. the electrode is the site of connection between the body and the collection system” (Acierno et al., 1995). The function of wire electrodes will be the focus since this is the EMG measuring device in this study. Wire electrodes were used because they are readily available and because, they have a relatively small pick up area which enables the electrode to detect individual motor unit action potentials. Wire electrodes use an insulate wire inserted into a hypodermic needle. The needle is used for inserting the wire into the desired muscle area. The tip of the wire is bare and serves as the detection area.

Basmajian et al. (1985) defines the EMG signal as "the electrical manifestation of the neuromuscular activation associated with a contracting muscle." Impedance to this signal varies with electrode type, size, and location. Noise from outside sources can contaminate the EMG signal. Various biological tissues may impose impedance to the transmission of the electrical signal. Needle and wire electrodes have less impedance than surface electrodes.

2.4 Bipeds vs. Quadrupeds

A Biped is a two-foot human or animal with five lumbar vertebrae and the gravity vector parallel to the spine. A quadruped is a four-footed animal with seven lumbar

vertebrae and gravity vector perpendicular to the spine. It is important to note that a quadruped or feline model, which consists of seven lumbar vertebrae, will be used in this study. This model will be used because there is a common relationship between the viscoelastic properties of the feline's tissues and the biped's tissues. Also, there is a similarity between the neuromuscular system within both the biped and the quadruped. Wirth et al. (2001) states that the advantage of using an anesthetized animal model is the ability to control the stimulation and dissect necessary muscle groups, thus allowing invasive procedures in and “in-vivo” preparation.

CHAPTER 3-OBJECTIVES

Previous studies were performed under displacement control, using both cyclic and static lumbar flexion, to evaluate creep developed within the viscoelastic structures of the spine and to assess the recovery of these structures. In the studies conducted by Solomonow et al. (1999) and Williams et al. (2000) the subjects underwent 50 minutes of constant cyclic and static loading respectively, which is highly unlikely for industrial workers due to its long duration. Therefore, one of the goals of this current study is to assess the creep and muscular activity of the feline while undergoing 20 minutes of cyclical loading. Solomonow, M, Karasulu, S. et al. (in press) performed a load control study of the lumbar spine under static flexion. The subjects underwent various magnitudes (20N, 40N, and 60N) of static loading for twenty minutes of loading and seven hours of rest. The results of this study demonstrated creep within the viscoelastic structures of the spine, an exponential decline in muscular activity and also three parameters associated with the recovery period. These three parameters were 1) an initial hyperexcitability ten minutes after the loading period 2) a steady state recovery period and 3) a final hyperexcitability of muscle activity in the end of recovery. Solomonow et al. (in press 1) examined the neurological responses to static lumbar flexion under constant peak load; however, the neurological responses to cyclic lumbar flexion under constant peak load are still unknown. To assess these neurological responses, the objectives established in this study are; to determine the behavior of reflexive muscle activity after various magnitudes of cyclic loading, then 7 hours of rest; to assess the development of creep in the spine's viscoelastic structures; and to develop a model for both the 20-minutes of cyclic loading and the 7-hours rest period.

CHAPTER 4-METHODOLOGY

4.1 Preparation

Twenty adult cats were anesthetized with a single dose of chloralose (60 mg/kg) in a protocol approved by the Institutional Animal Care and Use Committee (IACUC). The skin over the lumbar spine was dissected from the thoracic level to the sacral level and reflected laterally to expose the dorsolumbar fascia. After dissection, the preparation was placed in a rigid stainless steel frame that allowed the isolation of various lumbar levels by external fixation. A gauze pad soaked with saline fluid was applied over the incision during the experiment to prevent the exposed tissue from drying.

4.2 Instrumentation

Six pairs of stainless steel fine wire electromyographic (EMG) electrodes were inserted 3 to 4 mm inter-electrode distance via hypodermic needles, into the multifidus muscles of the L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 on the right side, 5-6 mm from the midline. The wire electrodes were insulated except for a 1-mm exposed tip. A ground electrode was inserted in the gluteus muscle. Each electrode pair constituted the input to a differential amplifier of 110 dB common mode rejection ratio, a gain capability of up to 200,000 and a band pass filter of 6-500 Hz. EMG responses from each channel were monitored on oscilloscopes and stored in a computer at a sampling rate of 1000 Hz.

An “S” shaped stainless steel hook was inserted around the middle of the supraspinous ligament of the L4/L5 motion segment and connected to the vertical actuator of a Bionic 858 Material Testing System (MTS, Inc., Minneapolis, MN). The load was applied by the MTS actuator with a computer controlled loading system

operated in a load control mode. The vertical displacement of the actuator and the load cell output were also measured (at 50 Hz), and logged into the computer.

Two external fixators were used to isolate the lumbar spine; a first fixator to the L1 posterior spinal process and a second fixator to the L7 process. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral/pelvic structures. The intention of the external fixation was not, however, to prevent any motion. A schematic of the experimental set-up is shown in Figure 6.

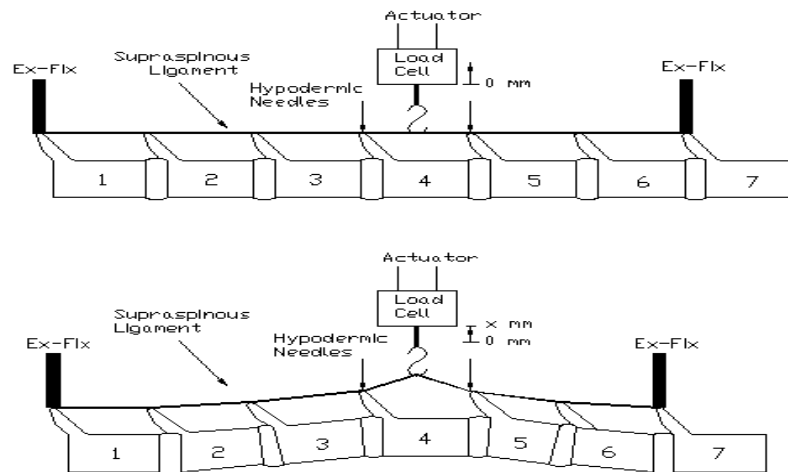


Figure 6. Schematic of feline supraspinous ligament along the seven lumbar vertebrae without and with load respectively.

4.3 Protocol

The stainless steel hook applied to the L-4/5 supraspinous ligament was pulled up by the MTS actuator from a resting position with a preload of 1 N applied just before a 20-minute cyclic loading period, immediately after the 20-minute cyclic period was terminated, and immediately after a 7 hour rest period. The 1 N preload was applied to

offset ligament laxity. Two short hypodermic needles were inserted into the spinous processes of L4 and L5. The length of the supraspinous ligament between these two needles was measured by using a digital electronic caliper immediately before and immediately after the 20-minute cyclic loading application and at the end of the 7 hours rest period, while the static tension was reset to 1 N. The cyclic loading was applied at a frequency of .10 Hz for twenty minutes followed by a 7 hour rest period. EMG and load were recorded over 10 second windows as follows: during the 20-minute cyclic and for a single cycle test loading, after 10-minutes of rest, 30-minutes of rest, 60-minutes and every hour thereafter.

This protocol was used for different peak loads of 20 N (n=6), 40 N (n=7), and 60 N (n=7). Each group was subjected to only one load magnitude. These loads were selected to cover the range from excitation threshold (15N) to just below the maximal strain of the ligament (70N). The creep (at 20min) and the residual creep (at the end of 7 hours recovery) values were calculated separately for each of the three loads applied. Five preparations were used as controls. In these preparations, the dissection was performed as usual; however, these animals were not subjected to loads and were left undisturbed for 20 minutes plus 7 hours. Only EMG was recorded from this control group.

4.4 Analysis

Ten-second windows of electromyogram, cyclic loading applied to the spine, the vertical displacement at the L-4/5 supraspinous ligament were sampled immediately at the beginning of the loading period, and continuously for the 20-minute of cyclic loading, as well as for the short tests in the recovery period. Each electromyogram

sample was integrated (IEMG) over the 10-second window. The EMG recorded from each channel at the beginning of the 20 minutes loading period was used as a basis for the normalization of the EMG recorded subsequently and during the 7 hour recovery period. The initial IEMG in each channel was designated as 1.0, and the IEMG values recorded thereafter were represented as a percentage. The normalized IEMG (NIEMG) of each corresponding recording period (after the initial IEMG recording) for each of the twenty cats were pooled, and the means and standard deviations were calculated and plotted on a normalized IEMG vs. time plot for the six channels recorded from L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7. Displacements of all preparations at the three loads were pooled, and the mean (\pm SD) was calculated and plotted as displacement vs. time plot.

The measurements of the supraspinous ligament length at 1N preload before and immediately after the 20 min. load was applied and immediately after the recovery period, and the associated vertical displacement of the supraspinous ligament, were used to calculate the creep and residual creep, respectively, in the ligament by using equations 1 and 2, derived from Figure 7.

$$L_f = 2\sqrt{\left(\frac{1}{2}Lo^2\right) + Vd^2} \quad (1)$$

$$\text{Residual Strain} = \frac{L_f - Lo}{Lo} * 100\% \quad (2)$$

Where,

Lo = the distance between the two hypodermic needles inserted into L4 and L5 processes

Vd = the vertical displacement of the MTS cross head

L_f = the final length of the of the supraspinous ligament while the load was 1N.

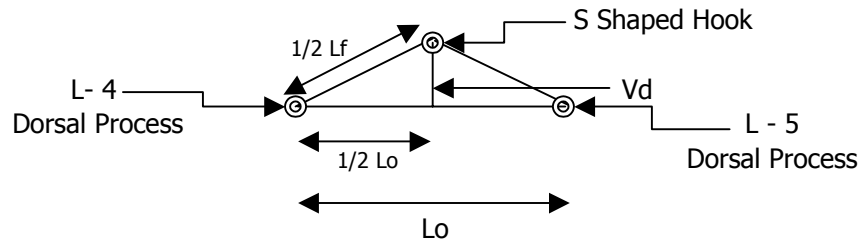


Figure 7. Schematic representation of residual axial strain calculation.
(Figure from Solomonow et. al., 2001)

4.5 Model Development

The pooled NIEMG data of the three lumbar levels from the multifidus muscle as well as the displacement recorded from the load cell were fitted to a model, in the form of an exponential function. An exponential model was chosen because the viscoelastic components of the spine decay exponentially under loads. The model for NIEMG and actuator displacement in the loading period is similar to the one developed by Solomonow et al. (2000) and Jackson et al. (2001), which are shown in Equations 3 and 4.

For the NIEMG:

$$\text{NIEMG}_{(t)} = A e^{-t/T1} + \text{NIEMG}_{ss} \quad (3)$$

Where,

NIEMG (t) = Normalized Integrated Electromyography as a function of time

A= Exponential Component initial amplitude (unitless)

T1= Exponential decay time constant (minutes)

NIEMG_{ss}= Steady state NIEMG amplitude (unitless)

t = Time

The displacement followed an exponential model as follows:

$$\text{DISP}_{(t)} = D_0 + D_L (1 - e^{-t/T_2}) \quad (4)$$

Where,

Disp(t) = Actuator vertical Displacement as a function of time (millimeters)

D₀ = Elastic component of displacement (millimeters)

D_L = Viscoelastic component amplitude (millimeters)

T₂ = Time Constant (minutes)

t = Time

The models defined in equations 1 and 2 were applied to each of the collected data sets associated with each of the three load levels used. Similarly, exponential models were chosen to describe the NIEMG and displacement during the 7 hour recovery period. The model for NIEMG and actuator displacement in the loading period is similar to the one developed by Solomonow, M, Karasulu, S., et al. (in press) which are shown in Equations 3 and 4, respectively. The model for the displacement was:

$$\text{DISP}(t) = D_0 + R + (D_L - R)e^{-t/T_3} \quad (5)$$

Where,

Disp(t) = Actuator vertical Displacement as a function of time (millimeters)

D₀ = Displacement at the end of the 20-minutes loading (millimeters)

R = Residual Creep at the end of recovery (millimeters)

t = Time

T₃ = Recovery time constant (minutes)

For the NIEMG, the model format was:

$$\text{NIEMG}(t) = E (1 - e^{-t/T_4}) + tB e^{-t/T_5} + C (t - T_d) e^{-(t - T_d)/T_6} + \text{NIEMG}_{ss} \quad (6)$$

Where,

$E (1 - e^{-t/T_4})$ represents the steady state (permanent) recovery component

$tB e^{-t/T_5}$ represents the initial transient hyperexcitability component.

$C (t - T_d) e^{-(t - T_d)/T_6}$ represent a delayed transient hyperexcitability (“morning after”).

NIEMG_0 represents the residual response at the end of 20 minutes constant load.

In this model, the constraint of $E + \text{NIEMG}_0 = 1$ is used to insure that full recovery results in a normal (unity) response.

CHAPTER 5-RESULTS

5.1 Raw EMG

The raw EMG represents the direct muscle activity of each preparation while under loading. This EMG is then analyzed to further examine the muscle activity of the group of preparations within a loading category. Figures 8, 9, and 10 shows the raw EMG of a subject at 20 N, 40 N, and 60 N, respectively.

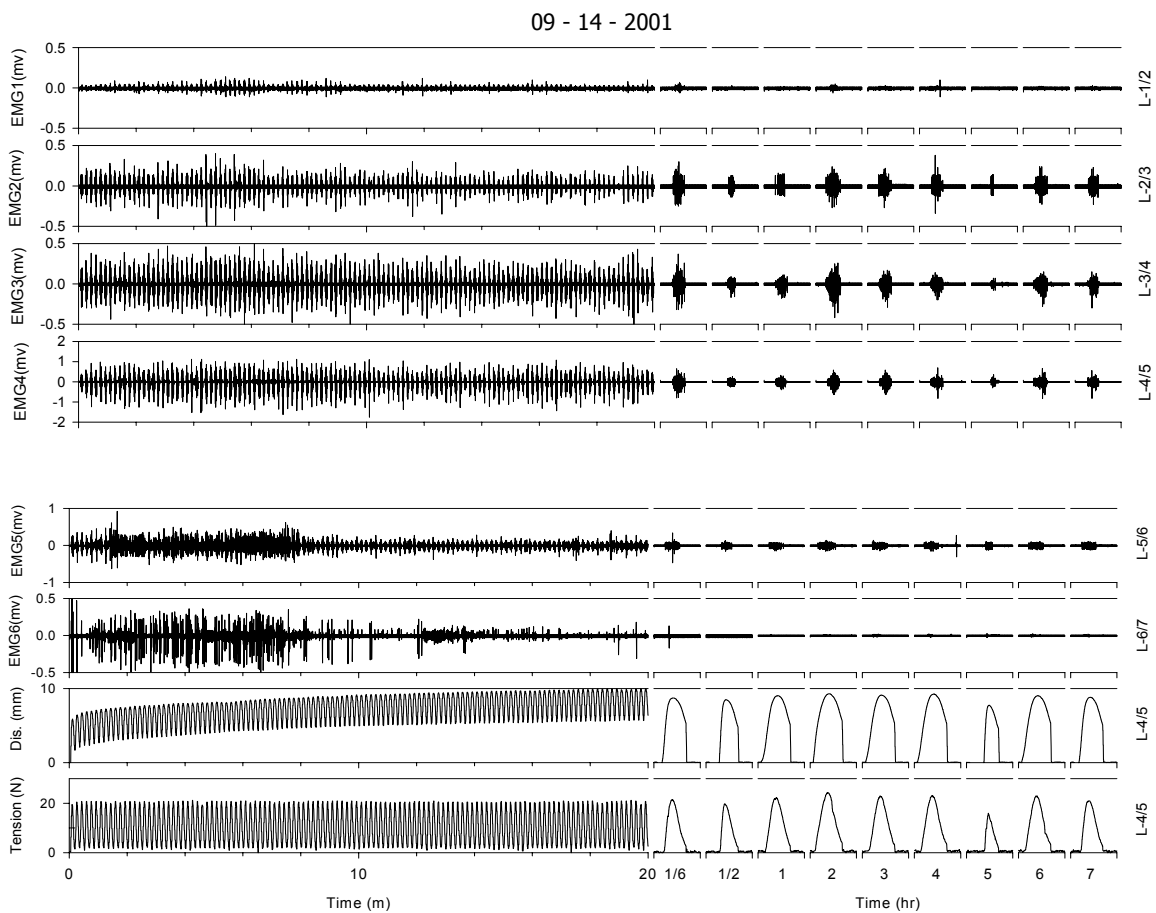


Figure 8. Raw EMG responses from the six channels, lumbar spine displacement and tension for the six subjects under 20N load.

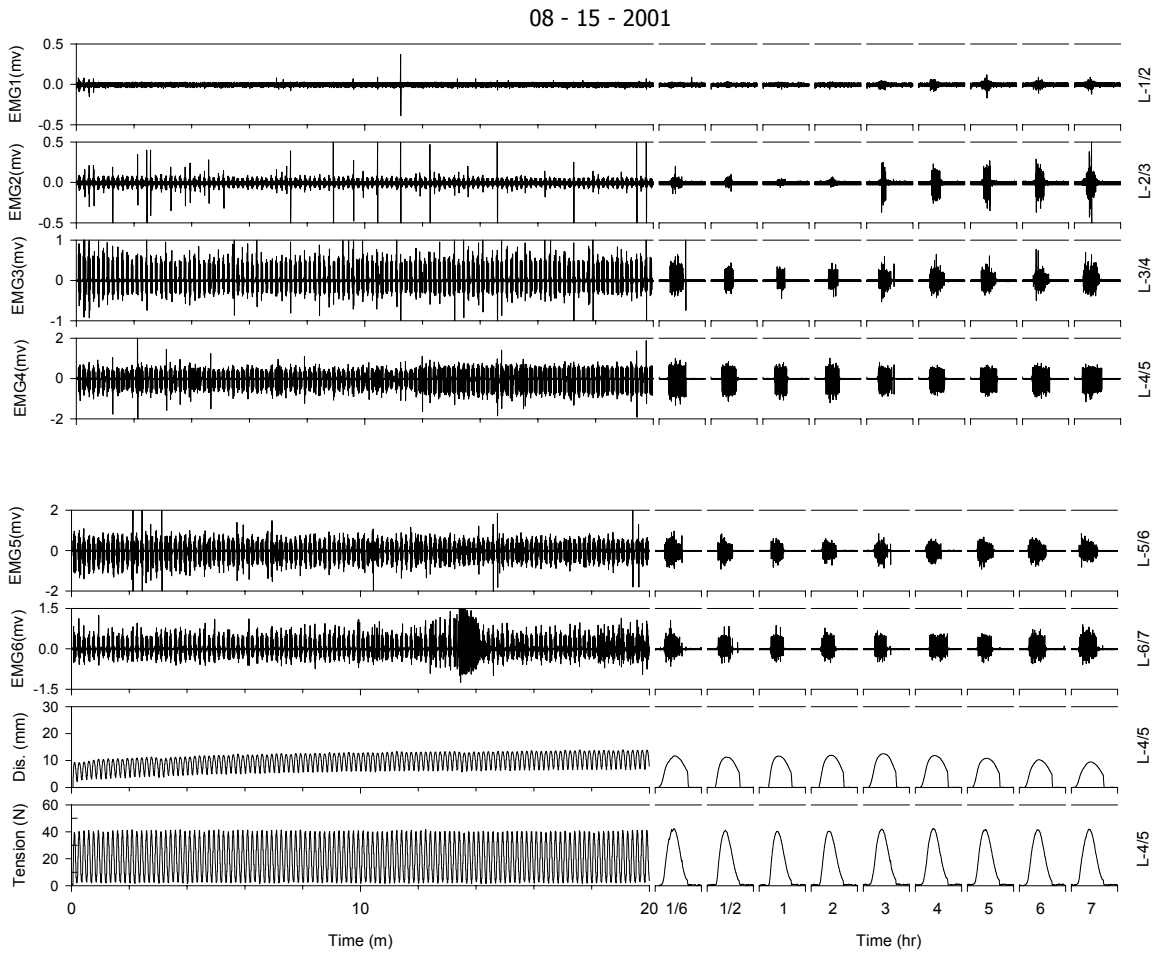


Figure 9. Raw EMG responses from the six channels, lumbar spine displacement and tension for the seven subjects under 40N load.

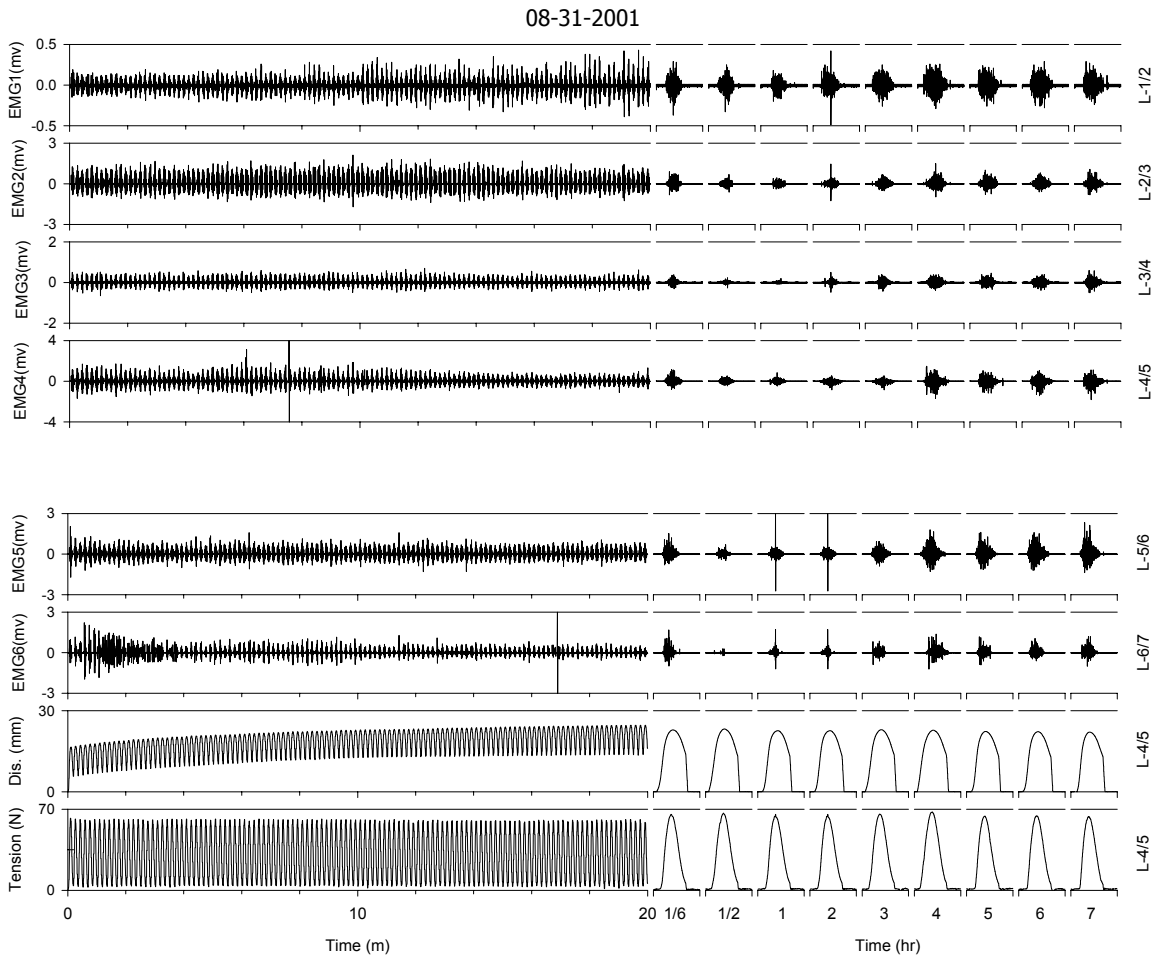


Figure 10. Raw EMG responses from the six channels, lumbar spine displacement and tension for the seven subjects under 60N load.

5.2 NIEMG Results

5.2.1 20N Loading

There was low EMG activity of the multifidus muscle at L-1/2, L-2/3, or L-6/7 throughout loading or recovery. This is likely due to the fact that they are the lumbar levels farthest away from the load point (L-4/5). However, there was still evidence of exponential decay of multifidus muscle activity. The most activity was evident at L-3/4, L-4/5 and L-5/6. The mean NIEMG values at the end of the 20-minute loading period for the 6 preparations for the 20 N load decreased to, 84%, 85%, 77%, 58%, 56% , and 64%of the initial EMG for L-1/2, L-2/3, L-3/4, L-4/5 , L-5/6 and L-6/7 respectively. Spasms occurred throughout the 20 minutes of loading, as they are a direct response to pain and tissue damage. At the different levels we can see varying magnitudes of spasmodic activity. In the first 10 minutes of rest, there was initial hyperexcitability, as evidenced by the increase in EMG. The mean NIEMG of the multifidus muscles increased to 87%, 91%, 87%, 88%, 77% and 76% of their initial value for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6 and L-6/7 respectively. Thirty minutes into the resting period the EMG decreased and then began to rise steadily thereafter. The EMG peaked at the fifth hour of recovery and the values were 91%, 102%, 108%, 148%, 146%, and 146% of the initial NIEMG value for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively. EMG steadily decreased during the sixth and seventh hours of recovery. At the end of the recovery period the EMG decreased to 99%, 113%, 108 % , 134%, 126 % , and 135% of the initial NIEMG value for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively.

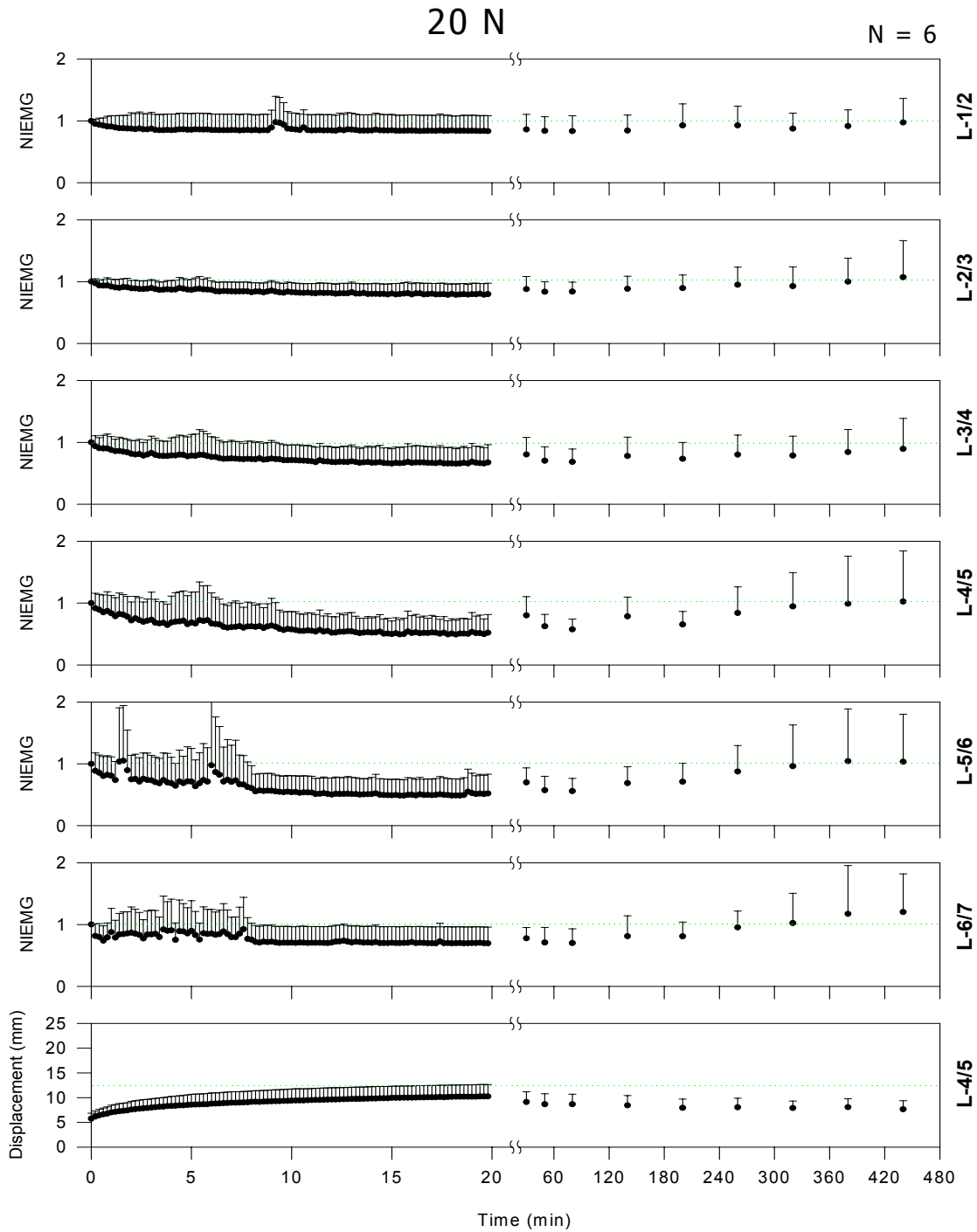


Figure 11. Mean NIEMG for all lumbar levels at 20N load and lumbar spine displacement at L-4/5

5.2.2 40N Loading

The preparations tested at 40N demonstrated higher EMG activity at L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 as opposed to L-1/2. Again, this is most likely because L-1/2 is the farthest vertebra from the loading point. The NIEMG showed the exponential decay of electrical activity at each lumbar level during the 20-minute loading period. The mean NIEMG values at the time of the 20-minute loading period for the 7 preparations in the 40 N load is as follows 70%, 78%, 65%, 68%, 66% and 63% of the initial EMG for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively. Spasms occurred throughout the 20 minute loading period. In the first 10 minutes of rest, there was initial hyperexcitability, EMG increased to 77%, 84%, 74%, 89%, 91% and 79% of their initial values for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively. The mean NIEMG decreased after the first 10-minutes of rest to the end of the first hour. The mean NIEMG gradually increased thereafter and at the end of the 7 hours recovery the values were 87%, 128%, 136 %, 134%, 110%, and 100% of the initial NIEMG value for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively.

5.2.3 60N Loading

In general, at 60 N, the typical results show high EMG activity at every lumbar level for both the 20 minute loading period and the recovery period. The mean NIEMG values at the end of the 20-minute loading period for the 7 preparations in the 60 N load is as follows, 73%, 79%, 76%, 74%, 63%, and 59% of the initial EMG for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively. Spasms were present throughout the loading period. In the first 10 minutes of rest, there was initial hyperexcitability, EMG increased

to 84%, 96%, 104%, 91%, 89%, and 77% of their initial value for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively. The mean NIEMG decreased after the first 10-minutes of rest to the end of the first hour. The mean NIEMG gradually increased thereafter and at the end of the 7 hour recovery, it was 137%, 111%, 120%, 110%, 133%, and 85% of the initial NIEMG value for L-1/2, L-2/3, L-3/4, L-4/5, L-5/6, and L-6/7 respectively.

5.3 L-4/5 Supraspinous Ligament Creep

Axial strain was performed on the supraspinal ligament by pulling the ligament with the “S shaped” hook. While the discs and capsules underwent shear strain due to the constant activation of the muscles, the displacement sensor within the MTS machine measured lumbar spine displacement. Since the lumbar spine displacement is an indirect measurement of the creep of the discs, ligaments, fascia and capsules, this measurement is sufficient enough to examine the overall spinal creep. However, it was necessary to measure the axial strain to obtain an actual physical measurement that would enable a calculation of the creep within the L-4/5 supraspinous ligament. Mean creep at the end of 20 minutes and 7 hours was calculated using equations (1) and (2). Table 1 shows the values for these parameters along with the percent recovery at 20, 40 and 60 N. The supraspinous ligament was 3.32 % longer at the end of 20 minutes for the 20 N loads and recovered to 2.31% after the 7 hour resting period. This was a recovery of 30.4%. As the load increased, the creep of the supraspinous ligament increased at the end of the 20-minute loading period. This trend was also evident for the residual creep after 7 hours of rest. A residual creep of 2.31% was present for the group subjected to the 20N load, and residual creep of 2.29 %, and 3.71% was present for the groups subjected to 40N, and

60N loads, respectively. For 40N and 60N the mean creep values at the end of 20 minutes loading were 7.26 % and 15.85 % greater than the initial ligament length, respectively. At the end of the 7-hour resting period, the 20 N, 40N, and 60N loads recovered to 30.4%, 68.5%, and 76.6% respectively. In essence, larger recovery was seen for preparations exposed to larger loads.

Full recovery was not observed in any of the preparations. Seven hours of rest proved to be insufficient time for full recovery of the creep during the 20-minute cyclic loading period.

Table 1. Mean Creep of the Supraspinous Ligament

Load N	Mean Creep at end of 20 minutes	Mean Creep at end 7 hours	% Recovery
20N	7.01 ± 4.37%	2.83 ± 1.02%	30.40%
40N	10.54 ± 5.96%	4.71 ± 1.45%	68.50%
60N	26.01 ± 14.2%	5.07 ± 2.35%	76.60%

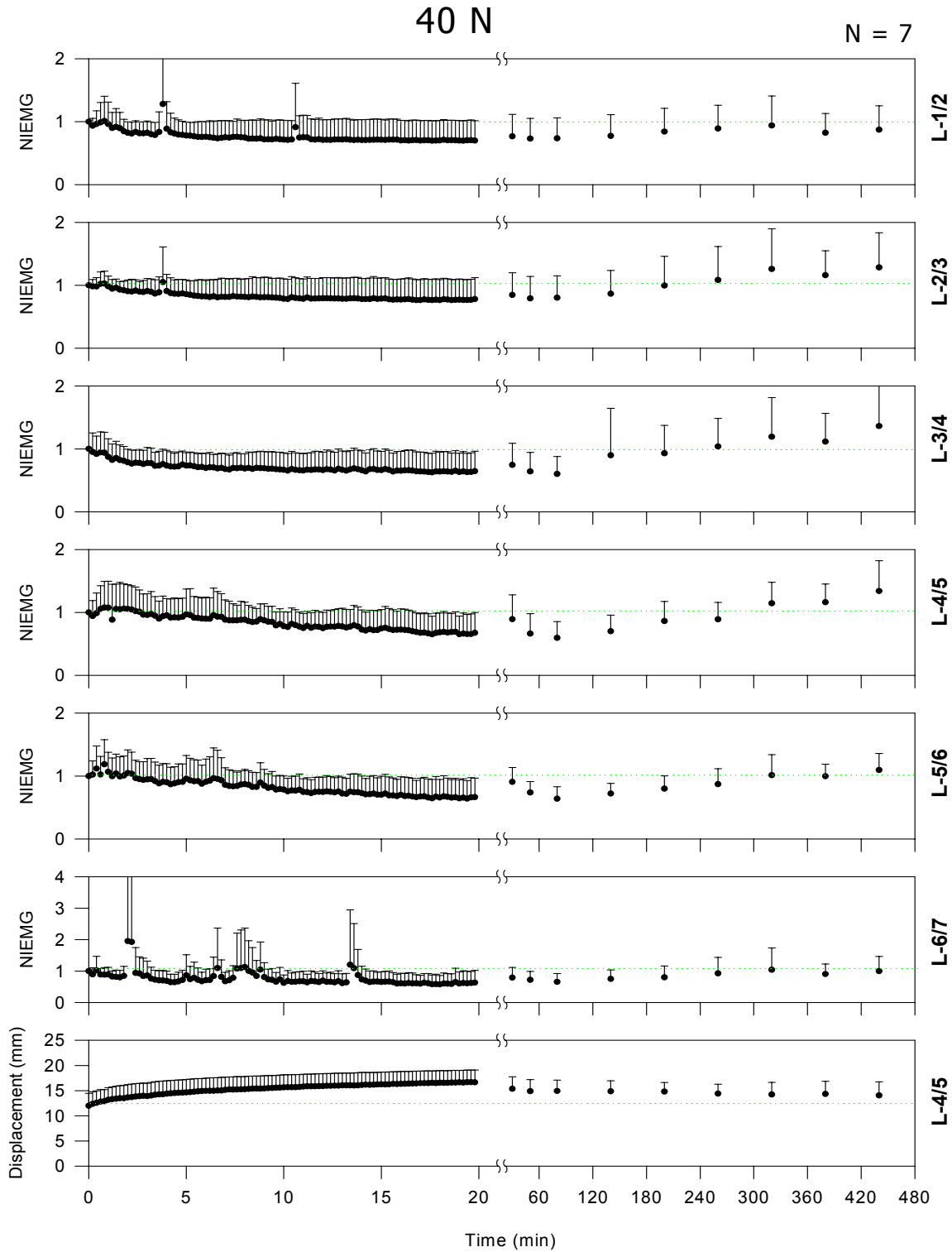


Figure 12. Mean NIEMG for all lumbar levels at 40N load and lumbar spine displacement at L-4/5

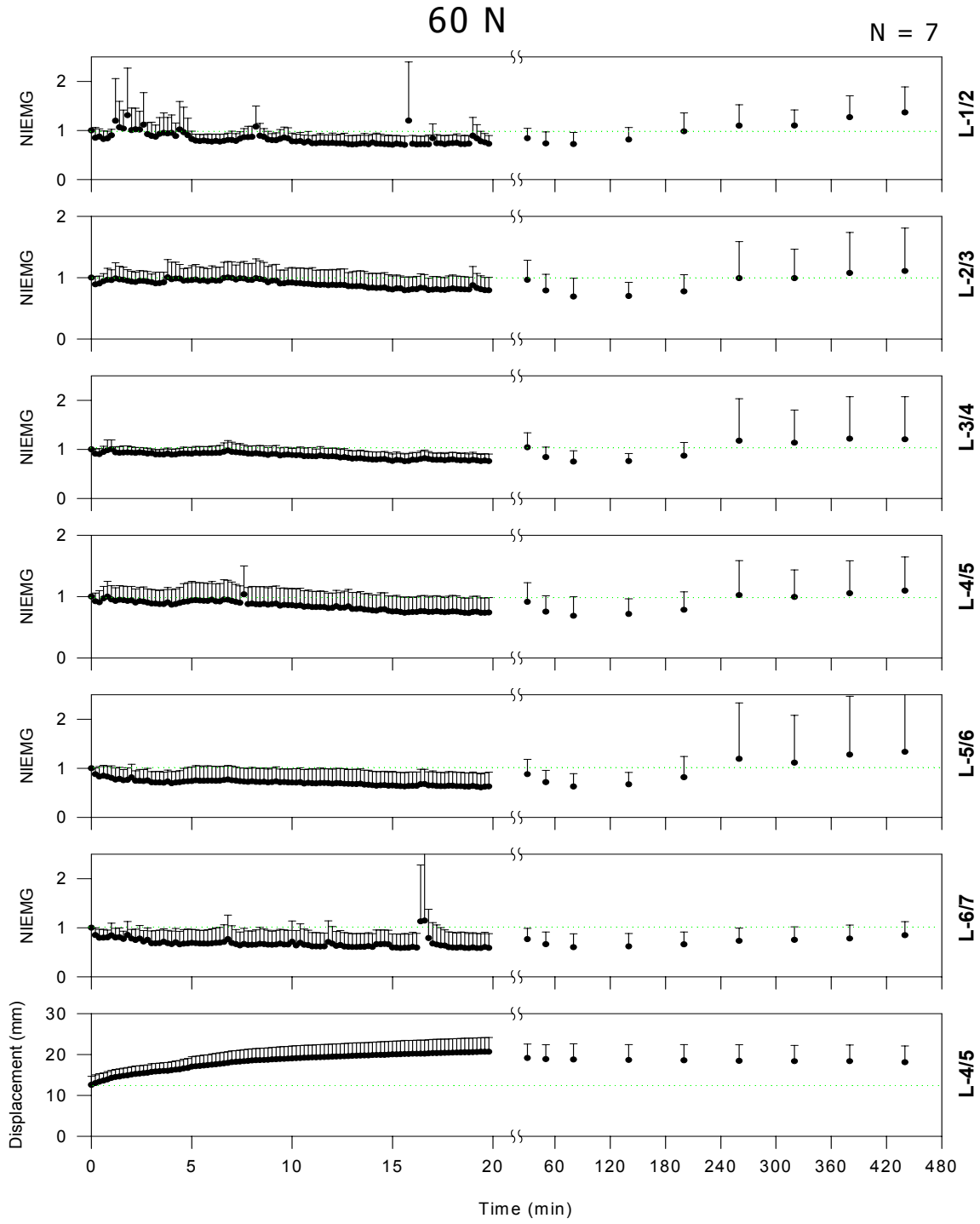


Figure 13. Mean NIEMG for all lumbar levels at 60N load and lumbar spine displacement at L-4/5

5.4 Lumbar Spine Displacement

The vertical displacement of the lumbar spine at the L-4/5 level during the application of 20 minutes of cyclic loading and 10 second tests during recovery is an indirect measure of the overall creep developed in the viscoelastic tissues of the spine.

Figure 14 shows the mean displacement for each of the three loads applied throughout the study. Table 2 provides the initial mean vertical displacement, displacement at the end of 20 minutes test and at the 7th hour of recovery for each of the three load intensities used in this study.

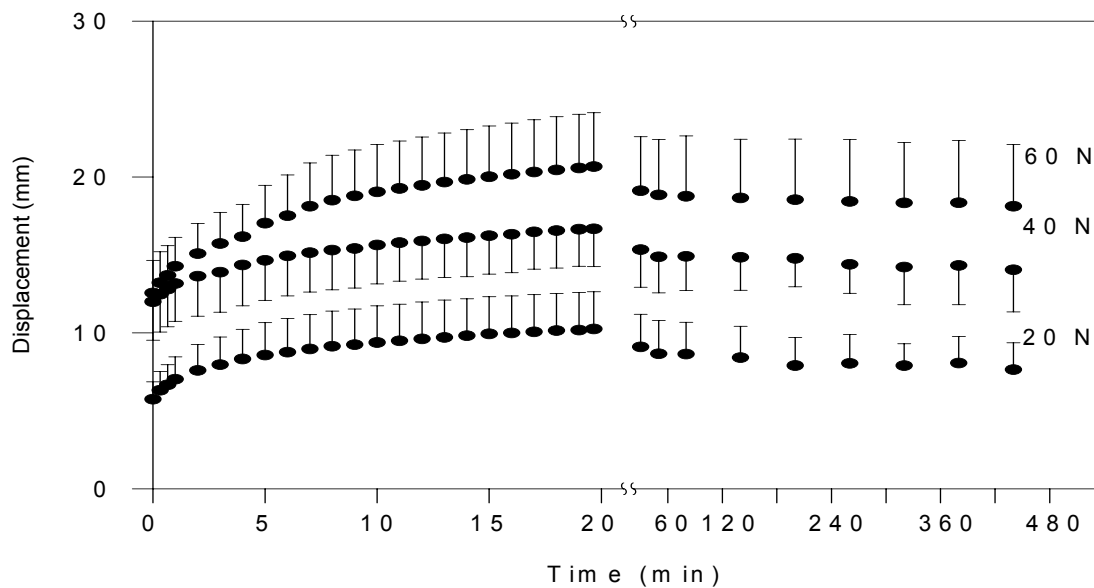


Figure 14. Displacement of Lumbar Spine at L-4/5

Table 2. Mean and S.D. of Lumbar spine displacement

Load N	Mean Initial Displacement	Mean Displacement at end of 20-Minutes	Mean Displacement at end of 7 hours
20 N	5.74 ± (1.12) mm	10.25 (± 2.39) mm (+ 78.6%)	7.64 (± 1.72) mm (+ 33.2 %)
40N	11.99 ± (2.46)mm	16.67 (± 2.412) mm (+ 38.9 %)	14.04(± 2.70) mm (+ 17.1 %)
60N	12.56 ± (2.10) mm	20.67 (± 3.46)mm (+ 64.6%)	18.12 (± 3.97) mm (+ 44.3%)

For all three loads, the 20-minute loading periods demonstrated an exponential increase in lumbar spine displacement and the recovery period demonstrated exponential decrease. For the six preparations exposed to the 20N load, the mean initial displacement was 5.7386 ± 1.1174 mm and at the end of 20 minutes it was 10.2487 ± 2.394 mm, a 78.6 % increase. The displacement recovered to 7.6414 ± 1.7249 mm; however, it was still 33.2 % over its initial value. This 33.2% shows that the lumbar spine never fully recovered over the duration of the study.

For the seven preparations exposed to 40N load, the mean initial displacement was 11.9948 ± 2.4585 mm and at the end of 20 minutes it was 16.6672 ± 2.4167 mm, a 38.9% increase. The displacement recovered to 14.0428 ± 2.6989 ; however, it was still 17.1 % over its initial value.

For the seven preparations exposed to 60N load, the mean initial displacement was 12.5551 ± 2.1042 mm and at the end of 20 minutes it was 20.6658 ± 3.4585 mm, a 64.6 % increase. The displacement recovered to 18.1178 ± 3.9652 ; however, it was still 44.3 % over its initial value.

The larger loads brought forth a larger initial vertical displacement of the lumbar spine. The residual displacement at the end of the recovery period ranged from 17.1% for the 40 N load, to 33.2% and 44.3% for 20 N and 60 N loads respectively. Total recovery was never achieved for either of the loads.

5.5 Statistical Analysis

Repeated measures ANOVA was used to determine if there were any differences between the load factor of 20N, 40N, and 60N. ANOVA was also used to determine if there were any differences between the time factor of 0 min (before load testing), 20 min (at the end of load testing), and 440 min (at the end of recovery), and to establish if there were any differences between the time*load interaction. The evaluations of load, time and the interaction of time and load were all based on displacement measures. Table 3 shows the results of the ANOVA test on the loads, time, and load*time interaction. The result for the load testing displayed a P-value<0.0001, therefore there was a difference for the load factor based on the displacement measures. For the time factor a P-value<0.0001 was displayed, which indicated a difference within the time factor. The load * time interaction displayed a P-value<0.1306, therefore there was no difference and no further testing warranted. The ANOVA allowed us to see that there was a difference within the load factor and within the time factor; however we needed to use a post-hoc test to demonstrate where the differences were within these factors. Duncan's multiple

range test was used to display the differences between 20N, 40N, and 60 N and to display the differences between 0 min, 20 min, and 440 min.

Table 3. Repeated Measures Statistical Analysis

	F Value	Pr>F	Results
Load (20N, 40N, 60N)	21.61	0.0001	Significant Difference
Time (Initial, 20 min, 7hours)	55.35	0.0001	Significant Difference
Load*Time	1.87	0.1306	No Significant Difference

The results for the post-hoc test on load are displayed in Table 4. The load test indicated that there were significant differences between 20N, 40N, and 60N. The results showed that the mean displacement for the subjects under 60N load was greater and significantly different than the mean displacements of those subjected to the 40N load and the 20N load. Those subjected to the 40N load had a mean displacement greater and significantly different than that of the 20 N load. The load Post-hoc test indicated that 60 N had the most effect on the ligament’s displacement. The results for the post-hoc test on time are displayed in Table 5. The Post-hoc test on time indicated that there were differences in displacement at 0 min, 20 min, and 440 min. The mean displacement at 20 minutes was greater and significantly different than that of both 0 minute and 440 minutes. The indication that the mean displacement at 20 minutes is greater than that of 0 minute states that creep did occur over the loading period. The indication of the mean displacement at 20 minutes being greater than that at 440 minutes states that the ligament did recover to some extent over the 7 hour resting period. At 440 minutes, the mean

displacement was greater and significantly different than that at 0 minute and this indicates that the ligament never fully recovered after 7 hours of rest.

Table 4. Evaluation of Load Differences

Load Differences Evaluated		
N	Load	Mean Displacement within Loads
18	20N	8.1
21	40N	14.2
21	60N	17.6

Table 5. Evaluation of Time Differences

Time Differences Evaluated		
N	Time	Mean Displacement between Loads
20	Initial/ t=0	10.4
20	20 min	16.2
20	7 hours	14.1

5.6 Model Development

A mathematical model was used in by Gedalia et al. (1999) to determine the rest required for full recovery of reflexive muscular activity. This biexponential recovery model included two exponential components and a residual value. The two components the model focused on were the fast component, first component, which specifically describes the recovery of the ligaments and the slow component, second component,

which described the recovery of the discs over time. However, there is no third component such as that which Solomonow, M, Hatipkarasulu, S. et al., (in press) examined as they evaluated the recovery of the multifidus after prolonged static lumbar flexion. This component is referred to as the initial hyperexcitability component. In Solomonow, M, Hatipkarasulu, S. et al., (in press), they indicated the mathematical model for recovery by the summation of three behaviors within the recovery period. The first behavior, which is the initial hyper excitability, has the model $tBe^{-t/T5}$. The second parameter of recovery, which is the normal recovery, has the model of $C(1-e)^{-t/T4}$. The third parameter of recovery, which is delayed hyper excitability, has the model of $(t-Td)De^{-(t-Td)/T6}$. We would like to examine if these parameters occur under constant cyclic lumbar flexion.

In this study, the parameters for the 20 minute loading period and vertical displacement models fitted were obtained by using the Marquardt-Levenberg non-linear regression algorithm. The recovery models were fitted by using trial and error with a transform. The parameters for vertical displacement model, as in equation (4) are shown in Table 6 and graphically in Figure 15. Within the 20 minutes of this test, only the fast decaying exponential component, which attribute to ligamentous viscoelasticity, is seen.

Table 6. Vertical Displacement Model during 20 Minutes Loading Period

Load	D₀	D_L(mm)	T₂ (min)	r²
20N	6.321	4.251	5.488	0.9984
40N	12.34	4.49	7.209	0.9923
60N	12.88	8.16	7.042	0.9975

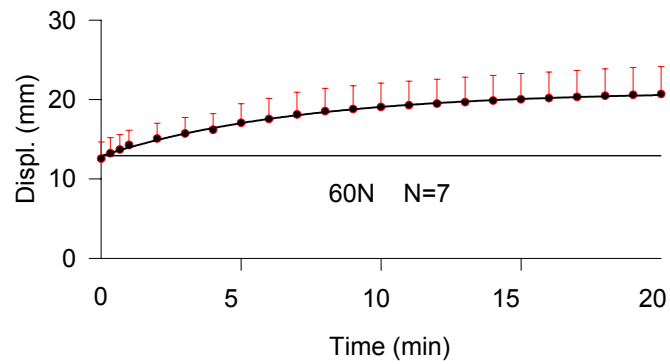
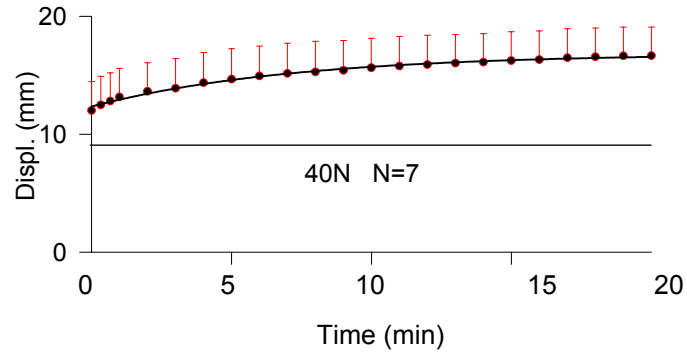
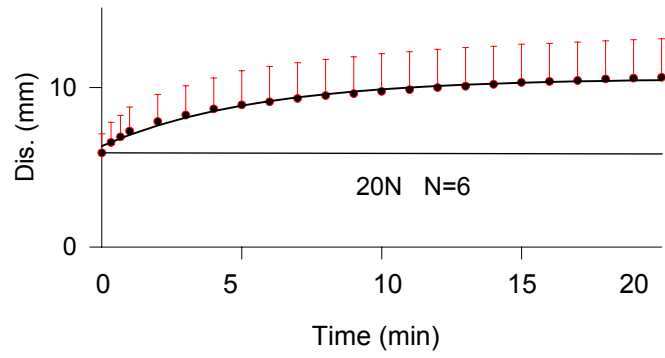


Figure 15. Vertical Displacement Model during 20 Minutes Loading Period.

Equation (3) describes the behavior of the 20 minutes of loading. The pooled NIEMG values for the 20-minutes of cyclic loading period take a decaying exponential form. The model parameters that produce the best model fit are shown in Table 7 and in a graphical form in Figure 16 superimposed on the experimental data. Overall, the models for 20N, 40N, and 60N of cyclic loading show similar behavior, where the exponential decay rates increase with higher amounts of load. For higher load magnitudes, it takes longer to achieve steady state. Overall, the L-3/4 shows a slower behavior at higher NIEMG values. L-5/6 also showed a slow behavior, however, its NIEMG values were lower than that of L-3/4 and L-4/5. At the load application point (L-4/5) the NIEMG values were medial with the highest decay rate. For the L-4/5 and L-5/6 in the 40N load, the NIEMG at approximately 1 minute increased significantly above 1. This is due to spasmodic activity in the musculature. This results in the offset of the exponential decay in the curve and also the decrease in the quality of model fit.

Table 7. NIEMG Model during 20 Minutes Loading Period

NIEMG Models During Cyclic Loading					
Load N	Level	NIEMGss	A	T₁ (min)	r²
20 N	L- 3/4	0.778	0.22	1	0.822
	L- 4/5	0.597	0.463	1.5	0.752
	L- 5/6	0.57	0.43	1	0.364
40N	L- 3/4	0.654	0.346	2.7	0.977
	L- 4/5	0.68	0.32	8.3	0.789
	L- 5/6	0.65	0.35	8	0.801
60N	L- 3/4	0.76	0.24	8	0.7
	L- 4/5	0.76	0.24	6	0.707
	L- 5/6	0.66	0.34	1.5	0.72

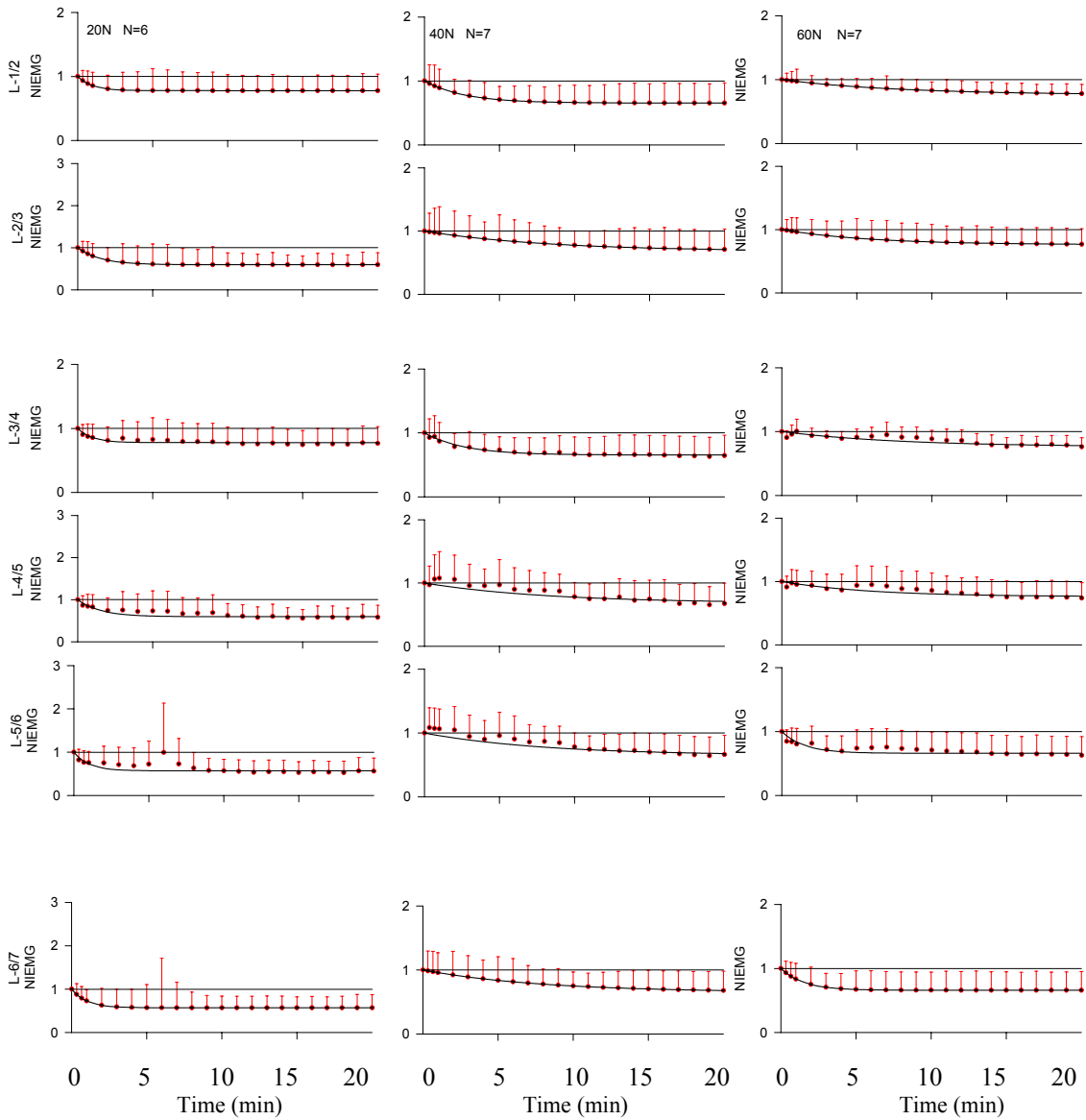


Figure 16. NIEMG Model during 20 Minutes Loading Period

The parameters for displacement recovery models are tabulated in Table 8 and shown in Figure 17. The recovery increases with increasing load, as do the amount of recoverable strain, represented by the column under the parameter D_R .

Table 8. Vertical Displacement Model Parameters during 7 hour Recovery

Load	D_o	D_L(mm)	T₃ (min)	R	r²
20N	6.321	4.251	33.3	1.618	0.855
40N	12.34	4.49	11.2	2.175	0.819
60N	12.88	8.16	7.6	5.61	0.881

Equation (6) is the complex model used to fit and explain physiologically the recovery behavior of the NIEMG. The modeling constraint $E + NIEMG_0 = 1$ guarantees that after some indefinite period of sufficient rest, spinal sensitivity to ligament strain returns to normal. The time constant for this component is based on earlier work and was constrained to last between 4 to 8 hours (Haig et al., 1993). The transient hyperexcitability we see in this response is similar to what was observed in earlier work, (Haig et al., 1993) with time constant in the order of 6 to 16 minutes. Compounding this is a delayed hyperexcitability of long duration, with a time constant in the order of several hours. This component has been termed “morning after” behavior, parallel to the delayed soreness and stiffness associated with both cyclic and static lumbar loading. Its onset appeared earlier at higher loads, with a time constant in the order of 2 to 6 hours. Table 9 provides the model parameters and its graphical representation is given in Figure 19.

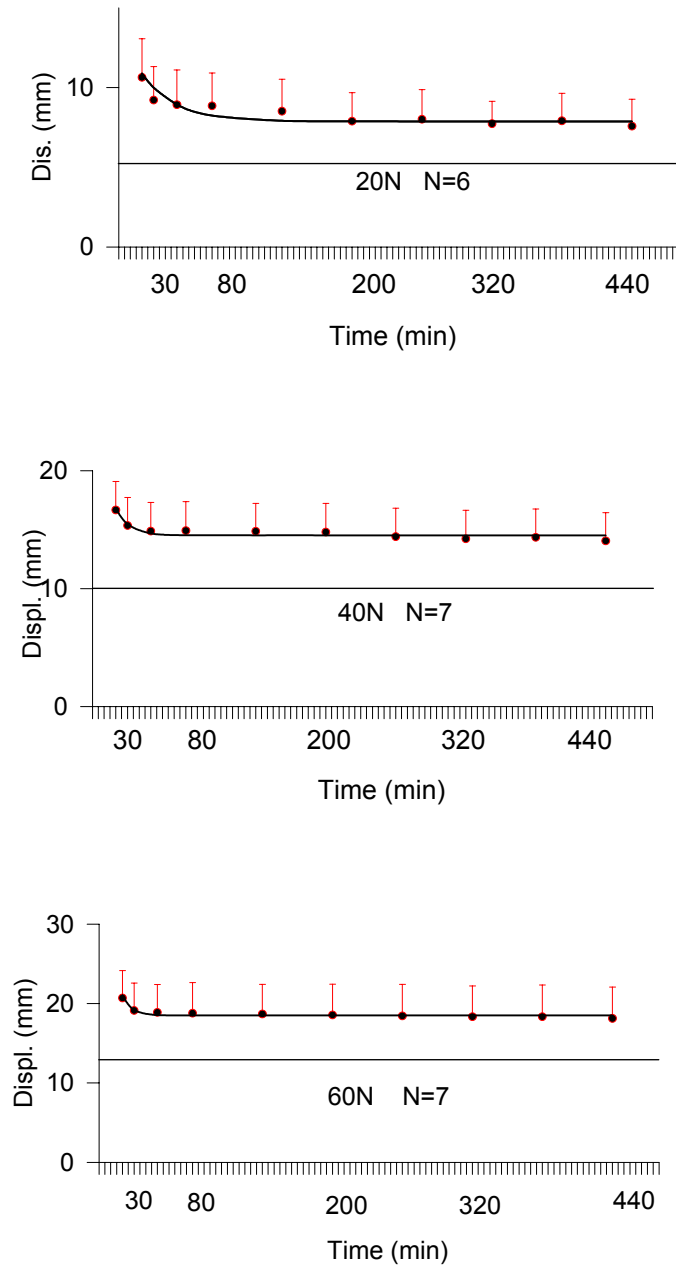


Figure 18. Vertical Displacement Model during 7 hour Recovery Period

Table 9. NIEMG Model during 7 hour Recovery

Load	Level	E	T₄(min)	B	T₅(min)	C	T₆ (min)	T_d(min)	NIEMG_{ss}	r²
20 N	L 3-4	0.35	150	0.06	10	0	120	220	0.65	0.889
	L 4-5	0.44	100	0.07	10	0.01	120	220	0.56	0.978
	L 5-6	0.49	140	0.05	10	0.01	100	200	0.51	0.979
40 N	L 3-4	0.44	300	0.1	6	0	300	150	0.56	0.942
	L 4-5	0.46	210	0.18	6	0	210	250	0.54	0.934
	L 5-6	0.45	210	0.09	10	0	210	250	0.55	0.916
60 N	L 3-4	0.33	300	0.09	10	0	270	180	0.67	0.96
	L 4-5	0.37	350	0.1	8	0	200	240	0.63	0.935
	L 5-6	0.45	350	0.09	10	0	400	190	0.55	0.982

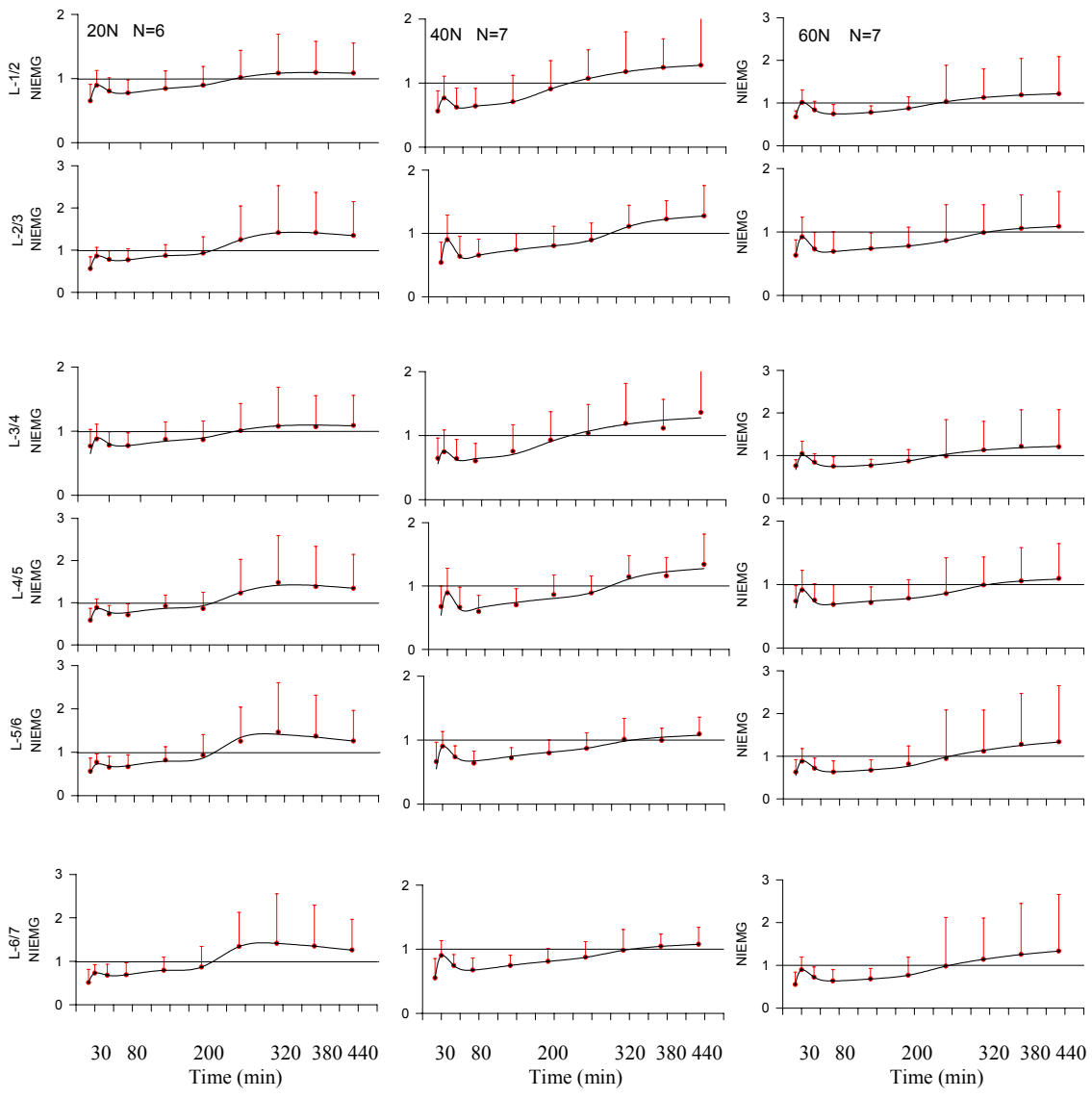


Figure 19. NIEMG Model during 7 hour Recovery Period

CHAPTER 6-SUMMARY AND CONCLUSION

There were many important findings which resulted from this study. The primary finding is that cyclic loading causes a complex and transient neuromuscular disorder independent of the load sustained by the viscoelastic structures. The neuromuscular disorder seems to occur due to the creep in the viscoelastic tissues. Creep occurred at each load and larger recovery was seen at larger loads, however, after 7 hours of rest there was no full recovery for either load. This allows the spine to be exposed to cumulative injury.

From experimental analysis, it is fair to say that many neuromuscular disorders, specifically four, occur due to cyclic loading of various magnitudes. The first component of the neuromuscular disorder noted in this study was an exponential decay of EMG over time. In the 20 minutes of cyclic loading a decrease in muscular activity is evident. The decrease in activity denotes a decrease in spinal protection, which leaves the spine prone to injury. The exponential decay is described by the term $Ae^{-t/T1}$. The major source of the decrease in muscle activity is the creep in the viscoelastic structures. For the 20 N load, the NIEMG at the end of 20 minutes flexion was 56% - 77% of the initial NIEMG in the L-3/4 to L-5/6 vertebrae. Similarly, for 40 N and 60 N, the NIEMG at the end of the 20-minutes cyclic loading decreased to 65% - 68%, and 63 -76% of the initial NIEMG, respectively.

Secondly, there were recurring spasms during the 20 minutes of loading. These spasms were observed in all loads, however, their intensity and appearance frequency was unpredictable. Spasms are considered a disorder directly related to tissue damage and they are a direct result of pain response. Therefore, the spasms show that there may

be damage within the viscoelastic structures as creep occurred. The exact cause of spasms has not yet been established.

The third neuromuscular disorder noticed was the transient (initial) hyperexcitability of the muscles during the first hour of recovery. This disorder is described by the term; tBe^{-t/T^2} . The peak of the hyperexcitability was observed within the first ten minutes after the cyclic loading for all load magnitudes. There was no difference in the transient hyperexcitability based on load. The time constant during this period ranged from 6 minutes to 10 minutes for all load magnitudes. Since micro-damage was present within the viscoelastic tissues due to the 20 minutes of cyclic loading, the pain receptors within were triggered when an attempt was made to stretch the tissues after 10 minutes of rest. The triggering of the pain receptors caused higher than normal muscle forces to protect the spine due to the lack of protection from the viscoelastic tissues.

The fourth component of the neuromuscular disorder was the delayed hyperexcitability which is described by the term; $C(t-Td)e^{-(t-Td)/T^3}$. The time delay indicates that at some time, 2-4 hours after the rest period was initiated a physiological/metabolic process began; inflammation. It takes several hours for inflammation to approach maturation; therefore, we can see after the steady recovery period, the EMG began to increase. When the body begins to recognize the damage, Neutrophils infiltrate the tissues to ingest and digest bacteria or damaged cells (Bainton 1908). This is the most important mechanism of host defense because it arranges the attack on the injurious agent and leads to the repair of the affected tissue (Bainton, 1980). Metabolically, the supraspinous ligament was in the inflammation process nearing the

end of recovery and due to the decreased capacity for the viscoelastic structures to generate force, the muscle activity increased. The ligament's inability to withstand further tension forced the muscles to activate, and in turn, aid in the protection of the spine from injury. The physiological/metabolic response at the end of the recovery period where there is a steady EMG increase is commonly known as the "morning after" response. In most cases pain becomes most prevalent the "morning after" the activity hence the term "morning after" response.

This increase in muscle activity during recovery, due to inflammation, began to peak approximately 5 hours for the 20 N load however, the peak is unknown for both the 40 N and 60 N loads. Since the data was collected during a 7-hour rest period after the 20 minutes loading, it was not possible to observe the exact time at which this hyperexcitability reached its peak in all cases. However, the fact that the hyperexcitability peaked at the 5th hour and slightly decreasing for 20 N, suggests that the process matured.

Everyone becomes familiar with the four components of neuromuscular disorder while performing strenuous work for an extended period of time. As an individual bends to lift boxes or other loads, the tension in the supraspinous ligament causes mechanoreceptors to signal to the spine that it needs support. The ligaments then trigger the muscles to activate. However, as previously mentioned, creep developed in ligaments and the desensitization of the mechanoreceptors causes dramatically diminished muscular activity which allows exposure to instability and injury. Also, the passive forces that the viscoelastic structures generate decrease significantly due to tissue damage. The residual creep remains, although the job is finished, and the worker may start the next day with

this creep. If the worker does this everyday there eventually will be an accumulation of residual creep and the acute inflammation will become chronic. This will ultimately result in a cumulative trauma disorder. Along with the four components of neuromuscular disorder, cyclic loading exposes the spine to injury by other physiologic mechanisms. The first source is the increased laxity in the intervertebrae junction, caused by creep in the viscoelastic structures. The second physiologic mechanism is the additional reduction in muscular forces induced by prolonged active cyclic contraction.

This study can make a significant contribution to industry because of its demonstration of the effects of creep as it relates to the muscle activity of a task simulating bending and lifting. The modeling of this study shows that the muscle activity will decrease while under continuous cyclic loading. Continuous bending and lifting will eventually result in discomfort or pain. The pain will be slightly relieved upon extension. However, as the individual bends again to continue their work, another round of discomfort is felt in the back muscles. This is the initial (transient) hyperexcitability. The pain will then be dormant if the individual discontinues the work. The individual will then feel discomfort/pain and stiffness the morning after. This is the delayed hyperexcitability of the muscles and it may last 1-3 days, following which the episode is forgotten. This discomfort/pain is the inflammatory effects due to the sub-acute damage, which cause inflammation and the delayed hyperexcitability.

Due to the metabolic properties of a live animal, it was only feasible to perform the studies for up to 8 hours for each subject. While under anesthesia, the metabolic properties of the cat will begin to deteriorate after 8 hours and the electrical activity of the muscles would not be accurate. However, after data extrapolation, it was determined that

9 hours of rest is required for the muscle activity of the subject to fully recover after undergoing 20 N of constant cyclic loading. It would take approximately 13.5 hours for full recovery after 40 N of loading and 21 hours of rest is required for full recovery after undergoing 60 N of constant cyclic loading.

There are still many other research directions which could be evaluated after obtaining the results of this study. One major future research direction could be to examine optimal work-rest periods. Instead of giving seven continual hours of rest, the subjects could have short intervals of rest between loads. For example, 20 N of load could be applied to the supraspinal ligament then 10 minutes of rest. This could continue for eight hours to simulate a full workday. The effects of this on the ligament could then be determined using similar principles as in this study. Another future research possibility is using various frequencies of cyclical loading and examining if this has unique effects on creep and the recovery of reflexive muscular activity.

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APPENDIX --PUBLICATION--“NEUROMUSCULAR DYSFUNCTION
ELICITED BY CYCLIC LUMBAR FLEXION”

ABSTRACT: An attempt was made to develop an *in vivo* model that could explain the neurophysiological and biomechanical processes active in the development of the idiopathic low back disorder common in workers who perform repetitive lifting tasks in industry. Passive cyclic flexion of the feline lumbar spine at 0.1 Hz for 20 min resulted in creep of the supraspinous ligament and other lumbar viscoelastic tissues as well as spasms superimposed on a decreasing electromyogram (EMG) elicited reflexly from the multifidus muscles. Rest for 7 h did not allow full recovery of the viscoelastic creep; the multifidus EMG gradually increased with initial and delayed hyperexcitability. Increasing the peak load of the cyclic flexion resulted in larger creep in the passive tissues and required a longer time for recovery of reflex EMG activity and longer delayed hyperexcitability, but development of spasms and hyperexcitability was unaffected. It is conceivable that damage to the viscoelastic tissues elicits an inflammatory process that in turn triggers a transient neuromuscular disorder. The present findings provide a biomechanical and neurophysiological explanation for a common idiopathic low back disorder as well as for the development of a cumulative trauma disorder often seen in workers engaged in repetitive lumbar flexion.

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NEUROMUSCULAR DYSFUNCTION ELICITED BY CYCLIC LUMBAR FLEXION

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Workers engaged in occupational activities requiring prolonged static and cyclic activities report up to ten times more musculoskeletal disorders than the general population.⁴⁰ Such occupational disorders, specifically low back disorders (LBDs), are a costly burden on society. Repetitive (cyclic) lumbar flexion (such as in loading and unloading boxes) causes the development of creep (e.g., elongation) of the various lumbar viscoelastic tissues (e.g., ligaments, disks, and joint capsules).^{1,4,13,18,21} The ligaments, when creep has developed, are longer and lax, whereas the disks lose fluid and have a deformed collagenous shell.¹ The creep in these viscoelastic tissues may introduce laxity in the intervertebral joint and the possibility of excessive motion and injury. Yet, the

overall stiffness of the intervertebral joint is dependent mostly on the forces developed by various lumbar muscles.^{19,21,24} Normal and balanced muscle function is, therefore, paramount for the stability and safety of the lumbar spine.³⁵

Ligaments in most joints are endowed with mechanoreceptors,^{26,27,34,43} and reflex activation of muscles associated with the stability of that joint is elicited when these afferents are excited.^{32,34} Similarly, afferents exist in the lumbar ligaments, disks, and capsules,^{14,25,42} and the reflex activation of the lumbar musculature occurs when these tissues are stimulated mechanically or electrically.^{15,16,35,39} Furthermore, passive cyclic lumbar flexion with constant displacement drastically decreases reflex activation of the lumbar muscles and requires more than 6–7 h of rest to fully recover.^{7,17,36} Thus, decrease in the reflex activation of the lumbar muscles compounded by degradation in the mechanical properties of the spinal viscoelastic structures may have an important role in the development of LBD.

Significant differences exist in the mechanical responses of the spinal tissues when subjected to

Abbreviations: CTD, cumulative trauma disorder; EMG, electromyogram; LBD, low back disorder; NIEMG, normalized integrated EMG

Key words: creep; cyclic; electromyography (EMG); disorder; ligaments; muscles; reflex; spine

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flexion under constant load as opposed to constant displacement.⁸ The neurological responses to cyclic lumbar flexion under constant peak load are unknown. It is also not known whether different peak loads have an impact on the neurological response of the lumbar musculature.

The objective of this investigation was to determine the response of the reflex muscular activity of the multifidus muscles to a short period of cyclic lumbar flexion in load control followed by a long period of rest. It was also of interest to assess whether variation in the peak flexion load amplitude affected the responses during flexion and in the rest period.

METHODS

Preparations. Twenty-five adult cats, weighing 4.0 ± 0.65 kg, were anesthetized with a single injection of chloralose (60 mg/kg) in a protocol approved by the institutional animal care and use committee. The skin directly over the lumbar spine was dissected from the thoracic to sacral level and allowed to retract laterally, exposing the intact dorsolumbar fascia. The preparation was then placed in a rigid stainless-steel frame that allowed isolation of the lumbar spine by external fixation. A gauze pad soaked with saline was applied over the incision throughout the experiment to prevent the exposed tissue from drying.

Instrumentation. Three pairs of stainless steel fine-wire EMG electrodes were inserted, via hypodermic needles, into the multifidus muscles at L-3/4, L-4/5, and L-5/6, on the right side, 5–6 mm from the midline. The wire electrodes were insulated except for a 1.0-mm exposed tip, and the interelectrode distance of each pair was 3–4 mm. A ground electrode was inserted in the gluteus muscle. Each electrode pair constituted the input to a differential amplifier with a 110-dB common mode rejection ratio, a gain capability of up to 200,000, and a band-pass filter of 6–500 Hz. EMG responses from each channel were monitored on oscilloscopes and stored in a computer at a sampling rate of 1000 Hz.

An S-shaped stainless-steel hook was inserted around the middle part of the L-4/5 supraspinous ligament and connected to the vertical actuator of a Bionix 858 Material Testing System (MTS, Inc., Minneapolis, MN). The load was applied by the MTS actuator with a computer-controlled loading system operated in load-control mode. The vertical displacement of the actuator and the load-cell output incorporated in it were also sampled into the computer along with the EMG data.

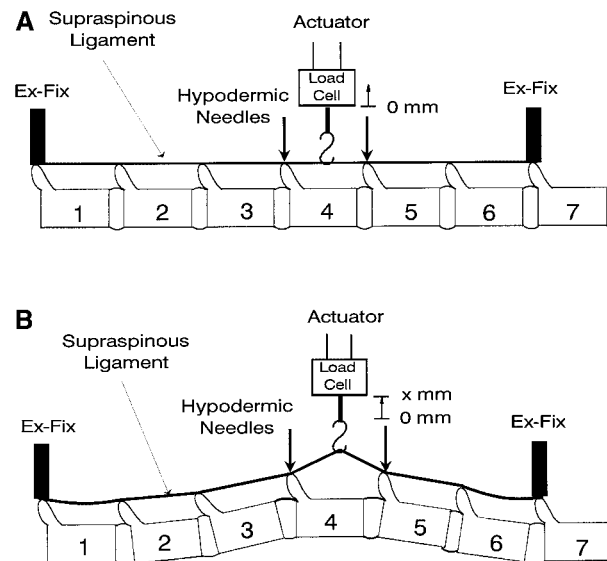


FIGURE 1. Schematic representation of the experimental arrangement showing the lumbar spine at rest (A), and during peak flexion (B).

Two external fixators were used to isolate the lumbar spine: a first fixator to the L-1 posterior spinal process and a second fixator to the L-7 process. The external fixation was intended to limit the elicited flexion to the lumbar spine and to prevent interaction of thoracic and sacral/pelvic structures, but not to prevent any motion. A schematic of the experimental set-up is shown in Figure 1.

Protocol. The stainless-steel hook applied to the L-4/5 supraspinous ligament was pulled up by the MTS actuator from a resting position with a preload of 1 N applied just before a 20-min cyclic load period, immediately after the 20-min cyclic load period was terminated, and immediately after a 7-h rest period. The cyclic load consisted of a sinusoidal waveform of 0.1-Hz frequency. Vertical displacement (in millimeters) at the L-4/5 supraspinous ligament was measured from an MTS actuator sensor on each occasion when the tension was 1 N. Two short hypodermic needles were inserted into the spinous processes of L-4 and L-5. The length of the supraspinous ligament between these two needles was measured by using a digital electronic caliper immediately before and after the load application and at the end of the rest period, while the static tension was reset to 1 N. The vertical displacement values at 1-N load and L-4/5 supraspinous ligament length were used to estimate the creep in the L-4/5 supraspinous ligament. Electromyograms from the three multifidus muscles, load, and displacement were recorded continuously during one loading period. During the rest

period, brief 10-s single-cycle tests of 0.1 Hz were applied to assess vertical displacement and EMG recovery. Tests were applied after 10, 30, and 60 min of rest, and every hour thereafter.

The same protocol was used for each of three different peak loads of 20 N ($N = 6$), 40 N ($N = 7$), and 60 N ($N = 7$). Each group was subjected to only one load magnitude. The load values were selected to cover the complete range from just above the reflex excitation threshold load of the ligament (15 N) to just below the maximal physiological strain of the ligament (70 N), as found in pilot studies.³⁹ The creep (at 20 min) and residual creep (at the end of 7 h, recovery) values were calculated separately for each of the three loads applied. Five preparations ($N = 5$) were used as controls. In this set of animals, the dissection and other arrangements were performed as usual, but the animals were not subjected to loads and were left undisturbed for the same period (20 min, plus 7 h). Only EMG was recorded from this control group, and if EMG spasms or changes above baseline occurred in these animals, the results from the experimental group would have had to be considered as unrelated to the applied load.

Analysis. A 10-s window of the EMG from L-3/4, L-4/5, and L-5/6, the associated load cycle, and the vertical displacement at the L-4/5 supraspinous ligament were sampled immediately at the beginning of the 20-min loading period and every 20 s for the first minute. For the remaining 19 min, samples were taken at 1-min intervals. During the 7-h recovery, each 10-s test was also treated as follows. Each EMG sample was integrated over the 10 s and normalized with respect to the integrated electromyogram of the first cycle in the 20-min loading period, to yield the normalized integrated EMG (NIEMG). The NIEMGs of all preparations subjected to the same peak load at the respective window were pooled, and the mean and standard deviation were calculated and plotted on an NIEMG vs. time plot for each of the peak loads used in this study. The NIEMG was selected to eliminate any interpreparation differences such as size and appropriateness of electrode location and contact in the tissue. The NIEMG will also smooth the raw EMG to some extent, allowing better representation of the overall muscular activity over time and estimation of possible force changes.

The displacements of the respective window of all preparations subjected to the same peak load were also pooled and presented as mean displacement (\pm SD) vs. time. Analysis of variance with repeated measures was applied to the displacement data to

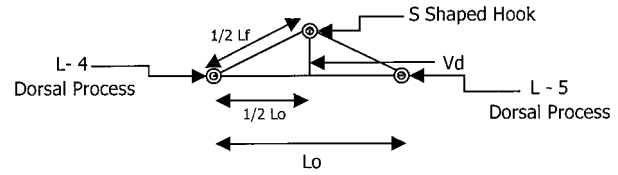


FIGURE 2. Schematic representation of the supraspinous ligament loaded with a 1-N test load before and after 20-min cyclic loading, and at the end of the recovery (rest) period, to determine the creep that developed in the ligament due to cyclic loading and the residual creep after recovery.

determine whether changes in displacement over time and with respect to the three load levels (20 N, 40 N, and 60 N) were statistically significant.

The measurements of the supraspinous ligament length at 1 N preload before and immediately after the 20-min load was applied and immediately after the recovery period, and the associated vertical displacement of the supraspinous ligament, were used to calculate the creep and residual creep, respectively, in the ligament by using eqs. (1) and (2), derived from Figure 2.

$$Lf = 2\sqrt{(\frac{1}{2}Lo)^2 + Vd^2} \quad (1)$$

$$\text{Creep} = \frac{Lf - Lo}{Lo} * 100\% \quad (2)$$

where Lo is the resting distance between the two hypodermic needles inserted into L-4 and L-5 processes, Vd is the vertical displacement of the MTS crosshead, and Lf is the final length of the supraspinous ligament while the load was 1 N. All measurements are in millimeters, and the creep is expressed as percentage elongation of the ligament.

Model Development. The pooled NIEMG data from each of the three lumbar levels from the multifidus muscle as well as the displacement recorded from the load cell were fitted to a model, in the form of an exponential function. An exponential model was chosen because it represents the classic response of viscoelastic materials to loads or elongation. The model structure for NIEMG and actuator displacement in the loading period was similar to the one developed by Solomonow et al.,^{17,36} which takes the form shown in eqs. (3) and (4), respectively. All constants were unitless, since the EMG was normalized with respect to the peak discharge at the beginning of the 20-min loading session to yield 1.0.

For the NIEMG:

$$\text{NIEMG}(t) = Ae^{-t/T_1} + \text{NIEMG}_{ss} \quad (3)$$

where A is the exponential component initial amplitude (unitless), T_1 is the exponential decay time constant (in minutes), NIEMG_{ss} is the steady-state NIEMG amplitude (unitless), and t is time. The displacement followed an exponential model:

$$\text{DISP}(t) = D_0 + D_L(1 - e^{-t/T_2}) \quad (4)$$

where $\text{DISP}(t)$ is the actuator vertical displacement as a function of time (in millimeters), D_0 is the elastic component amplitude of displacement (in millimeters), D_L is the viscoelastic component amplitude (in millimeters), T_2 is the time constant (in minutes), and t is time.

The models defined in eqs. (3) and (4) were applied to the means of each of the collected data sets associated with each of the three load levels used.

Similarly, exponential models were chosen to describe the NIEMG and displacement during the 7-h recovery period. The model for the displacement was:

$$\text{DISP}(t) = D_0 + R + (D_L - R)e^{-t/T_3} \quad (5)$$

where D_0 is the elastic component amplitude of displacement (in millimeters), D_L is the viscoelastic component amplitude at the end of 20 min (in millimeters), R is the residual creep at the end of recovery (in millimeters), and T_3 is the recovery time constant (in minutes).

For the NIEMG, the model format was:

$$\begin{aligned} \text{NIEMG}(t) = & E(1 - e^{-t/T_4}) + tBe^{-t/T_5} \\ & + C(t - T_d)e^{-(t - T_d)/T_6} + \text{NIEMG}_0 \quad (6) \end{aligned}$$

where $E(1 - e^{-t/T_4})$ represents the steady-state recovery component, tBe^{-t/T_5} is a transient hyperexcitability component, and $C(t - T_d)e^{-(t - T_d)/T_6}$ the delayed transient hyperexcitability (“morning after”). This term becomes functional only for $t \geq T_d$. NIEMG_0 represents the residual response at the end of 20-min constant load (unitless).

In this model, the constraint of $E + \text{NIEMG}_0 = 1$ is used to ensure that full recovery results in a normal (unity) response. E , B , and C are unitless. T_4 , T_5 , T_6 , and T_d are expressed in minutes.

The second and third terms, therefore, are transient features that first increase and then reverse (decrease) over time to finally arrive to near zero as the effect of hyperexcitability diminishes with rest. Furthermore, the third term, which represents the delayed hyperexcitability, becomes effective only af-

ter $t \geq T_d$; that is, the effect of this term is null until recovery time exceeds T_d . Overall, the model provides a unique prediction of the NIEMG at any given time during a rest period following a cyclic loading period.

The parameters for all models fitted were obtained by using the Marquardt–Levenberg nonlinear regression algorithm.

RESULTS

Three typical recordings of EMG, displacement, and the corresponding cyclic loads of 20 N (top), 40 N (middle), and 60 N (bottom) are shown in Figure 3. In general, the peak displacement, representing the overall creep that developed in the viscoelastic tissues of the spine, demonstrated the development of creep as expressed by an exponential-shaped increase during the 20-min cyclic loading. This was followed by a decrease in displacement during the recovery period, indicating that recovery of the creep in the tissue toward its baseline properties was underway.

The EMG discharge in response to each stretch-release cycle demonstrated a slow gradual decrease in peak-to-peak amplitude with time during the 20 min of cyclic loading, followed by a gradual increase during the recovery period. Frequently, EMG bursts were evident during the cyclic loading. They were triggered randomly and unpredictably, sometimes in midcycle and during each of several following cycles, and at other times continuously over one to three cycles. Figure 4 shows two typical bursts in a time-expanded scale of the top two traces of Figure 3, that is, for 20- and 40-N loads. Figure 5 displays the displacement and NIEMG from L-3/4, L-4/5, and L-5/6 from the pooled data at each of the peak cyclic loads of 20 N, 40 N, and 60 N.

Viscoelastic Tissue Creep. The vertical displacement of the supraspinous ligament indirectly represents the overall creep that developed in the viscoelastic tissue (ligaments, disks, and capsules) of the lumbar spine.

The mean initial peak displacement of the first cycle of loading at 20 N peak was 5.9 mm, and it gradually increased throughout the loading period, exhibiting the development of creep within the viscoelastic tissues. The mean displacement reached 10.6 mm at the end of the 20-min loading period, a 79.6% increase. At the end of the 7-h recovery period, the mean displacement decreased to 7.57 mm, representing a 28.3% residual displacement due to

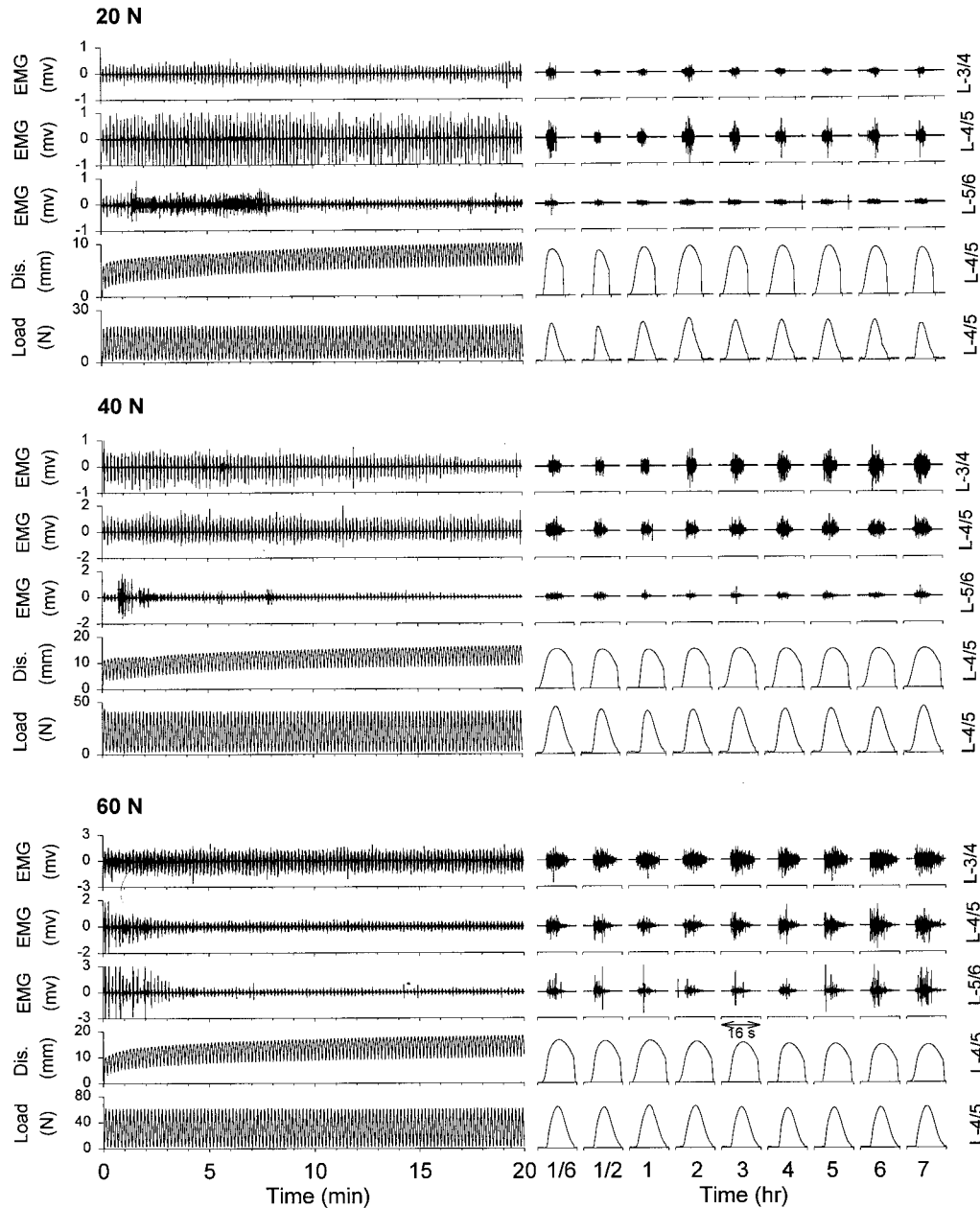


FIGURE 3. Typical EMG recordings from the L-3/4, L-4/5, and L-5/6 motion segments and the displacement and load associated with cyclic loading with 20-N (top), 40-N (middle), and 60-N (bottom) loads. The test cycles during the recovery period are shown on an expanded time scale after the 20-min marker and up to 7 h.

creep (e.g., the residual displacement was 28.3% larger than the initial displacement of 5.9 mm). The displacement decreased in an exponential-like pattern throughout recovery, and none of the preparations reached full recovery within the 7 h of rest. The analysis of variance of the displacement with respect to time indicates that the changes were statistically significant ($P < 0.0001$).

The mean displacement associated with the first cycle with peak 40-N load was 11.99 mm fol-

lowed by an exponential-like increase to the end of the 20 min, culminating with a peak mean displacement of 16.6 mm, a 38.4% increase due to creep development in the viscoelastic tissues. During the recovery period, the displacement decreased in an exponential fashion, reaching a mean value of 14.04 mm at the end of 7 h, which corresponding to 17.1% residual displacement (or 17.1% larger than the initial displacement of 11.99 mm). Full recovery of the displacement was not

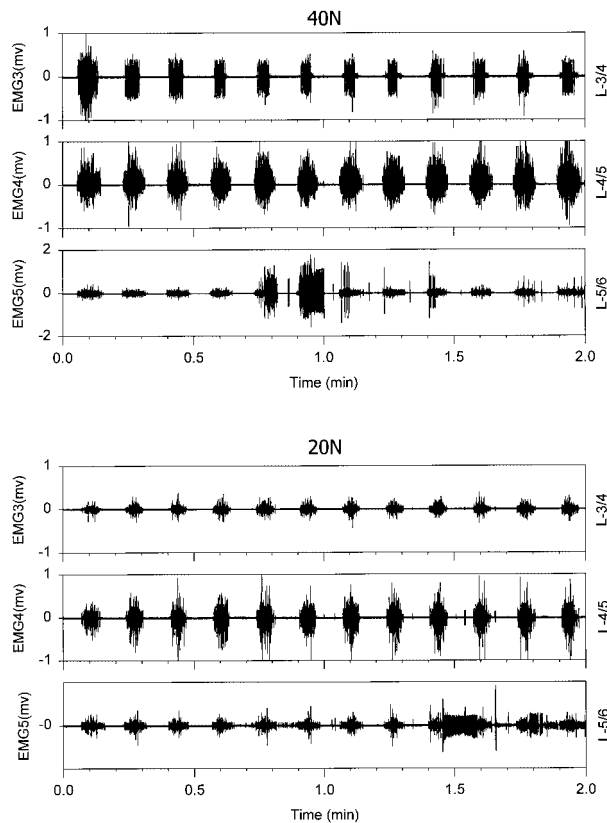


FIGURE 4. Expanded time scale of the first 2 min of a typical EMG recording from Figure 4 for the 20-N (bottom) and 40-N (top) loads showing spasms superimposed on the reflex EMG activity elicited by the lumbar flexion. In the bottom panel, spasms are evident as the outbursts after the 1.5-min marker in the L-5/6 multifidus and as large-amplitude compound action potentials superimposed on the reflex EMG activity. In the top panel, spasms are superimposed on the reflex EMG activity after the 0.8-min marker for the L-5/6 level and as large-amplitude compound action potentials superimposed on the EMG in the last few cycles.

observed in any of the preparations subjected to this load. Similarly, the changes in displacement over time were statistically significant ($P < 0.0001$).

The mean displacement associated with the first loading cycle at 60 N was 12.56 mm and increased exponentially throughout the 20 min to 20.6 mm, a 64% increase. The displacement throughout the recovery period decreased exponentially to 18.1 mm at the end of 7 h. This corresponds to 44.2% residual displacement due to creep in the ligaments, disks, and capsular tissues. Full recovery was not apparent in any of the preparations subjected to this load. The changes of displacement with time were statistically significant ($P < 0.0001$) for this group as well.

The statistical analysis also confirmed that the changes in displacement for the 20-, 40-, and 60-N loads were statistically different from each other ($P < 0.0001$).

Supraspinous Ligament Creep. The mean creep of the supraspinous ligament subjected to the 20-N cyclic load for 20 min was $7.01 \pm 4.37\%$. The mean residual creep at the end of the 7 h of rest was $2.83 \pm 1.02\%$. Full recovery of the creep was not observed in any of the preparations subjected to this load.

Similarly, the mean creep calculated for the preparations subjected to 40-N peak cyclic load was $10.5 \pm 5.96\%$ at the end of the 20-min load. The mean residual creep of the supraspinous ligament was $4.71 \pm 1.45\%$ at the end of 7 h of rest. Full recovery was not observed in any of the preparations.

For the 60-N peak load, the mean creep after 20 min of cyclic load was $26 \pm 14.2\%$, and a residual creep of $5.07 \pm 2.35\%$ was present after 7 h of rest. Full recovery was not evident in any of the preparations in this group.

The pattern observed, therefore, shows that higher peak loads resulted in larger creep at the end of the loading period as well as larger residual creep after 7 h of rest. Larger loads, however, were associated with a higher percentage of creep recovery with rest.

EMG Response. For the cyclic load with a peak load of 20 N, the mean NIEMGs decreased at the end of the 20 min to 77%, 59%, and 57% of the initial values for the multifidus muscles of L-3/4, L-4/5, and L-5/6, respectively. At the end of the first 10 min of rest, the mean NIEMGs for the same spinal levels recovered to 88%, 88%, and 77% of the initial values. NIEMGs temporarily decreased thereafter to 77%, 71%, and 66% at the end of the first hour of rest, and then started to increase again. At the end of 7 h of rest, the mean NIEMGs of the L-3/4, L-4/5, and L-5/6 were 109%, 134%, and 126% of the initial values.

For cyclic loading with a peak load of 40 N, mean NIEMGs decreased at the end of 20 min to 63%, 65%, and 64% for L-3/4, L-4/5, and L-5/6, respectively. The first 10 min of rest resulted in an increase of mean NIEMGs to 74%, 89%, and 91%, for the respective spinal levels. NIEMGs decreased thereafter to the end of the first rest hour and then started increasing again. At the end of 7 h of rest, mean NIEMGs were 136%, 134%, and 110% of initial values (i.e., the beginning of the loading period).

The preparations subjected to a peak load of 60 N demonstrated a decrease in mean NIEMGs during

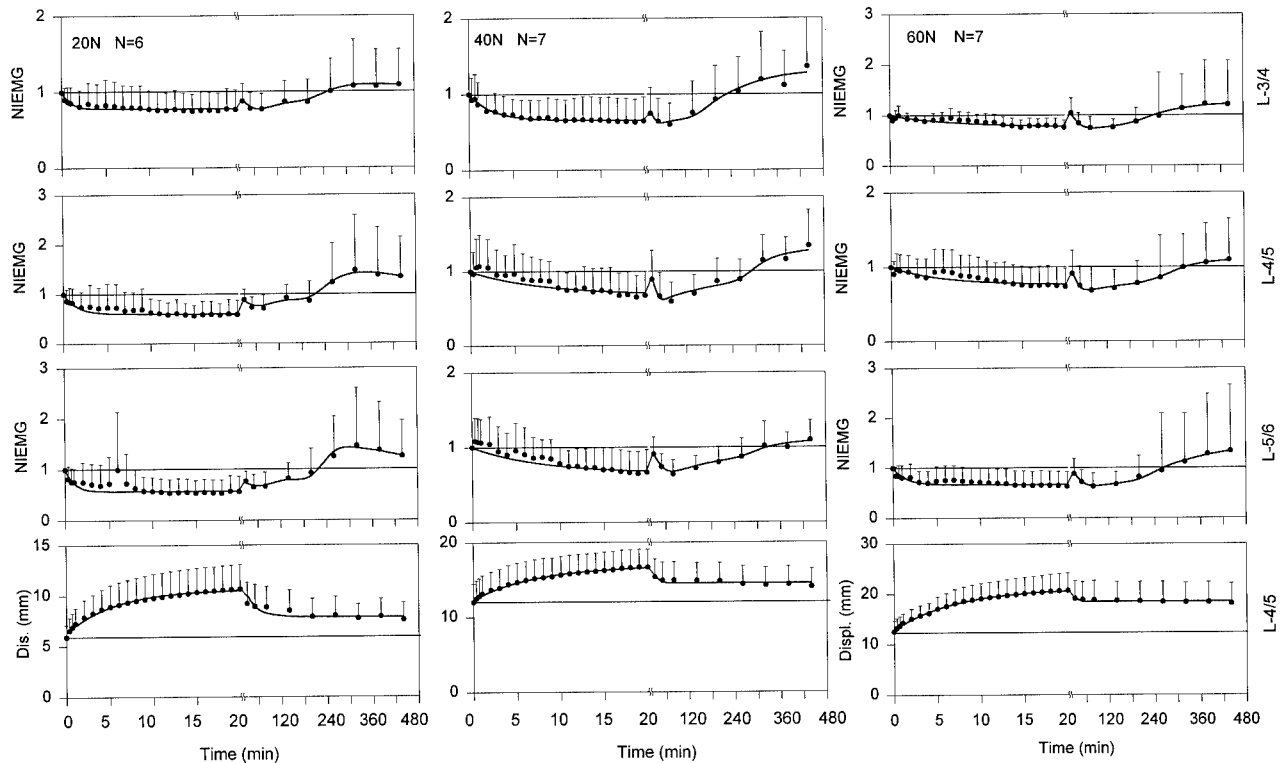


FIGURE 5. The mean and standard deviations of the pooled data from all preparations subjected to 20-N (left), 40-N (middle), and 60-N (right) loads. The models developed for the data are shown superimposed on the data.

the 20 min of cyclic loading, ending with 79%, 75%, and 64% of the initial values for L-3/4, L-4/5, and L-5/6, respectively. The first 10 min of rest allowed for recovery of mean NIEMGs to 104%, 91%, and 88% for the same respective spinal levels. At the end of the first hour of rest, NIEMGs decreased to 75%, 69%, and 63%, respectively, and increased thereafter. At the end of the 7-h recovery period, mean NIEMGs reached 120%, 110%, and 133% of the initial values for L-3/4, L-4/5, and L-5/6, respectively.

Control Group. The EMG data recorded from the control group, which was not loaded at all, remained at the baseline level throughout the 20-min period when loading occurred in the experimental groups, plus a 7-h rest period. This confirmed that the spasms and EMG changes recorded from the exper-

imental groups were directly related to the load applied and not to any other artifactual factor associated with the protocol.

Displacement Model

The displacement model described in eq. (4) was fitted to the mean data collected during the 20-min cyclic loading, and the constants as well as the r^2 values resulting from the statistical analysis are given in Table 1 for the three peak loads. The r^2 values exceeded 0.99 for the models of each of the three loads indicating that an exceptionally accurate description of the physiological data was achieved.

For the 7-h recovery period, the constants associated with eq. (5) are presented in Table 2. In this model, the component R represents a residual of the displacement that may or may not be fully recovered in the short term, that is, in a matter of days. The

Table 1. Vertical displacement model during cyclic loading.

Load	D_o	D_L (mm)	T_2 (min)	r^2
20 N	6.321	4.251	5.488	0.9984
40 N	12.34	4.49	7.209	0.9923
60 N	12.88	8.16	7.042	0.9975

Table 2. Vertical displacement model during recovery.

Load	D_o	D_L (mm)	T_3 (min)	R	r^2
20 N	6.321	4.251	33.3	1.618	0.855
40 N	12.34	4.49	11.2	2.175	0.819
60 N	12.88	8.16	7.6	5.610	0.881

Table 3. NIEMG models during cyclic loading.

Load	Level	NIEMG _{ss}	A	T_1 (min)	r^2
20 N	L-3/4	0.778	0.22	1.0	0.822
	L-4/5	0.597	0.463	1.5	0.752
	L-5/6	0.57	0.43	1.0	0.364
40 N	L-3/4	0.654	0.346	2.7	0.977
	L-4/5	0.68	0.32	8.3	0.789
	L-5/6	0.65	0.35	8.0	0.801
60 N	L-3/4	0.76	0.24	8.0	0.700
	L-4/5	0.76	0.24	6.0	0.707
	L-5/6	0.66	0.34	1.5	0.72

statistical analysis points out that the model represents a good fit to the physiological data as r^2 ranged from 0.81 to 0.88.

The actual models are superimposed on the experimental data shown in Figure 5.

EMG Model. The model representing EMG behavior during the 20 min of cyclic loading is given in eq. (3), and the constants derived by the analysis are shown in Table 3 for each of the spinal levels evaluated. The statistical analysis indicates that a fair estimation of the physiological data was obtained as r^2 ranged mostly from 0.707 to 0.977, the only exception being the model for the L-5/6 level subjected to 20-N peak load, which resulted in a poor fit ($r^2 = 0.364$). It is noted that the spasms were superimposed on the EMG, causing a deterioration of the model fit to the actual data, as discussed later.

For the recovery period, the proposed model is given in eq. (6), and the constants determined by the analysis are given in Table 4. A very good fit to the physiological data was obtained as the r^2 values ranged from 0.889 to 0.982. Table 4 confirms that the different peak loads applied to the respective experimental groups resulted in different responses. The time constant T_4 , which governs the recovery rate of reflex EMG activity during rest, indicates that higher loads required longer rest time for the reflex

EMG to recover to its initial level observed at the beginning of 20-min cyclic flexion.

Table 4 also confirms that the duration of the delayed hyperexcitability depended on the load, as evidenced by the manner that T_6 changed with the load. Larger loads resulted in longer delayed hyperexcitability; that is, the period required for the delayed hyperexcitability to diminish was longer for heavier loads. Indeed, Figure 5 demonstrates that, for the 20-N load, the NIEMGs at the end of the recovery periods tended to decrease, indicating that the delayed hyperexcitability was diminishing. In the NIEMG patterns of the mean data for the 40- and 60-N loads, the late phase of the recovery period was still increasing or just leveling off to a peak.

DISCUSSION

We found that 20 min of cyclic lumbar flexion results in a multifactorial neuromuscular disorder lasting well beyond 7 h of rest. The neuromuscular disorder consists of four distinct components: spasms and reduced reflex activity of the multifidus during the cyclic flexion, followed by initial and delayed hyperexcitability over the rest period.

Spasms appeared frequently during the cyclic flexion period and were unpredictable in timing, duration, intensity, or frequency of appearance. The appearance of spasms indicates that some type of tissue damage is present.²⁵ Spasms and increased activity of posterior lumbar muscles have been confirmed electromyographically in patients with idiopathic and pathological low back pain.^{5,11,12,22,28,30} It is conceivable, therefore, that the chain reaction of tissue damage leading to pain and spasms is the process that took place in the cyclic loading of this investigation. Indeed, the literature confirms that prolonged static or repetitive exposure of tendons or ligaments to loads within their physiological limits may result in creep and microtrauma to the collagen fibers.^{2,3,6,29,38} A ligament in such a state displays a

Table 4. NIEMG model during recovery.

Load	Level	E	T_4 (min)	B	T_5 (min)	C	T_6 (min)	T_d (min)	NIEMG _{ss}	r^2
20 N	L-3-4	0.35	150	0.06	10	0.0030	120	220	0.65	0.889
	L-4-5	0.44	100	0.07	10	0.0100	120	220	0.56	0.978
	L-5-6	0.49	140	0.05	10	0.0130	100	200	0.51	0.979
40 N	L-3-4	0.44	300	0.10	6	0.0035	300	150	0.56	0.942
	L-4-5	0.46	210	0.18	6	0.0044	210	250	0.54	0.934
	L-5-6	0.45	210	0.09	10	0.0018	210	250	0.55	0.916
60 N	L-3-4	0.33	300	0.09	10	0.0030	270	180	0.67	0.960
	L-4-5	0.37	350	0.10	8	0.0027	200	240	0.63	0.935
	L-5-6	0.45	350	0.09	10	0.0035	400	190	0.55	0.982

disorganized fiber structure and degradation of its original functional properties (i.e., stress–strain relations). As investigated elsewhere, the supraspinous ligament was strained well below its physiological limits^{24,41} and the creep that developed at the end of 20-min cyclic flexion ranged from 7% to 26%, confirming creep and microdamage in the viscoelastic tissues.^{2,3,6} With microdamage of the collagen fibers, bare nerve endings in the lumbar ligaments, disks, and capsules^{14,25,42} become active, relaying pain signals and eliciting random reflex muscle activation (i.e., spasms of the multifidus).

The receptors in the lumbar viscoelastic tissues are both fast- and slow-adapting. Slow-adapting receptors exhibit an initial high rate of discharge upon application of a sufficient stimulus. The discharge rate becomes reduced as steady-state conditions are reached, remaining relatively stable as long as the stimulus remains stable. Fast-adapting receptors, however, exhibit a high rate of discharge upon stimulus application, and a relatively fast decay to complete silence. The compounded response of the fast- and slow-adapting receptors yields an initially high discharge rate that reflexly activates the muscles at a higher level than in the steady state when the sum of the afferent discharge is moderately decreased. Furthermore, we previously found that afferents seem to be more responsive to a tension stimulus when compared with an elongation stimulus.³³ Since the stimulus applied in this investigation consists of constant peak load (as opposed to elongation), the decrease in the reflexly elicited EMG was relatively small compared with the large decrease in EMG observed with constant peak elongation stimulus.³⁵ Overall, the decrease in the reflex activation level of the multifidus muscles results in reduced stiffness of the lumbar spine and therefore in diminished stability and increased exposure to injury.²³

The model describing the decreasing EMG during the 20 min of load-control cycling flexion demonstrated that most phasic activity diminishes within the first 5–10 min, after which further decrease is minimal. It should be noted that the mean NIEMG values in Figure 5 were not influenced by the exponentially increasing creep alone, as the effect of the spasms was also present. This causes several data points to be placed outside the predicted model, and in turn to also artificially decrease the r^2 value. However, the model represents only one of the two components present in the data—that is, the phasic decrease in EMG due to the phasic transient response of the fast and slow mechanoreceptors. The spasm component is not represented in the model. It should also be noted that compounding the experi-

mental data from all the preparations subjected to the same load attenuates the effect of the spasms to some degree, but does not eliminate it completely. Therefore, although the r^2 values resulting from the model fit are reasonable, ranging from 0.707 to 0.977 (except the L-5/6 level at 20 N, which yielded $r^2 = 0.364$), this value could have been drastically improved if it had been possible to isolate and remove the effect of the spasms.

The model of muscular activity during the 7-h recovery period was relatively complex, with three separate exponential components. The first component represented the gradual recovery of the EMG toward its original level. As the lumbar spine was resting, the creep was recovering and with it the phasic response of the mechanoreceptors. The time constant for this component (T_4) had a range of 100–150 min for the group subjected to a 20-N load, 210–300 min for the group subjected to a 40-N load, and 300–350 min for the preparations loaded with a 60-N load. The model, therefore, indicates that larger loads require longer rest to allow reflex muscular activity to recover to its original level. Larger creep develops in response to larger loads, and requires a longer time to recover.

The initial hyperexcitability at the beginning of the recovery period was diminished nearly completely within the first hour. Indeed, Table 4 shows that T_5 ranged from 6 to 10 min and that this time constant was not substantially different for different load magnitudes. A substantial portion of the creep recovered during the first hour of the rest period. During that hour, the hyperexcitability of the muscles increased the stiffness of the intervertebral joint, thereby limiting further damage to the viscoelastic tissues. The majority of creep recovered in the first hour for all three load levels. Apparently, this did not require large increases in T_5 for larger loads, as there was no pattern of change in this time constant for increasing loads. Although microdamage in collagen fibers has been demonstrated with prolonged static or repetitive strain of ligaments and tendons, it is still difficult to diagnose such damage clinically, leading to the designation “idiopathic low back pain.”

This observation provides an important clue to the physiological/biomechanical explanation of what is commonly called cumulative trauma disorders (CTDs), diagnosed in workers subjected to long-term activities involving repetitive motion.^{31,40} Such workers, if exposed to daily lifting tasks for several hours a day, will develop significant creep in the lumbar viscoelastic tissues. The creep will not fully recover overnight, and the individual will start

the next day of work with residual creep. The creep developed in the second work day will be compounded with the residual creep, and so on. Over the long term, a significant residual creep will accumulate in the tissues, accompanied by chronic inflammation.²⁹ Chronic inflammation of tendons or ligaments is a difficult disorder to treat,²⁹ rendering the worker disabled with a chronic low back disorder.

As indicated previously, the load magnitude had an impact on the time constant (T_4) that governed the recovery of reflex EMG activity with rest. Larger loads required more time for the EMG to return to its original level. The lumbar displacement behavior was also dependent on the magnitude of the load applied, as shown in the Results section. In essence, larger loads resulted in larger creep at the end of 20 min, but showed a larger percentage of recovery. The final residual displacement, however, was largest for the largest load of 60 N. This did not manifest in more spasms, a larger decrease in reflex EMG activity, or in more initial hyperexcitability in the recovery period.

It was somewhat surprising that the smallest load of 20 N was sufficient to elicit a neuromuscular disorder. This low-magnitude load was just above the trigger force threshold of the reflex EMG from the multifidus, which was found to be 15 N.³⁹ This suggests that once residual creep develops in the viscoelastic tissues, the microdamage is done and the chain reaction of damage–pain–spasms–hyperexcitability is triggered.

Hagg reported disorganized spasms in the hand/wrist and shoulder muscles of workers performing at a very low force level (5% of maximal voluntary contraction) over prolonged periods.^{9,10} The data obtained herein support Hagg's observation and suggest that creep, regardless of the load applied, is the source of the spasms. Such a phenomenon may exist in other joints and their respective muscles when subjected to prolonged static or repetitive activity.

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VITA

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