Frequency-dependent changes in neuromuscular responses to cyclic lumbar flexion

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Abstract

Repetitive lifting in the workplace has been identified to be a cause of low back disorders. Epidemiologic data further supports an hypothesis that higher repetition rate (i.e., frequency) is an added risk factor. The objective of this study was to provide experimental data testing the above hypothesis. An in vivo feline model was subjected to 20-min of cyclic lumbar loading at frequencies of 0.1 Hz and 0.5 Hz while monitoring the EMG from the L-3/4–L-5/6 multifidus muscles and the creep at the L-4/5 level. Seven hours of rest were allowed after the cyclic flexion/extension was terminated. During this rest period, a single test cycle was performed every hour to assess recovery of EMG and lumbar creep. The results demonstrate that cyclic lumbar flexion elicits a transient neuromuscular disorder consisting of EMG spasms during the cyclic loading and initial and delayed muscular hyperexcitabilities during the rest period. Cyclic loading at 0.5 Hz resulted in significant ($p < 0.05$) increase in the hyperexcitability magnitude and duration during the recovery period. It was concluded that repetitive lumbar loading at fast rates is indeed a risk factor as it induces larger creep in the lumbar viscoelastic tissues which in turn intensify the resulting neuromuscular disorder.

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1. Introduction

Repetitive motion in the occupational environment has been identified as a risk factor for neuromuscular disorders in epidemiological studies (Silverstein et al., 1986; NIOSH, 1996, 1999; Department of Labor, 1995; Pope, 1996). Specifically, workers engaged in activities which require repetitive flexion/extension of the lumbar spine represent one of the most costly components of general musculoskeletal disorders based on lost wages, medical expenses, disability payments, etc. (Department of Labor, 1995; NIOSH, 1996, 1999; NAS, 2001). Silverstein et al. (1986) pointed out in an epidemiological study that the risk factors associated with repetitive motion work are multifactorial, being dependent on the magnitude of forces generated in each repetition, the number of repetitions for a given period and the duration of the work period as the primary factors.

Often, workers engaged in repetitive lumbar flexion/extension (loading/unloading boxes, assembly line workers, etc.) present with a low back disorder which cannot be diagnosed with routine medical procedures such as X-rays, MRI's, etc., due to the absence of obvious deficits as vertebral fractures, prolapsed disc, impingement, spinal cord stenosis, etc. A common denominator to this class of idiopathic low back disorders is the subjective report of pain from the patient and stiff lumbar musculature as evidenced from spasms or elevated EMG (Fisher and Chang, 1985; Haig et al., 1993; Hoyt et al., 1981; Miller, 1985; Roland, 1986; Shivonen et al., 1991). With the lack of evidence regarding the source of the symptoms, treatment of these conditions is often sub-optimal and lengthy. Our attempts to identify the source and the development of such a common idiopathic low back disorder revolved around the fact that the lumbar viscoelastic tissues (e.g., ligaments, discs and capsules) develop creep when subjected to prolonged cyclic loading (Adams et al., 1990; Ekstrom et al., 1996; Keller et al., 1989; Claude et al., 2003). Creep developed by the cyclic loading of ligaments was shown to result in micro-damage within the collagen fibers and modifications of their functional properties (Frank et al., 1985). Since the viscoelastic
tissues of the spine are endowed with a variety of mechanoreceptors (Hirsch et al., 1963; Pedersen et al., 1956; Yahia and Newman, 1991) which give rise to reflexive activation of paraspinous muscles (Indahl et al., 1995, 1997; Stubbs et al., 1998; Solomonow et al., 1998), micro-damage in these tissues can elicit a neuromuscular disorder (Williams et al., 2000; Jackson et al., 2001; Claude et al., 2003). In fact, Claude et al. (2003) demonstrated that 20-min of continuous cyclic flexion/extension of the feline spine results in substantial creep in the viscoelastic tissues as well as spasms as was evidenced from the EMG recorded from the multifidus muscles. The disorder associated with creep consisted of initial hyperexcitability of the multifidus muscles in the first hour of rest following the cyclic flexion/extension and delayed hyperexcitability several hours into the rest. It was further shown in that work that light load magnitudes resulted in significantly less severe disorder when compared to cyclic flexion/extension with larger peak loads.

Overall, the epidemiological data (Silverstein et al., 1986) is confirmed by experimental work (Williams et al., 2000; Jackson et al., 2001; Claude et al., 2003) that prolonged cyclic flexion/extension elicits a musculoskeletal disorder the extent of which is dependent on the load applied. Experimental work which determines the impact of the repetition rate (e.g., frequency) of flexion/extension on the resulting disorder is lacking. Some indirect data suggests that increase in the velocity or frequency of motion results in increased force development in the musculature (Marras et al., 1984; Marras and Mirka, 1993; Marras et al., 1995; Marras and Granata, 1997; Solomonow et al., 2001) and that may exceed the load levels which could be safely sustained by various structures (Marras and Granata, 1997). It is also known that higher rates of stretch applied to viscoelastic tissues may cause structural damage (Newman et al., 1994; Panjabi and Courtne, 2001).

In light of this findings, one can propose the hypothesis that cyclic flexion/extension of the lumbar spine at high frequencies may be a significant contributing factor to the development of neuromuscular disorders. Hence, the objective of this report was to determine the impact of the frequency of cyclic lumbar flexion/extension on the neuromuscular responses of paraspinous muscles. Specifically, the impact of the frequency of flexion/extension on the creep developed in the viscoelastic tissues and the hyperexcitabilities and spasms in the multifidus muscles were of interest.

2. Methods

2.1. Preparations

Nineteen adult cats weighing 4.1 ± 0.48 kg were anaesthetized 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 level to the 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 the isolation of the lumbar spine by external fixation (details in next section). A gauze pad soaked with isotonic saline was applied over the incision throughout the experiment to prevent the exposed tissue from drying. The preparation was placed prone on a platform with the forelimbs and hind limbs extended forward and rearward, respectively, and that constituted the resting position.

2.2. Instrumentation

Three pairs of stainless steel fine wire EMG electrodes were inserted via hypodermic needles into the multifidus muscles of the 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; 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 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 was monitored on oscilloscopes and stored in a computer at a sampling rate of 1000 Hz.

An “S” shaped stainless steel hook made of 1.5 mm rod was inserted around the middle part of the L4/L5 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.

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 fixators were made of 1 cm diameter stainless steel rods attached to an external metal frame. Each of the two rods has a Teflon cap at one end and the caps were machined to fit over the dorsal tip of the L-1 and L-2 spinous processes. 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 Fig. 1.

2.3. Protocol

The stainless steel hook applied to the L-4/5 supraspinous ligament was pulled vertically by the
MTS actuator from a resting position. The cyclic load consisted of a sinusoidal waveform of 0.1 Hz frequency. Electromyograms from the three multifidus muscles, load and displacement were recorded continuously during a 20-min loading period. During the following 7 h of rest, single cycle tests of the test frequency were applied to assess vertical displacement and EMG recovery. Tests were applied after 10-min of rest, 30-min of rest, 60-min and every hour thereafter.

The same protocol was used for each of the two different frequencies of 0.1 Hz \( (N = 7) \) and 0.5 Hz \( (N = 7) \). Each animal was subjected only to one frequency; i.e., 7 preparations were loaded only at 0.1 Hz and 7 other preparations at 0.5 Hz only. The peak load was selected to be 40 N which is in the mid-range from just above the reflex excitation threshold load of the ligament (15 N) to just below the load corresponding to the maximal physiological strain of the ligament (70 N) according to previous studies (Williams et al., 2000, Panjabi et al., 1982). Creep and residual creep were calculated separately for each of the two frequencies tested. Creep and residual creep were calculated as the percent increase in displacement at 20 min and at 7 h relative to the respective initial displacement \( D_0 \) at the very beginning of static flexion (see Fig. 3).

A third group of animals, a control group, with \( N = 5 \) was also prepared similarly with electrodes and hook but was not loaded. This was done to assert that any EMG recorded was indeed due to the load and not to any artifact associated with the protocol.

2.4. Analysis

Ten-second windows of electromyogram, from L-3/4, L-4/5 and L-5/6, the associated load cycle and 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 preparations tested at 0.1 Hz. For preparations tested at 0.5 Hz, windows of 2 s were used so a single full cycle could be analyzed. For the remaining 19 min, samples were taken at 1 min intervals. During 7 h of recovery, single cycle test windows were similarly analyzed. All windows were treated as follows; each electromyogram sample was rectified and integrated over the window duration 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 NIEMG of all the preparations subjected to the same frequency at the same time window were pooled. Means and standard deviations for each time window were calculated and plotted on a NIEMG vs. time plot for each of the two frequencies used in this study. The Normalized Integrated EMG was selected in order to eliminate any interpreparation differences such as size, electrode location and contact in the tissue, etc. The NIEMG also smooths the raw EMG to some extent, allowing better representation of the overall muscular activity over time and estimation of possible force changes.

A two-way analysis of variance (ANOVA) with repeated measures was applied to the NIEMG data from the two frequencies used. In the loading period, data were grouped by two levels of loading frequency (0.1 and 0.5 Hz) and 23 levels of time (0, 20, 40 and 60 s and every 1 min thereafter). In the recovery period, data were grouped by two levels of loading frequency and 9 levels of time (10 min, 30 min, 60 min and 2–7 h).

The displacement measured from the MTS was used to obtain a direct assessment of the shear creep of all the viscoelastic tissues (e.g., ligaments, discs, and capsules) in the lumbar spine. The displacements of each time window of all the preparations subjected to the same frequency were also pooled and presented as mean displacement \( (\pm SD) \) vs. time. Similarly, a two factor analysis of variance with repeated measures was applied to the displacement data in order to determine if changes in displacement over time and with respect to the two frequencies (e.g., 0.1 and 0.5 Hz) were statistically significant; interactions between time and frequency were determined through a post-hoc trend analysis. Significance was set at the 0.05 level.

2.5. 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 models in the form of exponential functions. Exponential models were chosen as they represent the classical response of viscoelastic materials to loads and/or elongation. The model structure for NIEMG and actuator displacement in the loading period is similar to the one developed by Solomonow et al. (2000), which takes the form shown in Eqs. (1) and (2), respectively. All constants in Eq. (1) are unitless, since the EMG was normalized with respect to the peak discharge in the beginning of the 20-min loading session to yield 1.0.

For the NIEMG:

\[
\text{NIEMG}(t) = Ae^{-t/T1} + \text{NIEMG}_{ss},
\]

where, \(A\) is the exponential component initial amplitude (unitless), \(T1\) the exponential decay time constant (in minutes), \(\text{NIEMG}_{ss}\) the steady state NIEMG amplitude (unitless), \(t\) the time (min).

The displacement followed a bi-exponential model as follows:

\[
\text{DISP}(t) = D_0 + D_{L1}(1 - e^{-t/T2}) + D_{L2}(1 - e^{-t/T3}),
\]

where \(\text{DISP}(t)\) is the actuator vertical displacement as a function of time (mm), \(D_0\) the elastic component amplitude of displacement (mm), \(D_{L1}\) the fast viscoelastic component amplitude of collagen tissues (mm), \(D_{L2}\) the slow visco-elastic component amplitude of discs (mm), \(T2\) the time constant (min), \(T3\) the Time Constant (min), \(t\) the time (min).

The models defined in Eqs. (1) and (2) were applied to the means of each of the collected data sets associated with each of the frequencies 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

\[
\text{DISP}(t) = D_0 + R + D_{R1}e^{-t/T4} + D_{R2}e^{-t/T5},
\]

where \(D_0\) is the elastic component amplitude of displacement (mm), \(D_{R1}\) the amplitude for recovery of the fast viscoelastic component (mm), \(D_{R2}\) the amplitude for recovery of the slow viscoelastic component (mm), \(R\) the residual creep at the end of recovery (in millimeters), \(T4\) the recovery time constant of the fast viscoelastic component (min), \(T5\) the recovery time constant for the slow viscoelastic component (min).

For the NIEMG, the model format was:

\[
\text{NIEMG}(t) = E(1 - e^{-t/T6}) + bEe^{-t/T7} + C(t - Td)e^{-(t - Td)/T8} + \text{NIEMG}_{0},
\]

where, \(E(1 - e^{-t/T6})\) represents the steady-state recovery component, \(bEe^{-t/T7}\) represents initial transient hyperexcitability component. \(C(t - Td)e^{-(t - Td)/T8}\) represents a delayed transient hyperexcitability (“morning after”). This term becomes functional only for \(t > T_d\). \(\text{NIEMG}_{0}\) 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 insure that full recovery results in a normal (unity) response. \(E, B, C\) are unitless. \(T_6, T_7, T_8\) and \(T_d\) are in minutes.

The second and third terms of Eq. (4), therefore, are transient features which first increase then 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 after \(t > T_d\), e.g., 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 of a rest period following a cyclic loading period.

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

3. Results

Typical responses of the L-3/4 to L-5/6 multifidus EMG as well as the associated displacement and the cyclic load from two preparations (one at 0.1 Hz and the second at 0.5 Hz) is shown in Fig. 2. In general, the reflexive EMG from the multifidi of the three levels decreased with time during the 20-min of cyclic loading. Spontaneous spasms were superimposed on the decreasing EMG. The timing, duration, intensity and lumbar level of the spasms was random, and unpredictable as is the definition of spasms. During the same period, the displacement demonstrated the effect of creep developing in the viscoelastic structures, increasing in an exponential like manner. During the recovery period, the peak displacement demonstrated a slow decrease whereas the EMG from the muscles was gradually increasing. Fig. 3 presents the mean \(\pm SD\) of NIEMG and displacement from all the preparations tested at 0.1 Hz and from all the preparations tested at 0.5 Hz during the 20-min of cyclic loading and the following 7 h of recovery.

3.1. Displacement and creep

For the data collected at 0.1 Hz the mean displacement increased from an initial value of 11.99 (\(\pm 2.46\)) mm to 16.65 (\(\pm 2.44\)) mm which corresponds to a creep of 38.9%. During the 7 h rest period, the mean displacement decreased from 16.65 (\(\pm 2.44\)) mm to 14.04 (\(\pm 2.41\)) mm, which still is 21.7% larger (or 21.7% residual creep) than the mean of the initial displacement of 11.99 (\(\pm 2.46\)) mm. Full recovery of the creep was not observed. Furthermore, the majority of the recovery occurred in the first hour, when the mean displacement decreased from 16.65 (\(\pm 2.44\)) mm to 14.9 (\(\pm 2.47\)) mm.

In the experimental group tested at cyclic loading of 0.5 Hz frequency, the mean initial displacement was 8.25
Fig. 2. Two typical recordings of the EMG from the L-3/4, L-4/5 and L-5/6 multifidus muscles as well as the associated displacement and the cyclic load of 40 N peak applied to the L-4/5 segment. The top recording is at a cyclic frequency of 0.1 Hz whereas the bottom one is at 0.5 Hz. Note the spasms in the EMG traces of the top figure at the 1–3 min markers and the associated decrease in the displacement due to the stiffening of the spine from the increased muscle force. Similarly, spasm in 17–18 min, in the lower trace, resulted in lowering of the displacement for the same reasons. The general features are gradual decrease of the EMG with time with superimposed spasms. Simultaneously, the displacement exhibits the development of creep with time as its peak amplitude gradually increases. During the recovery period, the peak displacement gradually decreases, but does not fully recover to its original rest.
The mean ± SD of the NIEMG and displacement of all the preparations tested in each of the two cyclic flexion/extension frequencies. The model developed for each of the two frequencies is superimposed on the experimental data.

Fig. 3. The mean ± SD of the NIEMG and displacement of all the preparations tested in each of the two cyclic flexion/extension frequencies. The model developed for each of the two frequencies is superimposed on the experimental data.

2.05 mm increasing to 15.63 (±1.89) mm at the end of the 20-min which corresponds to 89.4% creep. The majority of the recovery of the creep also occurred in the first hour of rest, decreasing to 12.4 (±1.96) mm. At the end of the 7 h of rest the mean displacement recovered to 11.19 (±1.63) mm, which was still 35.6% larger than the initial displacement (e.g., 35.6% residual creep). Full recovery was not observed in any of the preparations.

3.2. NIEMG

The mean NIEMG from the L-3/4, L-4/5 and L-5/6 multifidus collected at 0.1 Hz decreased from the initial
value of 1.0 to 0.63 (±0.3), 0.65 (±0.29) and 0.64 (±0.29), respectively, at the end of the 20 min of cyclic loading.

The mean NIEMG of the preparations tested at 0.1 Hz demonstrated a sharp increase followed by a decrease in the first hour of recovery. The largest increases, after the first 10 min of rest, were from 0.63 (±0.3) to 0.74 (±0.34), from 0.65 (±0.29) to 0.89 (±0.39) and from 0.64 (±0.29) to 0.91 (±0.23) for the L-3/4, L-4/5 and L-5/6 levels, respectively. At the end of the first hour of rest, the NIEMG of the same lumbar levels decreased to 0.60 (±0.28), 0.60 (±0.26) and 0.64 (±0.19), respectively. A gradual increase followed thereafter and resulted in NIEMG levels of 1.36 (±0.74), 1.34 (±0.48) and 1.1 (±0.26) in the L-3/4, L-4/5 and L-5/6, respectively, at the end of the 7 h of rest.

The mean NIEMG in the group tested at 0.5 Hz decreased throughout the 20 min of loading from 1.0 to 0.51 (±0.07), 0.44 (±0.25) and 0.4 (±0.27) in the L-3/4, L-4/5 and L-5/6, respectively. A sharp transient increase in NIEMG to 0.87 (±0.41), 0.9 (±0.47) and 0.92 (±0.27) was observed after the first 10 min of rest in L-3/4, L-4/5 and L-5/6 multifidi, respectively. The mean NIEMG of the same levels decreased to 0.83 (±0.31), 0.73 (±0.33) and 0.77 (±0.29) at the end of the first hour of rest. The mean NIEMG increased gradually thereafter, reaching values of 1.94 (±0.93), 1.78 (±0.9) and 1.79 (±0.84) in the L-3/4, L-4/5 and L-5/6 multifidi, respectively, at the end of 7 h of rest.

3.3. Statistical analysis

The statistical analysis was set up to determine statistically significant effects of time, frequency, and their interaction and is summarized in Table 1. During the 20 min loading period, significant effects of time (p<0.05) were found for all EMG channels and for the displacement. No effect of flexion frequency was found in any of the analyzed data, signifying that with the data available, no significant differences in EMG decline or displacement could be attributed to frequency during the loading period. However, significant trend interaction between time and frequency was found for the displacement, indicating that throughout the loading period, there are differences in the displacement trends between 0.5 and 0.1 Hz. Close examination of Fig. 3 reveals that the displacement obtained at a frequency of 0.5 Hz increases more rapidly than that at 0.1 Hz throughout the start of the loading period, slowly converging towards the displacement at 0.1 Hz, at the end of the loading period.

For the recovery portion of the data, the statistical analysis showed significant effect (p<0.05) of time for each of the three EMG channels and displacement. No significant effects of frequency were found in any of the three NIEMG channels, but there was a significant effect (p<0.05) of frequency on the displacement, with the displacement at 0.1 Hz being greater than at 0.5 Hz. Significant effects (p<0.05 for each of the three NIEMG channels) of interaction were found, meaning that the trends in the recovery curves are significantly different from each other; e.g. greater increases in NIEMG are found at 0.5 Hz than at 0.1 Hz. Similarly, frequency–time interaction (p<0.05) was found in the displacement recovery, indicating the steeper decrease of displacement in the 0.5 Hz data.

The statistical analysis confirms that both NIEMG and displacement are functions of time in the loading and recovery periods. Further significant differences exist in the pattern of the displacement over time for the

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Effect of frequency</th>
<th>Effect of time</th>
<th>Time-frequency interaction</th>
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<tr>
<td><strong>Loading</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L3-4 NIEMG</td>
<td>NS</td>
<td>F(11,132) = 4.93</td>
<td>NS</td>
</tr>
<tr>
<td>L3-4 NIEMG</td>
<td>NS</td>
<td>F(11,132) = 4.93</td>
<td>NS</td>
</tr>
<tr>
<td>L3-4 NIEMG</td>
<td>NS</td>
<td>F(11,132) = 4.93</td>
<td>NS</td>
</tr>
<tr>
<td>Displacement</td>
<td>NS</td>
<td>F(11,132) = 186.69</td>
<td>F(1,1,132) = 56.61</td>
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<td><strong>Recovery</strong></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>L3-4 NIEMG</td>
<td>NS</td>
<td>F(8,96) = 15.05</td>
<td>F(1,1,96) = 22.38</td>
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<td>F(8,96) = 15.05</td>
<td>F(1,1,96) = 22.38</td>
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<tr>
<td>L3-4 NIEMG</td>
<td>NS</td>
<td>F(8,96) = 15.05</td>
<td>F(1,1,96) = 22.38</td>
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<tr>
<td>Displacement</td>
<td>F(8,96) = 7.19</td>
<td>F(8,96) = 10.87</td>
<td>F(1,1,96) = 8.13</td>
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loading and recovery period. In the recovery period, statistically significant differences exist in the displacements resulted from the two frequencies applied. The pattern of the NIEMG over time is also different in the recovery period. From the experimental data and from the statistical analysis, one can conclude that significant differences in the development and recovery of creep over time as well as in the development of muscle hyperexcitabilities over the recovery period exist due to the increase in frequency of lumbar flexion.

3.4. The model

The graphical representation of the best-fit models developed for the experimental data at 0.1 Hz and 0.5 Hz cyclic loading is superimposed on the data points of Fig. 3, and a summary of the coefficients and the time constants for Eqs. (1)–(4) are given in Tables 2–5.

### Table 2
NIEMG models during cyclic loading

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>Level</th>
<th>NIEMG&lt;sub&gt;ss&lt;/sub&gt;</th>
<th>( T_1 ) (min)</th>
<th>( r^2 )</th>
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<tr>
<td>0.1</td>
<td>L-3/4</td>
<td>0.654</td>
<td>2.7</td>
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<td></td>
<td>L-4/5</td>
<td>0.346</td>
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<td>0.789</td>
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<td>0.5</td>
<td>L-3/4</td>
<td>0.65</td>
<td>8.0</td>
<td>0.801</td>
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<tr>
<td></td>
<td>L-4/5</td>
<td>0.918</td>
<td>1.7</td>
<td>0.967</td>
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<tr>
<td></td>
<td>L-5/6</td>
<td>0.352</td>
<td>4.8</td>
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### Table 3
Vertical displacement model during loading

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<th>Frequency (Hz)</th>
<th>( D_0 ) (mm)</th>
<th>( D_{L1} ) (mm)</th>
<th>( T_2 ) (min)</th>
<th>( D_{L2} ) (mm)</th>
<th>( T_3 ) (min)</th>
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<tr>
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<td>12.0</td>
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<td>0.999</td>
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<td>0.5</td>
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<td>0.43</td>
<td>5.3</td>
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### Table 4
Vertical displacement model during recovery

<table>
<thead>
<tr>
<th>Frequency (Hz)</th>
<th>( D_f ) (mm)</th>
<th>( D_{R1} ) (mm)</th>
<th>( T_4 ) (min)</th>
<th>( D_{R2} ) (mm)</th>
<th>( T_5 ) (min)</th>
<th>( r^2 )</th>
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<tr>
<td>0.1</td>
<td>14.0</td>
<td>1.3</td>
<td>3</td>
<td>1.3</td>
<td>210</td>
<td>0.965</td>
</tr>
<tr>
<td>0.5</td>
<td>11.0</td>
<td>3.1</td>
<td>4</td>
<td>1.6</td>
<td>200</td>
<td>0.985</td>
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### Table 5
NIEMG model during recovery

<table>
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<tr>
<th>Frequency (Hz)</th>
<th>Level</th>
<th>( E )</th>
<th>( T_6 ) (min)</th>
<th>( B )</th>
<th>( T_7 ) (min)</th>
<th>( C )</th>
<th>( T_8 ) (min)</th>
<th>( T_d ) (min)</th>
<th>NIEMG&lt;sub&gt;ss&lt;/sub&gt;</th>
<th>( r^2 )</th>
</tr>
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The major findings of this study revolve around the fact that cyclic loading of the lumbar spine results in a transient neuromuscular disorder of intensity and duration which are dependent on the frequency at which the flexion/extension is performed. Cyclic loading at low frequency is associated with muscular hyperexcitability which is lower in intensity and shorter in duration during the 7 h of rest. Furthermore, lower frequencies result in the development of less creep in the viscoelastic tissues of the lumbar spine and in better recovery of creep after cyclic flexion at 0.5 Hz. Overall, the data obtained in this study provide experimental confirmation of epidemiological data indicating that the frequency of a repetitive motion is a risk factor. The experimental data and the statistical analysis point out that the patterns of displacement and NIEMG over time are significantly different \( p < 0.05 \) for the two cyclic loading frequencies during the recovery period. During the recovery period, the initial hyperexcitability was nearly identical in intensity and duration. However, the delayed hyperexcitability was initiated earlier and lasted significantly longer when flexion at 0.5 Hz was performed. The magnitude of the delayed hyperexcitability was also significantly larger and it lasted significantly longer.

The statistical analysis showed no significant differences in the decrease in EMG during the 20 min of cyclic loading among the two frequencies, probably due to the large variability. The mean NIEMG at the end of the 20 min loading, however, was substantially lower for the loading frequency of 0.5 Hz.

The cyclic loading resulted in the development of creep within the viscoelastic tissues. It is established that creep resulting from cyclic loading causes micro-damage and degenerative structural changes in ligaments (Frank et al., 1985). Microscopic data shows micro-tears and disorganization in the alignment of fibers after cyclic loading. One can, therefore, regard the creep developed as the source of the resulting neuromuscular disorder. It is well established that viscoelastic tissues can reach large strains when the rate of loading is slow. At fast rates of loading, the tissue responds with development of lower strain as well as with a lower failure strain (i.e. damage or rupture), (Panjabi and Courtney, 2001; Newman et al., 1994). The loading of the lumbar spine at 0.1 Hz required 5 s to reach its peak load. In cyclic loading at 0.5 Hz, however, the peak load was developed within 1 s, or five times as fast. Therefore, the substantially faster rate of loading associated with the cyclic flexion at 0.5 Hz would have developed much more micro-damage in the collagen fibers of the various lumbar viscoelastic tissues which explains its different recovery pattern. The increased micro-damage also resulted in a delayed hyperexcitability of a larger magnitude and of a longer duration.

In general, the coefficients and time constants developed for the model confirmed the statistical analysis. The frequency at which the lumbar spine was loaded had a profound impact on the patterns of development and recovery of displacement and shear creep. It should be noted that the time constants associated with the fast viscoelastic components associated mostly with the ligaments and capsules varied significantly with frequency. The slower viscoelastic component, however, is mostly composed of the escape and re-uptake of fluids across the discs membrane. That seemed to be relatively independent of the frequency of loading; it depends mostly on prescribed rates of fluid transported across membranes dictated by membrane properties. Overall, the dependence of viscoelastic tissue strain on the rate of its loading was re-confirmed and seem to be a dominant factor on the development and recovery of creep in the viscoelastic tissues of the spine and probably the source of the resulting disorder.

The time delay associated with the initiation of the delayed hyperexcitability also seemed to be shorter in the model developed for the preparations subjected to loading at 0.5 Hz. Since the viscoelastic tissues were showing substantially more creep at the high loading frequency, it is reasonable to assume that they developed much more tissue micro-damage. That, in turn, results in a more severe acute inflammation (Solomonow et al., 2003a). Inflammation is developed by diffusion of neutrophils from the blood vessels within the tissues and

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therefore is a function of time. Due to the increased severity of the damage, larger numbers of neutrophil infiltrated into the tissue, taking a longer time to reach saturation and staying longer before the problem is resolved. Overall, it seemed that their effect on the delayed hyperexcitability was initiated earlier and lasted longer and that was properly reflected by the time constants $T_d$, $T_s$ and the coefficient amplitude $C$, all of which were larger at 0.5 Hz.

An important finding which was described in a previous report (Solomonow et al., 2001) is that cyclic loading at 0.5 Hz resulted in 27–75% increase in the mean absolute value of the peak EMG when compared to the corresponding value at a frequency of 0.1 Hz. Higher frequency of cyclic flexion/extension, therefore, is also associated with significant increase in muscle forces applied across the intervertebral joints. This phenomena was also assessed from various aspects including modelling and experiments with human subjects (Marras and Granata, 1997; Marras et al., 1984; Marras and Mirka, 1993; Parnianpour et al., 1990). The general results indicate that increasing the velocity of the lumbar movement results in increased muscular forces which are expected to add to the stability of the intervertebral joints in the face of rapidly occurring physical changes. One should consider, however, that stability developed by muscle forces in movements at high velocities are limited by the ability of the muscles to respond with rapid reflexive contraction; that, in turn, will depend on the muscles’ fiber type composition (Mannion, 1999). Fast twitch muscles could develop peak forces 2–3 times faster than slow twitch muscles. Overall, however, the physiology attempts to preserve the stability of the spine with increase in muscle forces to offset the larger moments developed by higher acceleration/deceleration. The cost of such dynamic regulation is the development of large loads over the disc which may be deleterious in the long term.

Several issues relative to assumptions or limitations associated with this study should be discussed. The “S” shaped hook could have inflicted injury during insertion and localized injury during loading. This was ruled out experimentally. First, in pilot studies the hook diameter was increased to 1.5 mm so it will distribute the load to a larger area, reducing local stress on the ligament. In following histological studies, longitudinal sections of the L-4/5 supraspinous ligament were analyzed for neutrophils (Solomonow et al., 2003a). There was no evidence of such localized inflammatory agents to suggest damage localized to the hooks contact surface. Furthermore, data collected from quadrupeds may need to be amended in order to apply results to human subjects. Preliminary results with a large group of human subjects in our Center (Solomonow et al., 2003b) suggest that spasms and changes in neuromuscular responses indeed are recorded in response to cyclic and static flexion. Adjustment for size, direction of gravity and evolutionary functions between the feline and the human will probably modify time constants and magnitudes of the various responses but not the phenomenon itself.

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References


