Short rest between cyclic flexion periods is a risk factor for a lumbar disorder

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Abstract

Background. The epidemiology identifies cyclic lumbar loading as a risk factor for cumulative trauma disorder. Experimental biomechanical and physiological confirmation is lacking. The objective of this study was to asses the impact of different rest durations applied between periods of cyclic loading on the development of an acute lumbar disorder which, if continued to be subjected to loading, may develop into a cumulative disorder.

Methods. Three groups of in vivo feline preparations were subjected to six sequential 10 min loading periods of cyclic lumbar flexion at 40 N with a frequency of 0.25 Hz applied to the L-4/5 level. The rest durations varied from 5 min in the first group, to 10 min in the second and to 20 min in the third. Reflexive EMG from the multifidi and lumbar displacement were used to identify significant ($P < 0.001$) effects of time and rest duration for post-load EMG and displacement. Single-cycle test were performed hourly for 7 h post-loading to assess recovery. A model developed earlier was applied to represent the experimental data.

Findings. The groups allowed 5 and 10 min rest exhibited an acute neuromuscular disorder expressed by a significant ($P < 0.001$) delayed hyperexcitability 2–3 h into the 7 h recovery period with the intensity of the hyperexcitability significantly higher ($P < 0.001$) for the group allowed only 5 min rest. The group allowed 20 min rest had a slow, uneventful recovery, free of delayed hyperexcitability.

Interpretations. Occupational and sports activities requiring repetitive (cyclic) loading of the lumbar spine may be a risk factor for the development of a cumulative lumbar disorder and may require sufficient rest, as much as twice as long as the loading period, for prevention. Comparison to similar data for static lumbar loading shows that cyclic loading is more deleterious than static loading, requiring more rest to offset the negative effect of the repeated acts of stretch.
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Keywords: Spine; Lumbar; EMG; Disorder; Repetitive; Cumulative

1. Introduction

Epidemiologic data identifies repetitive (cyclic) flexion to be a risk factor for cumulative trauma disorder (CTD) in the lumbar spine (Andersson, 1999; Bernard et al., 1997; Manchikanti, 2000; Melhorn, 2003). Up to 42% of the costs associated with worker's compensation claims are associated with low back pain, many of which are diagnosed as idiopathic (Manchikanti, 2000). Identifying risk

factors and mechanisms of injury could help lessen the large financial burden and safety issue that CTD presents in the workplace. In the case of cyclic loading, a cumulative effect is observed by the presence of creep in the viscoelastic tissues caused by microdamage to the collagen fibers present in the tissue (Williams et al., 2000). Because of the cumulative effect, even moderate loads are sufficient to cause an acute neuromuscular disorder. Microdamage to the collagen fibers elicits an acute inflammatory response in an attempt to promote healing (Solomonow et al., 2003b; Solomonow, 2004). Given inadequate rest time and further exposure to load, acute inflammation can

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become chronic and can lead to a debilitating, cumulative neuromuscular disorder (Fransen et al., 2002; Solomonow et al., 2004). The symptoms of CTD include pain, weakness, spasms, and decreased range of motion (Barbe et al., 2003; Bernard et al., 1997; Fryer et al., 2004; Kang et al., 2002; Halbertsma et al., 2001). More insight is needed to understand the mechanisms and risk factors of CTD so as to prevent and treat the disorder more effectively.

Previous work with a feline model using static flexion has identified load magnitude (Sbriccoli et al., 2004a,b), load duration (LaBry et al., 2004), number of repetitions (Sbriccoli et al., 2004a,b), short rest periods (Courville et al., 2005), and work to rest ratios (Sbriccoli et al., 2006) to be risk factors in the development of an acute neuromuscular disorder. This disorder was manifested by EMG spasms and decreased EMG during the loading periods and by initial and delayed hyperexcitability during the rest period. Recent studies with cyclic flexion have examined the effect of load frequency (Lu et al., 2004), load magnitude (Claude et al., 2003), and number of repetitions (Navar et al., 2006) on a feline model. Furthermore, cyclic flexion has been shown to have a more harmful effect than static flexion for similar protocols investigating loading repetitions and load magnitude. Further exploration into the effect of rest durations on cyclic flexion and its relative effect to static flexion is needed and that is the focus of this study.

The objective is to investigate the effect of short rest durations on cyclic loading in order to provide biomechanical and physiological evidence to support and validate previous epidemiological findings. In a previous static loading study examining the effect of rest periods it was found that work to rest ratios of 1:1 or lower prevented the development of an acute neuromuscular disorder (Courville et al., 2005). We hypothesize that short rest durations between cyclic loading periods will cause an acute neuromuscular disorder. Also, we predict that cyclic loading will elicit a more intense neuromuscular disorder than static loading for identical load magnitudes, load durations and rest durations. Specifically, a work to rest ratio of 1:1 with cyclic flexion may not allow enough rest to prevent the development of an acute neuromuscular disorder.

2. Methods

2.1. Preparation

Twenty-one adult cats weighing $3.53 \, \text{kg} \pm 0.34$ were used in this study. Cats were anesthetized intravenously with 60 mg/kg chloralose with the pre-anesthetic intramuscular injection of xylazine, according to a protocol approved by the Institutional Animal Care and Use Committee (IACUC). The skin overlying the lumbar spine was dissected to expose the lumbar fascia, and an S-shaped stainless-steel hook was applied around the supraspinous ligament between L-4 and L-5. The preparation was then positioned in a rigid stainless-steel frame and fixed for subsequent EMG electrodes insertion. All the preparations were subjected to six work/rest periods at a load of 40 N. Preparations were divided into the following three experimental groups according to the length of the rest period allowed between consecutive cyclic work periods: 10 min work and 5 min rest or $6 \times 10{:}5$ ($N = 7$), 10 min work and 10 min rest or $6 \times 10{:}10$ ($N = 7$), and 10 min work followed by 20 min rest or $6 \times 10{:}20$ ($N = 7$).

2.2. Instrumentation

The lumbar spine was isolated by means of two external fixators, which were applied to the L-1 and L-7 posterior process, respectively. 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. Three pairs of fine stainless-steel wire EMG electrodes with an inter-electrode distance of 3–4 mm were inserted in the right L-3/4, L-4/5, L-5/6 multifidus muscles, 8 mm laterally from the posterior spinal processes. A ground electrode was inserted into the gluteus muscle. Each electrode pair constituted the input to a differential EMG amplifier with a 110-db common mode rejection ratio, a gain of up to 200,000 and a band pass filter in the range of 6–500 Hz. The EMG was recorded using a sampling rate of 1000 Hz, and it was also continuously monitored on an oscilloscope. Since the multifidi are slow twitch postural muscles and their power spectra indicates 95% of the power is within the 0–500 Hz range, the upper limit of the bandpass filter was set at 500 Hz. This allowed sampling at a lower rate and significant savings in memory and time in EMG processing. The S-shaped stainless-steel hook inserted around the L-4/5 supraspinous ligament was connected to the crosshead of the 858 Mini Bionix II Material Testing System (MTS, Inc., Minneapolis, MN, USA), in which a load cell was located. The load was applied through the MTS actuator with a computer controlled loading system. The vertical displacement of the actuator was also monitored continuously. The load cell and displacement outputs of the 858 Mini Bionix II MTS were sampled into the computer along with the EMG signals.

2.3. Experimental protocol

The three experimental groups were subjected to the protocol described below. For all of the experimental groups, a pre-load of 1 N was applied just prior to the cyclic loading period. For all groups, the cyclic load consisted of a 0.25 Hz sinusoidal waveform with a moderate peak load of 40 N applied to the lumbar spine via the S-shaped stainless-steel hook. The loading of the L-4/5 supraspinous ligament elicited a moderate lumbar flexion (Williams et al., 2000). This cyclic load was applied for six 10-min intervals with different rest period durations for each experimental group, during which no load was applied. In the

first group, the rest duration between working periods was 5 min (a work to rest ratio of 2:1); in the second group, the rest duration was 10 min (a work to rest ratio of 1:1); in the third group, the rest duration was 20 min (a work to rest ratio of 1:2). The EMG signals from each of the three channels, the load and the vertical displacement were recorded continuously during the six loading periods. A 7-h recovery period followed the 6 loading/rest periods. Nine 4-s single-cycle loading tests of 0.25 Hz were performed during the 7 h of recovery. Tests were applied after 10, 30, and 60 min of rest, and every hour thereafter, recording EMG, load and displacement during every test.

2.4. Data analysis

Four-second windows of EMG, cyclic load applied to the spine, and vertical displacement of the L-4/5 supraspinous ligament were sampled at the beginning of the loading period and every 20 s thereafter for each 10-min cyclic loading period, as well as for each test in the recovery period. EMG data was full-wave rectified and integrated over the 4-s window and normalized with respect to the value obtained from the first window of the first 10-min load period. The normalized integrated EMG (NIEMG) of all the preparations subjected to the same rest period duration were pooled, and the mean and standard deviation was calculated and plotted on a NIEMG versus time plot for each of the three groups. Displacements of the respective window of all preparations subjected to the same rest period duration were pooled, and the mean ($\pm$SD) was calculated and plotted versus time.

2.5. Model

The model is based on our previous work where a continuous 20-min loading period was followed by a 7-h recovery period (Solomonow et al., 2003a,b,c). The equations were then modified to describe a series of work periods spaced by rest periods, introducing new time component variables, T_W and T_R, representing the work period time and rest period time, respectively (Courville et al., 2005). The rest period time, T_R, was 5 min, 10 min, and 20 min for the three experimental groups, respectively. Eq. (1) describes the NIEMG behavior during each work period, and is shown below:

$$\text{NIEMG}(t) = A_n e^{-\frac{[t-n(T_W+T_R)]}{T_{n1}}} \Big|_{n(T_W+T_R)}^{(n+1)T_W+nT_R} + \text{NIEMG}_{0n} \quad (1)$$

It was assumed that A and NIEMG_0 are not constant throughout the work–rest session i.e., A and NIEMG_0 are changing from one work period to the next. It was also assumed that T_1 might not be the same between subsequent work periods. During the rest periods, the equation is modified as follows, assuming that the amplitudes of NIEMG_0, B, and T_2 will vary from one rest period to the next, shown in Eq. (2) below:

$$\text{NIEMG}(t) = (t - [(n+1)T_W + nT_R])B_n e^{-\frac{\{t-[(n+1)T_W+nT_R]\}}{T_{n2}}} \Big|_{(n+1)T_W+nT_R}^{(n+1)(T_W+T_R)}$$
$$+ \text{NIEMG}_{0n} \quad (2)$$

The NIEMG during the 7-h recovery after the loading period was modeled by Eq. (3):

$$\text{NIEMG}(t) = E(1 - e^{-\frac{t}{T_4}}) + tBe^{-\frac{t}{T_5}} + C(t - T_d)e^{-\frac{(t-T_d)}{T_6}} + \text{NIEMG}_0 \quad (3)$$

The equation describing the development of displacement, an indirect measurement of creep in the viscoelastic tissues, during a series of work periods spaced by rest periods is shown below in Eq. (4):

$$\text{Disp}(t) = \left[D_{0n} + D_{Ln}\left(1 - e^{-\frac{[t-n(T_W+T_R)]}{T_{n5}}} \right) \right] \Big|_{n(T_W+T_R)}^{(n+1)T_W+nT_R} \quad (4)$$

where $\text{Disp}(t)$ is the displacement as a function of time, D_0 is the elastic component of amplitude, D_L is the viscoelastic component of amplitude, and T_5 is the time constant governing the development of creep during flexion. It is also assumed that D_0, D_L, and R change from one work/rest session to the next.

The recovery of the displacement during rest periods is described below in Eq. (5). It is assumed that T_5 and T_6 vary from one period to the next:

$$\text{Disp}(t) = \left[D_{0n} + R_n + (D_{Ln} - R_n)e^{-\frac{t-[(n+1)T_W+nT_R]}{T_{n6}}} \right] \Big|_{(n+1)T_W+nT_R}^{(n+1)(T_W+T_R)} \quad (5)$$

The displacement during the 7-h recovery period was modeled by Eq. (6):

$$\text{Disp}(t) = D_c + D_R e^{-\frac{t}{T_3}} \quad (6)$$

Once the mean $\pm$ SD of the experimental data were calculated, attempts were made to generate the best-fit models described above using the Marquardt–Levenberg non-linear regression algorithm. In some cases, the algorithm failed to converge satisfactorily; in these cases, initial or final values were determined by sequential recursive iteration, optimizing for regression coefficient.

A two-way analysis of variance (ANOVA) was used to assess the effect of time and rest duration on the recovery period of the NIEMG and displacement. For all three experimental groups (5-min, 10-min, and 20-min rest), ANOVA was performed on all three lumbar levels recorded (L-3/4, L-4/5, L-5/6). The level of significance was set to $P < 0.05$ to determine significant differences.

3. Results

Fig. 1 depicts a typical response of one preparation subjected to six 10-min working periods separated by 5-min rest periods. The top three traces depict the raw EMG
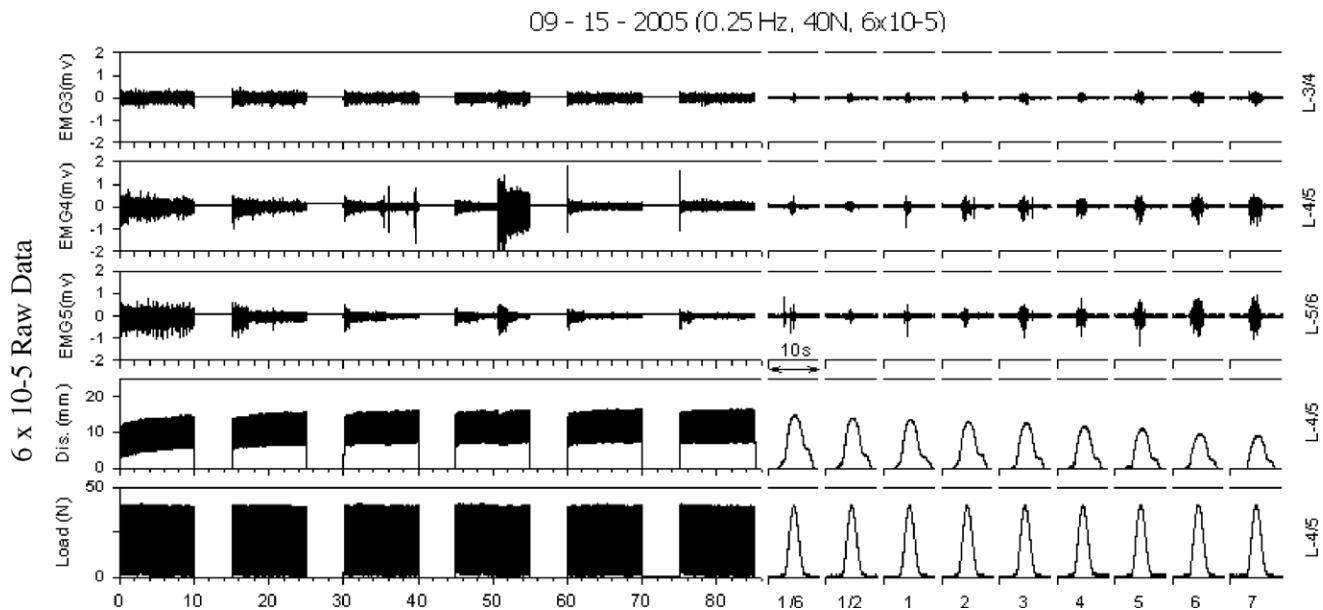
Fig. 1a. Typical recording of one preparation subjected to 5 min of rest between six 10 min loading periods followed by 7 h of recovery. The top three traces show the EMG recorded from the L-3/4, L-4/5, and L-5/6 lumbar levels and the bottom two traces show the displacement and load, respectively. Note the presence of a particularly large spasm in the 4th loading period across the L-4/5 and L-5/6 lumbar levels and the corresponding sharp decrease in displacement in the trace below.

signal from electrodes placed between L-3/4, L-4/5, L-5/6, respectively, the fourth trace shows the displacement and the bottom trace the applied load. The EMG amplitude decreases over time during the working periods, and random spasms are present throughout. Creep is evident in the displacement traces and accumulates over time, as the ligament length only partially recovers after each loading cycle.

The mean (±SD) data for NIEMG and displacement from the pooled data of all preparations for the 5-min, 10-min and 20-min rest groups are shown in Figs. 2–4, respectively. In each figure, the three upper traces show the mean NIEMG data and the bottom trace shows displacement.

In the group subjected to 5-min rest in between working periods ($6 \times 10{:}5$ protocol shown in Fig. 2) the mean initial displacement in the first 10-min working period was 8.72 mm. At the end of the first 10-min working period, the displacement was 12.77 mm, resulting in a mean creep of 46.44%. At the end of the sixth 10-min working period, the mean displacement was 15.09 mm, resulting in a mean cumulative creep of 72.50%. During the 7 h recovery period, the displacement decreased, but did not return to the original length. The final mean displacement was 10.13 mm, resulting in a mean residual creep of 16.05%.

In the group subjected to 10-min rest in between working periods ($6 \times 10{:}10$ protocol, shown in Fig. 3) the mean initial displacement in the first 10-min working period was 7.28 mm. At the end of the first 10-min working period, the displacement was 11.99 mm, resulting in a mean creep of 64.63%. At the end of the sixth 10-min working period, the mean displacement was 15.73 mm, resulting
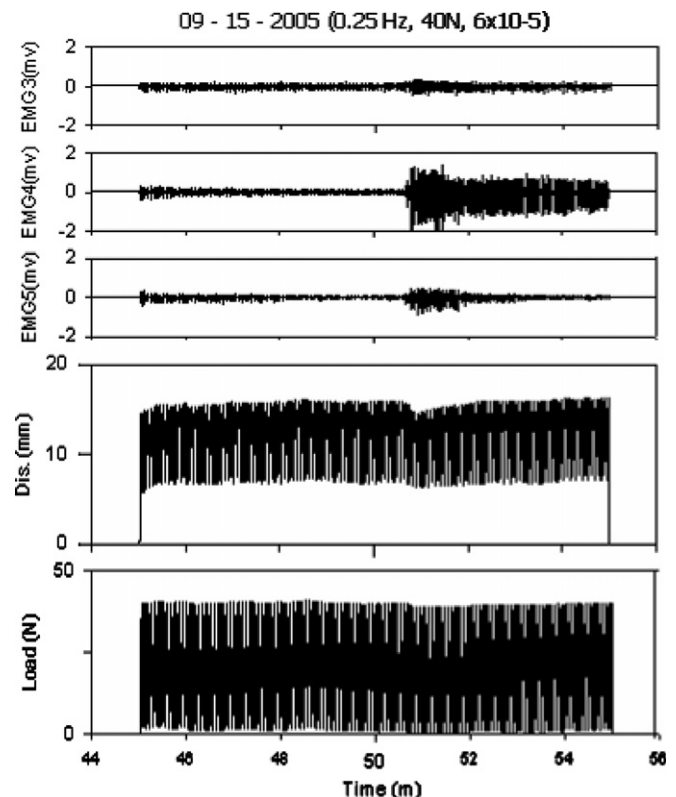

Fig. 1b. Enlarged view of the significant spasm in the fourth loading period of the same preparation subjected to 5 min of rest between six 10 min loading periods followed by 7 h of recovery. Notice that the displacement decreases at the same time that the EMG spasm is recorded.

in a mean cumulative creep of 115.96%. During the 7 h recovery period, the displacement decreased, but did not
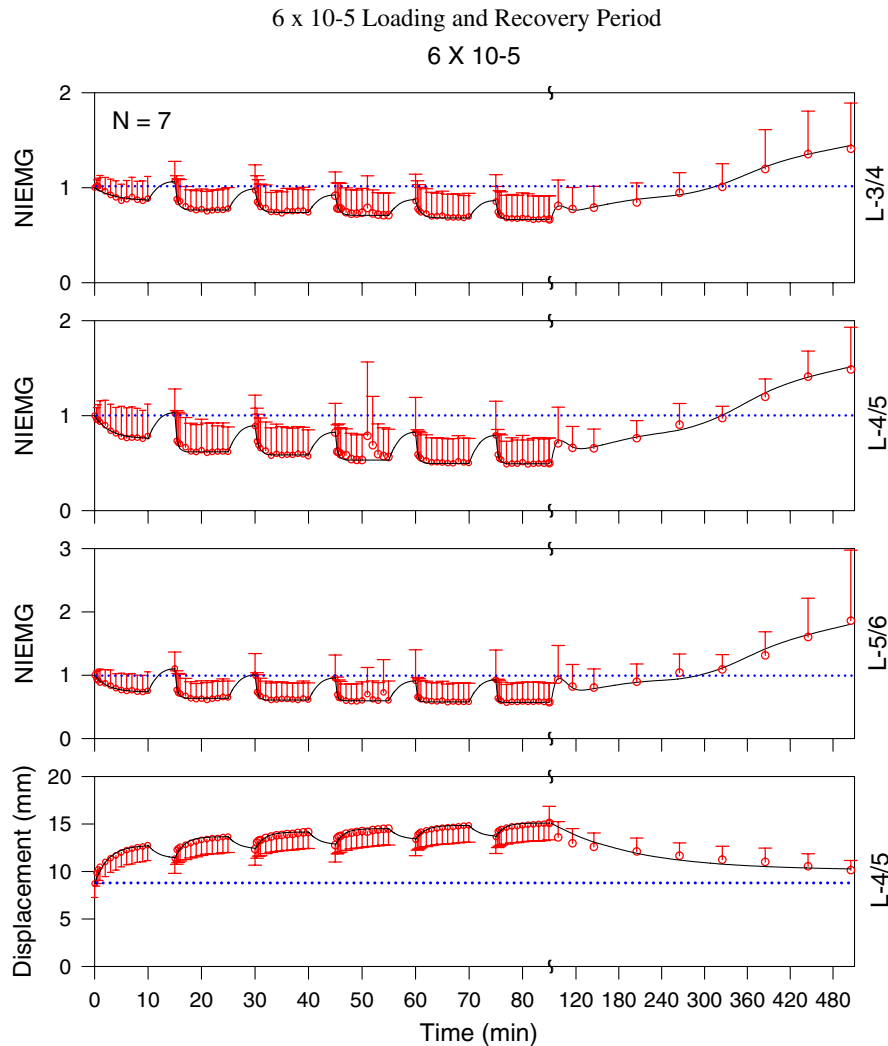
6 x 10-5 Loading and Recovery Period

Fig. 2. The mean (±SD) NIEMG of the L-3/4, L-4/5, and L-5/6 lumbar levels and displacement are shown for the group subjected to 5 min rest between loading periods. The solid lines through data points represent models generated to describe mean NIEMG and displacement during the loading and recovery periods. Note the presence of spasms in the fourth loading period lead to a poor fit for the model in that period. Also note the presence of EMG hyperexcitability in the recovery period.

return to the original length. The final mean displacement was 9.59 mm, resulting in a mean residual creep of 31.75%.

In the group subjected to 20-min rest in between working periods (6 × 10:20 protocol, shown in Fig. 4) the mean initial displacement in the first 10-min working period was 8.90 mm. At the end of the first 10-min working period, the displacement was 12.89 mm, resulting in a mean creep of 44.94%. At the end of the sixth 10-min working period, the mean displacement was 15.72 mm, resulting in a mean cumulative creep of 76.69%. During the 7 h recovery period, the displacement decreased, but did not return to the original length. The final mean displacement was 10.43 mm, resulting in a mean residual creep of 17.25%.

For the 6 × 10:5 group (shown in Fig. 2), the mean NIEMG decreased to 0.86 for the L-3/4 level, 0.76 for the L-4/5 level, and 0.73 for the L-5/6 level after the first 10-min working period. After the first 5-min rest period, the mean NIEMG recovered to exceed the initial values,

recording mean values of 1.07, 1.02, and 1.10 for the L-3/4, L-4/5, and L-5/6 levels, respectively. During the subsequent working periods, the mean NIEMG failed to completely recover, resulting in a net decrease after the duration of the last working period. The mean NIEMG values at the end of the six working periods were 0.66, 0.49, and 0.57 for the different lumbar levels.

During the first 10 min of recovery, there was a sharp increase in the mean NIEMG, referred to as initial hyperexcitability, to 0.81, 0.71, and 0.93 for the L-3/4, L-4/5, and L-5/6 lumbar levels, respectively. The mean NIEMG then decreased slightly between 10 min and 20 min rest, followed by a gradual increase in NIEMG over the remainder of the 7 h rest period. After 3 h recovery, the NIEMG values surpassed or almost reached to original pre-load values of 1.0, with recordings of 0.94, 0.90, and 1.04 for each lumbar level in descending order. The mean NIEMG values continued to increase, surpassing the pre-load values with 1.41, 1.48, and 1.86 for the three different lumbar
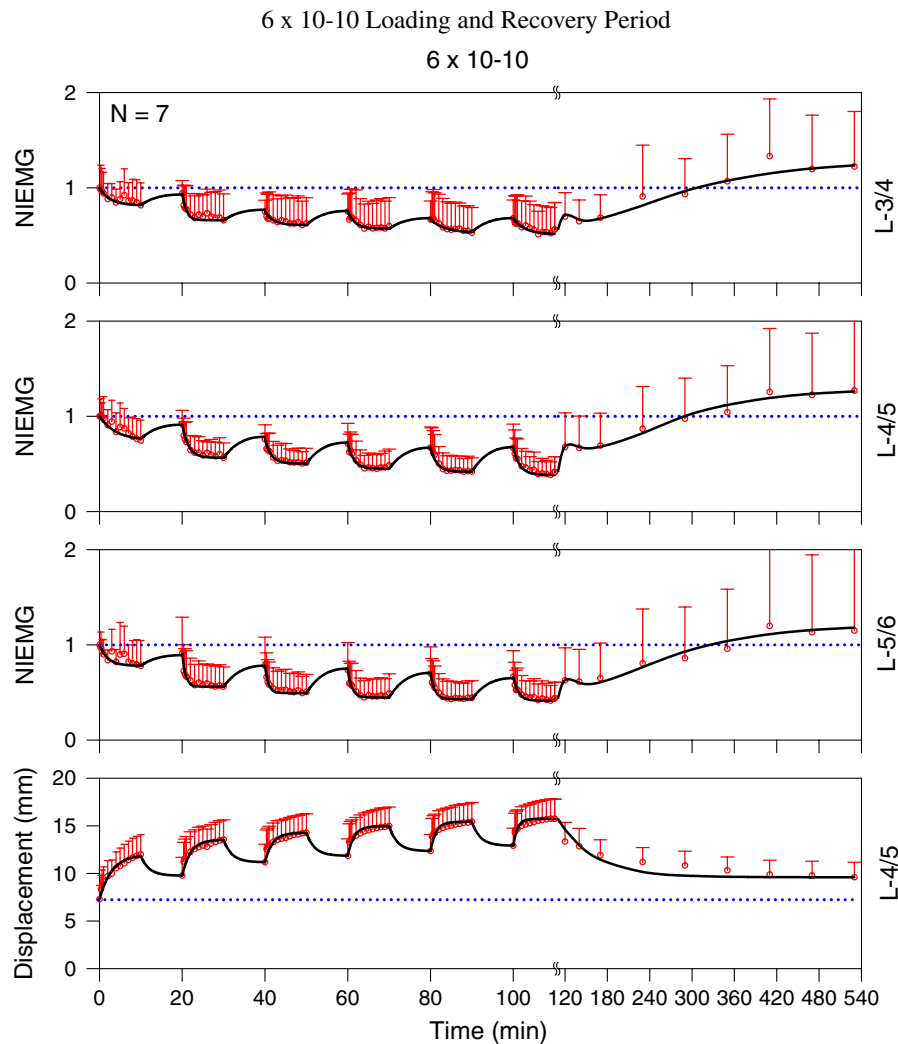
6 x 10-10 Loading and Recovery Period

Fig. 3. The mean (±SD) NIEMG of the L-3/4, L-4/5, and L-5/6 lumbar levels and displacement are shown for the group subjected to 10 min rest between loading periods. The solid lines through data points represent models generated to describe mean NIEMG and displacement during the loading and recovery periods. Note the presence of EMG hyperexcitability in the recovery period.

levels at the end of the 7 h recovery period. This phase is defined as delayed hyperexcitability.

For the $6 \times 10{:}10$ group (shown in Fig. 3), the mean NIEMG decreased to 0.82 for the L-3/4 level, 0.74 for the L-4/5 level, and 0.78 for the L-5/6 level at the end of the first 10-min working period. After the first 10-min rest period, the mean NIEMG partially recovered, recording mean values of 0.94, 0.92, and 0.91 for the L-3/4, L-4/5, and L-5/6 levels, respectively. During the subsequent working periods, the NIEMG further failed to completely recover, resulting in a net decrease after the last working period. The mean NIEMG values at the end of the six working periods were 0.56, 0.41, and 0.44 for the different lumbar levels.

During the first 10 min of recovery, there was an increase in the mean NIEMG to 0.70, 0.68, and 0.63 for the L-3/4, L-4/5, and L-5/6 lumbar levels, respectively. The NIEMG levels then decreased slightly between 10 min and 20 min rest, followed by a gradual increase in NIEMG over the remainder of the 7 h rest period. After 3 h recovery, the NIEMG values increased almost to the

original pre-load values of 1.0, with recordings of 0.93, 0.97, and 0.86 for each lumbar level. The mean NIEMG values continued to increase, surpassing with final values of 1.22, 1.27, and 1.15 for the three different lumbar levels. This experimental group, therefore, also displayed delayed hyperexcitability.

For the $6 \times 10{:}20$ group (shown in Fig. 4), the mean NIEMG decreased to 0.82 for the L-3/4 level, 0.69 for the L-4/5 level, and 0.71 for the L-5/6 level at the end of the first 10-min working period. After the first 20-min rest period, the NIEMG partially recovered, recording mean values of 0.83, 0.75, and 0.79 for the L-3/4, L-4/5, and L-5/6 levels, respectively. During the subsequent working periods, the NIEMG further failed to completely recover, resulting in a net decrease after the last working period. The mean NIEMG values at the end of the six working periods were 0.50, 0.36, and 0.50 for the three lumbar levels.

During the first 10 min of recovery, there was an increase in the mean NIEMG to 0.60, 0.51, and 0.64 for the L-3/4, L-4/5, and L-5/6 lumbar levels, respectively.
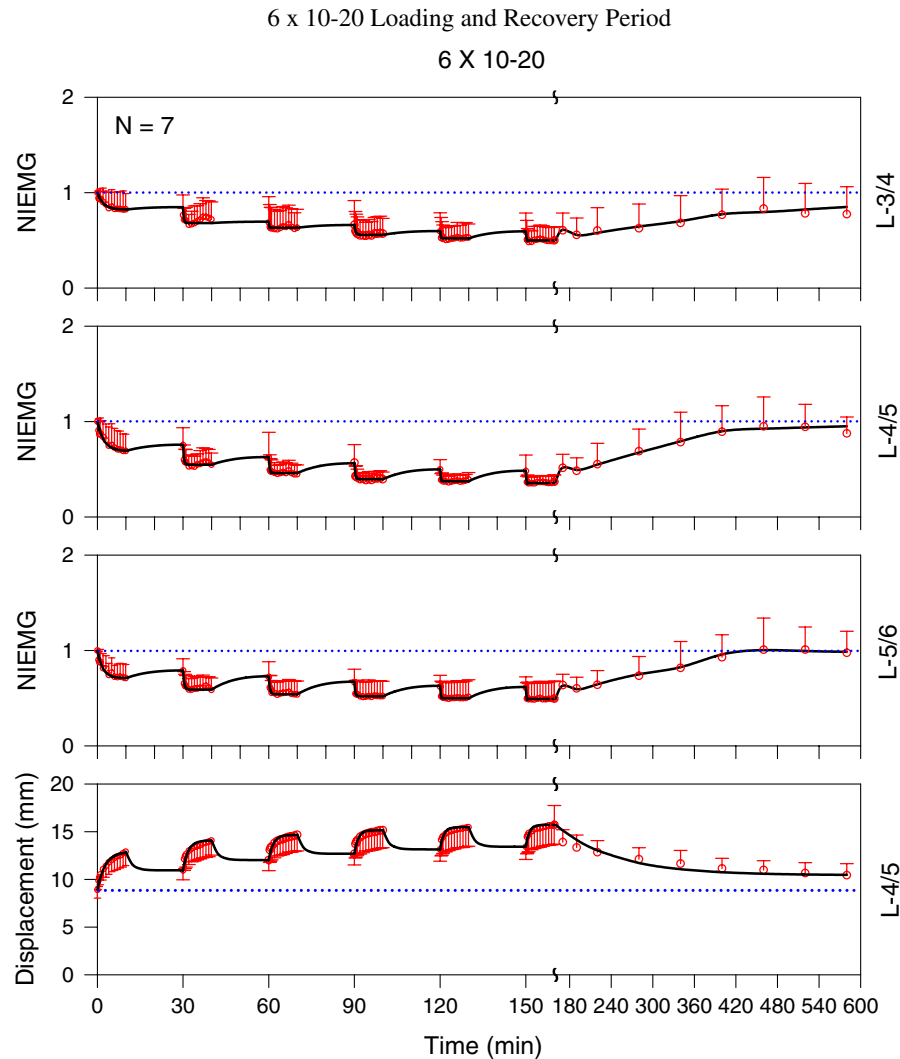
6 x 10-20 Loading and Recovery Period

Fig. 4. The mean (±SD) NIEMG of the L-3/4, L-4/5, and L-5/6 lumbar levels and displacement are shown for the group subjected to 20 min rest between loading periods. The solid lines through data points represent models generated to describe mean NIEMG and displacement during the loading and recovery periods. Note that there is no hyperexcitability present in the recovery period.

The NIEMG levels then decreased slightly between 10 min and 20 min rest, followed by a gradual increase in NIEMG over the remainder of the 7 h rest period. After 5 h recovery, the NIEMG values reached nearly their maximum values, with values less than or equal to the original pre-load values of 1.0 in two of the three lumbar levels recorded, with readings of 0.83, 0.95, and 1.01 for the three lumbar levels. The mean NIEMG values decreased slightly over the remaining 2 h of recovery, with values of 0.77, 0.88, and 0.98 for the for the L-3/4, L-4/5, and L-5/6 levels, respectively. No delayed hyperexcitability was present in this group.

The best-fit models for the NIEMG and displacement data are superimposed on the data in Figs. 2–4. The fit of the model for each loading period and for the recovery periods was usually above an r^2 value of 0.9. However, in some cases the fit was less than an r^2 value of 0.9 due to random, unpredictable EMG spasms in some of the loading periods that are seen in the NIEMG.

In the model describing the rest period, there were some key differences in the model parameters for each experimental group. The constant B, describing the magnitude of the initial hyperexcitability, decreased for the groups with increased rest periods. The B values for the $6 \times 10:5$ group were 0.045 for L-3/4, 0.055 for L-4/5 and 0.100 for L-5/6. In the $6 \times 10:10$ group, B dropped to 0.04, 0.05, and 0.04 for the L-3/4, L-4/5, and L-5/6, respectively. Lastly, the B values for the $6 \times 10:20$ group were 0.04, 0.04, and 0.045 for the three lumbar levels. Larger B values for the $6 \times 10:5$ protocol indicate a more pronounced initial hyperexcitability. Similarly, the T_4 time constant, describing delayed hyperexcitability, increased drastically with shorter rest periods. T_4 in the group subjected to 5-min rest was 450 min, 390 min, and 490 min for the L-3/4, L-4/5, and L-5/6 lumbar levels, respectively. In the 10-min rest group, T_4 was 400 min, 380 min, 320 min for the three lumbar levels. In the 20-min rest group, the value for the T_4 constant decreased drastically to 30 min for L-3/4, 60 min for L-4/5, and 60 min for

L-5/6. This drastic decrease further indicates a distinct difference in the physiologic response between the 6×10:20 group and the 6×10:10, 6×10:5 groups.

The results of two-way ANOVA revealed that there was a significant effect of time ($P < 0.001$) and rest duration ($P < 0.001$) for the NIEMG of the L-3/4, L-4/5 and L-5/6 lumbar levels. Also, there was a significant effect of time ($P < 0.001$) and rest duration ($P = 0.003$) for displacement. No interaction between time and rest duration was found for the NIEMG of all lumbar levels and displacement. Furthermore, when comparing the post-loading period of each experimental group, setting the significance

level to 0.05, it was found that there was no significant difference between the 5-min and 10-min rest duration groups for the L-3/4, L-4/5 levels, but that there was a significant difference between the groups subjected to 20-min rest and the other two groups. For the L-5/6 level, the 6×10:5 group was statistically significant from both the 6×10:10 and 6×10:20 groups. However, the 10-min and 20-min rest groups were not statistically significant from each other at the L-5/6 lumbar level. By visual inspection of the recovery period for the three experimental groups (see Fig. 5), there is a trend towards a difference in the physiologic response of each of the three experimental groups.
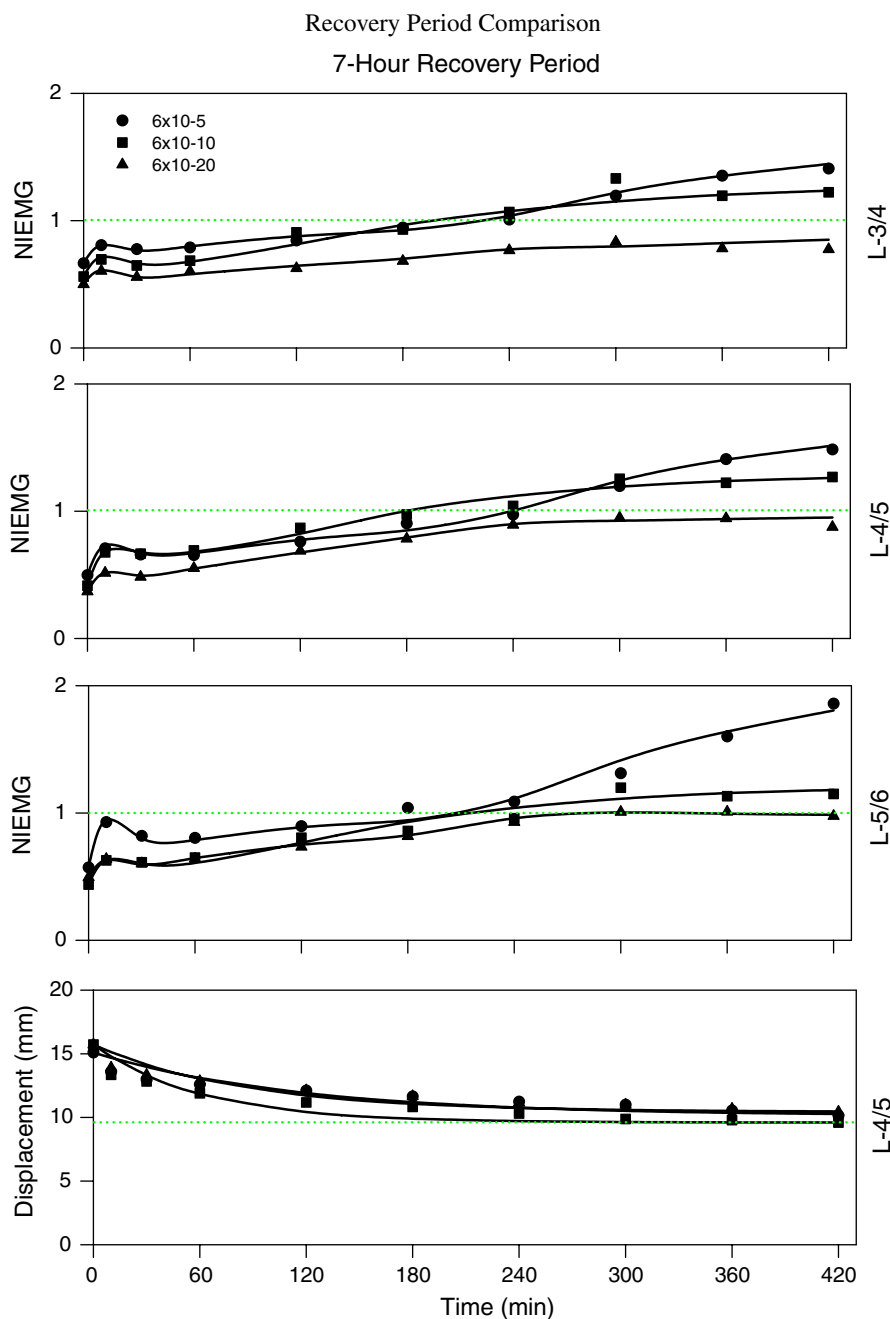

Fig. 5. The models developed for the 6×10:5, 6×10:10, and 6×10:20 groups are superimposed on the mean NIEMG data for the 7 h recovery period. The mean NIEMG only exceeds unity in the groups subjected to 5 min and 10 min rest.

4. Discussion

The major outcomes of this study show that short rest between periods of cyclic loading elicits an acute neuromuscular disorder. While a neuromuscular disorder is not present in the group subjected to 20-min rest, an acute neuromuscular disorder is present in the groups subjected to 10-min rest and 5-min rest. Furthermore, the intensity of the acute neuromuscular disorder increased with shorter rest periods. Caused by the accumulation of creep and microdamage to the soft tissues in the lumbar spine, this disorder is manifested in the results by the decreased EMG over the loading periods, random spasms of the multifidus muscle, and by the initial and primarily by the delayed hyperexcitability of the muscle over the course of the 7 h recovery period.

During the loading period, the EMG drastically decreases in the first few loading cycles, then levels off for the remaining cycles in all three experimental groups. Also, there are random spasms seen throughout the loading cycles in all three experimental groups. In the 4th loading period of Fig. 1a (enlarged in Fig. 1b), a large spasm can be seen across all three lumbar levels, and was particularly large in the L-4/5 lumbar level. These spasms provide evidence of damage to the viscoelastic tissues of the dorsal lumbar region and act to stiffen the joint, attempting to prevent further injury (Fryer et al., 2004; Jackson et al., 2001; Kang et al., 2002; van Dieën et al., 2003; Williams et al., 2000). The effect of the spasm seen in Fig. 1b is reflected in the sharp decrease in the corresponding displacement plot and provides experimental evidence of the relationship between control of creep in the soft tissue by EMG and spasms in the surrounding muscles.

The source of the decrease in EMG during the loading periods is traced to the developed laxity in the ligaments. As the ligaments develop creep, their baseline tension decreases and their length–tension curve display a drastic shift (Solomonow, 2004). The mechanoreceptors within the ligaments remain silent (e.g. sub-threshold) for a longer period during the rising stretch cycle without reflexively activating the muscle (Stubbs et al., 1998; Solomonow et al., 1998) As the laxity of the ligament increases with the developing creep, the muscles are triggered later in the stretch cycle and the peak EMG decreases as well, reflecting less active motor units. With rest, some of the creep is recovered and the mechanoreceptors trigger muscle activity earlier and at more pronounced amplitude, reflecting participation of more motor units. The effect of rest, therefore, is expressed by partial recovery of the EMG towards its original amplitude and triggered timing.

Considering the experimental conditions employed, the complete lumbar spine was exposed to the load which in turn elicited lumbar flexion. As such, the displacement recorded reflects the changes in the total stiffness of the intervertebral joints. The stiffness of the joints, in turn, was the sum of the stiffness of the ligaments, discs, facet capsules and muscles. In this context, the ligaments, discs and facet capsules are passive tissues which display increased creep with load, loading duration, repetitions, etc. and recovery of creep with rest. The muscles, however, are active tissues and have multiple sources of activation. Based on our previous work, we know that one such source is reflexive activation from mechanoreceptors in the ligaments (Stubbs et al., 1998) while a second source is microdamage in the ligaments which elicit spasms (Williams et al., 2000). A third source of muscular activation is the development of an acute inflammation in the ligaments which gives rise to the delayed hyperexcitability (Solomonow et al., 2003a,b,c). Muscular activation level therefore does not necessarily coincide with increasing or decreasing displacement/creep during the various phases of the experiment.

For example, the cumulative creep at the end of the six loading periods was 72.5% for the group allowed 5 min rest. In this group, the short rest elicited more frequent spasms of higher amplitude, increasing the stiffness of the lumbar spine and minimizing creep. The group subjected to 20 min rest between loading periods did not display spasms as frequently and their amplitude was lower. However, the rest period was four times longer than the first group, allowing larger amounts of the creep to recover, yielding a cumulative creep of 76.7%, which is very similar to the first group. The mechanism leading to similar creep values were, however, vastly different; frequent and strong spasms leading to high stiffness in the first and long rest leading to substantial creep recovery in the second.

The group subjected to 10 min rest displayed the largest cumulative creep at the end of the six loading periods. In this group, the rest period allowed only moderate creep recovery while the spasms were not very powerful or frequent. The net effect was that neither of the two factors (spasms or creep recovery) were dominant or effective, leading to a cumulative creep of 116.0%. In this case, the joint stiffness was not dominated by muscular forces or passive tissue tension.

Similarly, after 7 h of rest, insight may be gained into the relationship between creep and muscle activity by comparing the hyperexcitability of the multifidus muscle at the end of the rest period. After 7 h of recovery, the mean residual creep was 16.05% for the 5-min rest group, 31.75% for the 10-min rest group and 17.25% for the 20-min rest group. Consistent with the results for accumulated creep, the highest residual creep was seen in the 10-min rest group, followed by the 20-min rest group, with the 5-min rest group experiencing the least amount of residual creep. After 7 h of rest, and intense delayed hyperexcitability in the multifidus muscle was seen in the group subjected 5-min rest. This increase in muscle activity during the recovery phase would increase joint stiffness and minimize observation of creep in the viscoelastic tissues. In the group allowed 20 min rest, the residual creep was similar to the first group, but mostly due to the long rest periods, low cumulative creep and recovery. In the group allowed 10 min rest, however, a mild hyperexcitability was present

but was not strong enough to produce substantial stiffness increase, resulting in large residual creep. Overall, a direct, quantifiable relationship between the creep in the viscoelastic tissues and the magnitude of the acute neuromuscular disorder that develops in response to loading is difficult to develop as it depends on the stiffness of passive and active tissues which, in turn, depend on a multitude of factors such as load, rest, repetitions and, most of all, motor control, tissue damage and inflammation.

The first 10 min of the recovery period shows a sharp increase in the EMG recorded for all experimental groups. This is known as initial hyperexcitability and is a result of the physiological response, signaled by fast-acting mechanoreceptors in the supraspinous ligament and surrounding viscoelastic tissues of the joint, to the accumulated creep and microdamage of the viscoelastic tissues in the lumbar spine (McLain and Pickar, 1998; Petrie et al., 1998; Sekine et al., 2001; Solomonow, 2004; van Dieën et al., 2003). The initial hyperexcitability was increased in the experimental groups subjected to shorter rest periods, with average values for B, the model parameter describing the initial hyperexcitability, equal to 0.067, 0.043, and 0.042 for the $6 \times 10{:}5$, $6 \times 10{:}10$, and $6 \times 10{:}20$ groups, respectively. Note that here was a significant difference in the average B values between the group subjected to 5-min rest and the two other experimental groups, indicating that there may be a difference in the responses between groups as early as 10 min into the recovery period.

After the initial hyperexcitability peak the NIEMG slowly increases over the next few hours of the recovery period for all experimental groups. However, after 3–5 h into the recovery the NIEMG values rose above the initial pre-load values in both the $6 \times 10{:}5$ and $6 \times 10{:}10$ groups. For the $6 \times 10{:}20$ group, the NIEMG increased to just above the pre-load value for the L-5/6 lumbar level after 5 h of recovery, but then slightly decreased to below the pre-load value after 7 h of recovery. The NIEMG did not reach the pre-load value for either of the other two lumbar levels tested. For the $6 \times 10{:}10$ group, the NIEMG values exceeded unity after 3–4 h of recovery in all three lumbar levels, reaching an average of 1.21 at the end of the 7 h recovery period, indicating the presence of delayed hyperexcitability associated with acute inflammation of the ligamentous tissue (Solomonow, 2004; Solomonow et al., 2003a,b,c). In the $6 \times 10{:}5$ group the onset of delayed hyperexcitability occurred between 3 and 5 h into the recovery period. The NIEMG values began to increase sharply after this time, as seen in Fig. 5, and, by inspection, the characteristic curve of the recovery NIEMG is much different than the other two experimental groups, indicating the development of a more severe, prolonged acute neuromuscular disorder. NIEMG values for the $6 \times 10{:}5$ group reached an average of 1.58 after 7 h of recovery. The presence of a more severe neuromuscular disorder is further evidenced by the increase in time constant, T_4 in the recovery model. Longer T_4 values indicate more pronounced and prolonged changes in the neuromuscular activity. In

the $6 \times 10{:}20$ group the average T_4 value was 50 min. In the $6 \times 10{:}10$ experimental group the average T_4 value was 367 min. For the $6 \times 10{:}5$ group, the average T_4 value was 443 min. Note that the T_4 values increased drastically between the group subjected to 20-min rest, and the other two experimental groups. Furthermore, increases in the T_4 time constant from the 10-min rest group to the 5-min rest group suggest that 5 min of rest between loading periods produces a more pronounced and long lasting hyperexcitability period than 10 min of rest.

The results of this study show that a rest period duration of 5-min between cyclic loading periods of moderate load (40 N) cause an acute neuromuscular disorder that can lead to the development of CTD. Furthermore, rest durations of 10-min may also cause a sufficient damage to lead to the development of CTD. These results confirm previous studies with static loading periods that find short rest periods to be a risk factor for CTD (Courville et al., 2005; LaBry et al., 2004; Sbriccoli et al., 2006). These results build upon previous studies working with static loading have determined load duration (LaBry et al., 2004), number of loading periods (Sbriccoli et al., 2004a,b), work to rest ratios (Courville et al., 2005), and load magnitude (Sbriccoli et al., 2004a,b) to be risk factors for CTD. Also, previous work with cyclic loading has identified higher frequency (Lu et al., 2004) and excessive repetitions (Navar et al., 2006) as risk factors. The effects of load duration with similar work to rest ratios dealing with cyclic flexion are still unknown, and will need to be studied further to help determine safe work/rest schedules in an occupational environment.

Interestingly, this study provides evidence that cyclic loading may cause more damage than static loading (Courville et al., 2005; LaBry et al., 2004; Sbriccoli et al., 2006) for the same load magnitude, duration, and rest periods. Comparing static and cyclic loading of the same work and rest durations, as shown in Fig. 6, with experimental groups subjected to six 10-min loading periods spaced by 10-min rest periods, cyclic loading cases when subjected to the same peak load magnitude, duration of application, andin-between rest periods as in static loading, elicits a more severe neuromuscular disorder. Two-way ANOVA reveals that the difference in the two groups is statistically significant across all three lumbar levels ($P < 0.001$). Unlike the static case, where a 1:1 work to rest ratio (up to loading periods of 30-min) was sufficient to prevent an acute neuromuscular disorder, the same unity work to rest ratio with only 10-min loading periods causes a severe and pronounced disorder. These results show that cyclic loading (e.g. multiple stretch and release) is more damaging than static loading (e.g. stretch and hold) for identical load magnitudes, durations of application, indicating that the repeated acts of stretching of the viscoelastic tissues may cause more damage than the length of time in which the tissues are stretched (Navar et al., 2006). This would be consistent with identifying more repetitions and higher repetition frequencies to be risk factors in developing an
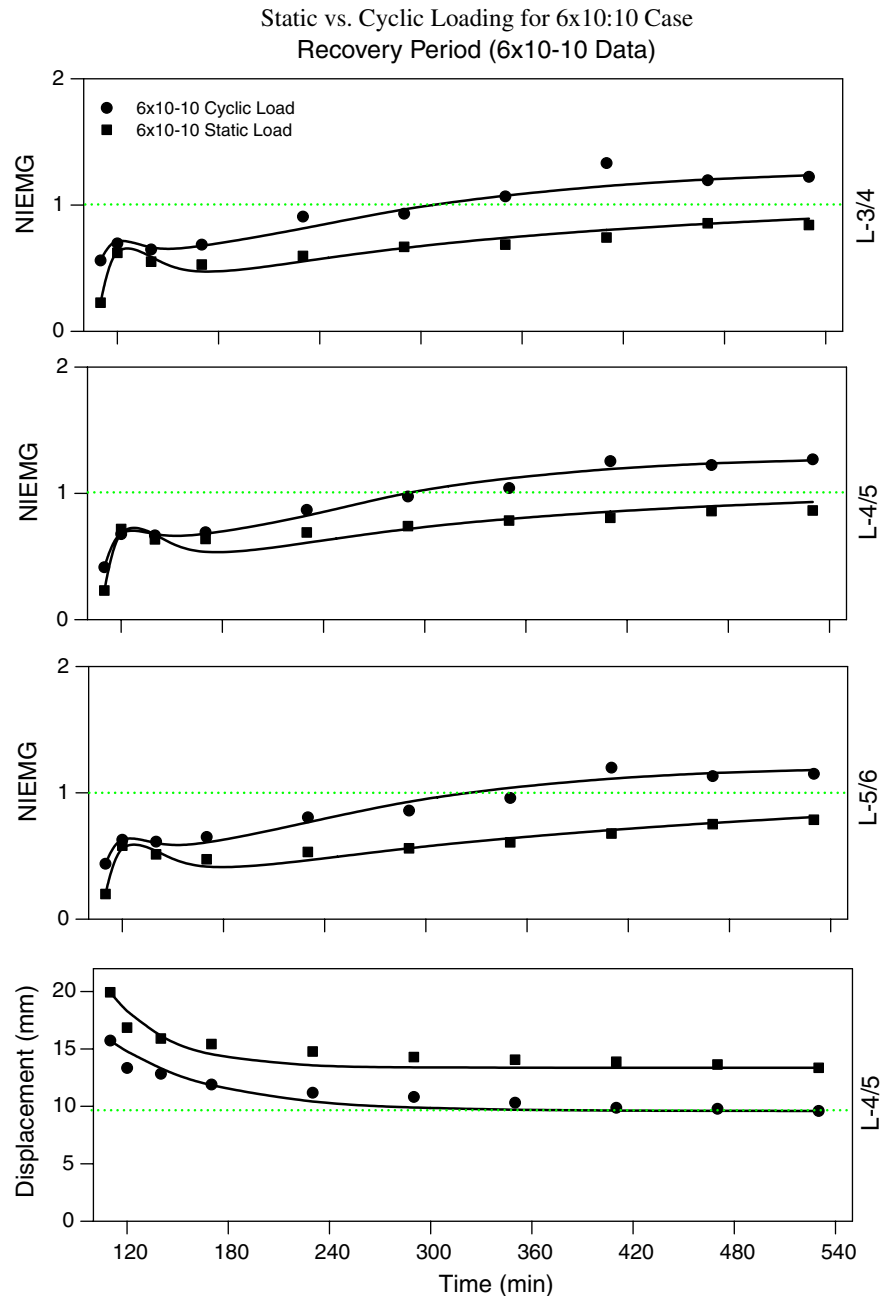
Fig. 6. The mean NIEMG data and the superimposed models during the recovery period are shown for the $6 \times 10{:}10$ groups with static and cyclic flexion. The static and cyclic groups are statistically different from each other ($P < 0.001$) across all three lumbar levels. Data from static experiment with same loading and rest periods was used from a previous experiment (Courville et al., 2005).

acute neuromuscular disorder, as both would increase the number of times the tissue is subjected to the act of stretching over the duration of the total loading period. Further support for this observation are found in Le et al. (2007), who have shown that cyclic loading at moderate loads elicits an acute neuromuscular disorder, compared to static loading where the disorder is present only at high loads.

The results from this study and previous feline studies cannot be directly extrapolated to humans without considering the anatomic and physiologic differences between the two species. Therefore, it is necessary to confirm the results

found in the feline model. It has been established that repetitive bending (cyclic flexion) causes creep and laxity in the viscoelastic tissues of the lumbar region in humans (Chow et al., 2004; Dolan and Adams, 1998; Little and Khalsa, 2005; Parkinson et al., 2004) and the onset of the flexion–relaxation phenomenon (Dickey et al., 2003; Olson et al., 2004) in which decreased muscle activity can lead to further risk of injury and destabilization of the joint. In previous human studies, where the lumbar or knee viscoelastic tissues are subjected to static (Solomonow et al., 2003a,b,c) or cyclic (Olson et al., 2004) flexion, a similar neuromuscular disorder develops. When human subjects

were asked to perform 10 min of cyclic lumbar flexion at 4 s intervals, the development of a neuromuscular disorder was evidenced by the presence of paraspinal muscle spasms and the enhancement of the flexion–relaxation phenomenon (Olson et al., 2004, 2006). Similarly, when subjects were subjected to a passive 0.1 Hz cyclic load of the anterior cruciate ligament of the knee joint a neuromuscular disorder developed (Sbriccoli et al., 2005). There was a decrease in the quadriceps EMG activity and spasms were observed in both the quadriceps and hamstrings. The similarity in symptoms that were recorded in the feline model as compared to the human model is a strong indicator that the mechanisms of injury and the response to injury are the same for the two species. While the time constants and parameters may need to be adjusted for humans, our model may be useful in determining optimal work to rest ratios and rest durations that allow for a maximum amount of work done, but would protect the health and safety of the worker. Further work needs to be done to establish these model parameters in humans, for use in clinical, occupational, and athletic environments.

In conclusion, it has been shown that allowing rest of only 10 min or less between six 10-min cyclic loading periods (work to rest ratios of 1:1 or higher) will induce an acute neuromuscular disorder. On the other hand, a work to rest ratio of 1:2 (20-min rest between six 10-min loading periods) will not lead to an acute disorder. It is also evident that cyclic loading presents a higher risk factor compared to static loading in the development of CTD. Further definition of optimal work to rest ratios in cyclic loading cases needs to be examined in greater detail in order to develop guidelines or a protocol that could be utilized in preventing or limiting occupational and athletic low back pain attributed to repetitive (cyclic) lumbar flexion.

Acknowledgement

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