

ABSTRACT: Occupations requiring frequent periods of static lumbar flexion are known epidemiologically to be risk factors for the development of cumulative low back disorder. The impact of the load magnitude sustained during a series of short static lumbar flexions followed by an equally long rest period on the development of a cumulative low back disorder was addressed in an in vivo feline model. Static loads of 20, 40, and 60 N were applied over 10 min of flexion followed by 10-min rest sessions that were repeated six times (for a total of 2 h) while monitoring lumbar viscoelastic creep (laxity) and reflex electromyographic (EMG) activity from the multifidus muscles. Creep and EMG were also monitored over 7 h of rest following the six flexion–rest sessions. It was found that the creep developed in the 10-min flexion periods did not recover completely during the following 10 min of rest, giving rise to a large cumulative creep at the end of the work–rest session. Muscle activity demonstrated spasms during the static flexion periods as well as initial and delayed hyperexcitability during the 7-h rest period. Loads of 20 and 40 N did not result in delayed hyperexcitability, whereas loads of 60 N resulted in delayed hyperexcitability. Statistical analysis demonstrated that increased load significantly intensified the magnitude of the hyperexcitabilities ($P < 0.05$). Thus, repeated periods of static lumbar flexion were found to result in a transient neuromuscular disorder with an intensity directly related to the load magnitude, which should be considered a compounding risk factor.

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STATIC LOAD MAGNITUDE IS A RISK FACTOR IN THE DEVELOPMENT OF CUMULATIVE LOW BACK DISORDER

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Cumulative low back disorder (CLBD) is common in workers exposed to daily occupational activities requiring static lumbar flexion over months or years. The disorder is characterized by pain, posterior muscle stiffness (spasms), limited range of motion, and weakness. It often results in permanent disability status and thus in loss to the workforce, as well as in increased medical expenses.^{14,15,17} The disorder was determined epidemiologically to depend on the

magnitude of the loads sustained, the duration over which the load was sustained, and the number of repetitions of such loading periods throughout the day. Heavier loads applied over longer periods and repeated more frequently seem to accelerate the development of CLBD.^{7,17,18,21,24} Experimental, physiological, and biomechanical data that provide scientific validation of the epidemiologic conclusions given above are not available.

We previously reported that a single period of static or cyclic lumbar flexion in a feline model^{3,25,26} and in humans²⁷ results in laxity of the spinal viscoelastic tissues (such as ligaments, disks, and facet capsules) and significant changes of lumbar muscle activity. As the viscoelastic tissues of the spine are subjected to load, afferents within these tissues reflexly activate the paraspinal muscles.^{8,9,11,28,31} With time, the loads cause creep (laxity) in the stretched

Abbreviations: ANOVA, analysis of variance; CLBD, cumulative low back disorder; EMG, electromyogram; NIEMG, normalized integrated electromyogram

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viscoelastic tissues,^{1,4,6,12,16,29,33} which is associated with changes in the reflex activation of the muscles^{3,25–27} as well as microdamage and acute inflammation in the viscoelastic tissues.^{2,26,36} Decreasing electromyographic (EMG) activity with superimposed spasms is commonly seen during static loading of the lumbar spine. Seven hours of rest following the static loading period has been associated with an immediate hyperexcitability in the first hour, followed by gradual recovery of muscle function, and then a delayed hyperexcitability about 3–7 h later.

Although the neuromuscular disorder just described is transient, diminishing within 2–3 days with the termination of acute inflammation, fragmented evidence supports the perception that long-term (i.e., weeks or months) exposure of ligaments to daily static loads results in chronic inflammation,^{2,30} permanent changes in the mechanical properties of the ligamentous tissues,^{32,36} pain, and increased muscle activity.^{5,23,34} Experimental evidence describing the relationships between the mechanical properties of the tissues, the corresponding changes of muscle function, and tissue pathology is lacking.

The objective of this study was to assess the development of creep (laxity) within the lumbar viscoelastic tissues and the associated changes in muscle activity during a series of static loading periods spaced by equally long rest periods and then by up to 7 hours of complete rest. The effect of different load magnitudes, within the physiological range, on the muscle responses was of particular interest. We hypothesized that a series of static load–rest periods would cause creep (laxity) in the tissues that would not recover over the rest periods and would accumulate over the series of load–rest periods. We also hypothesized that decreasing EMG activity superimposed with spasms would be observed during loading periods, and that muscle hyperexcitability with intensities related to load magnitude would be present during a long recovery period.

METHODS

Preparation. Twenty-three adult cats (4.43 ± 0.39 kg) were anesthetized with a single intraperitoneal injection of chloralose (60 mg/kg) in a protocol approved by the institutional animal care and use committee. The skin over the lumbar spine was dissected from the thoracic to sacral levels and allowed to retract laterally to expose the dorsolumbar fascia. After dissection, the preparation was 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 dur-

ing the experiment to prevent the exposed tissue from drying. Three groups were used, the first subjected to 20-N load ($N = 7$), the second to a 40-N load ($N = 7$), and the third to a 60-N load ($N = 9$).

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-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 having a 110-dB common mode rejection ratio, a gain capability of up to 200,000, and a bandpass filter of 6–500 Hz. The EMG response 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 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.

Two external fixators were used to isolate the lumbar spine: one attached to the L-1 posterior spinal process and the other 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; it was not to prevent any motion.

Protocol. The stainless-steel hook applied to the L-4/5 supraspinous ligament was pulled up by the MTS actuator from a resting position to create lumbar flexion with a constant load of 20 N, which was determined in previous work²⁸ to be in the low end of the physiological range, for a 10-min period. Next, the load was fully removed, allowing 10 min in the resting position. Six such load–rest periods were applied for a cumulative exposure time to load in static flexion of 60 min. The total loading–rest time was 2 h.

During the 7 h of rest following the load–rest sessions, 8-s tests were performed to assess vertical displacement (and associated creep) and EMG recovery. Tests were applied after 10 min of rest, 30 min of rest, 60 min of rest, and each hour thereafter. Each 8-s test consisted of a 6-s linear increase in load

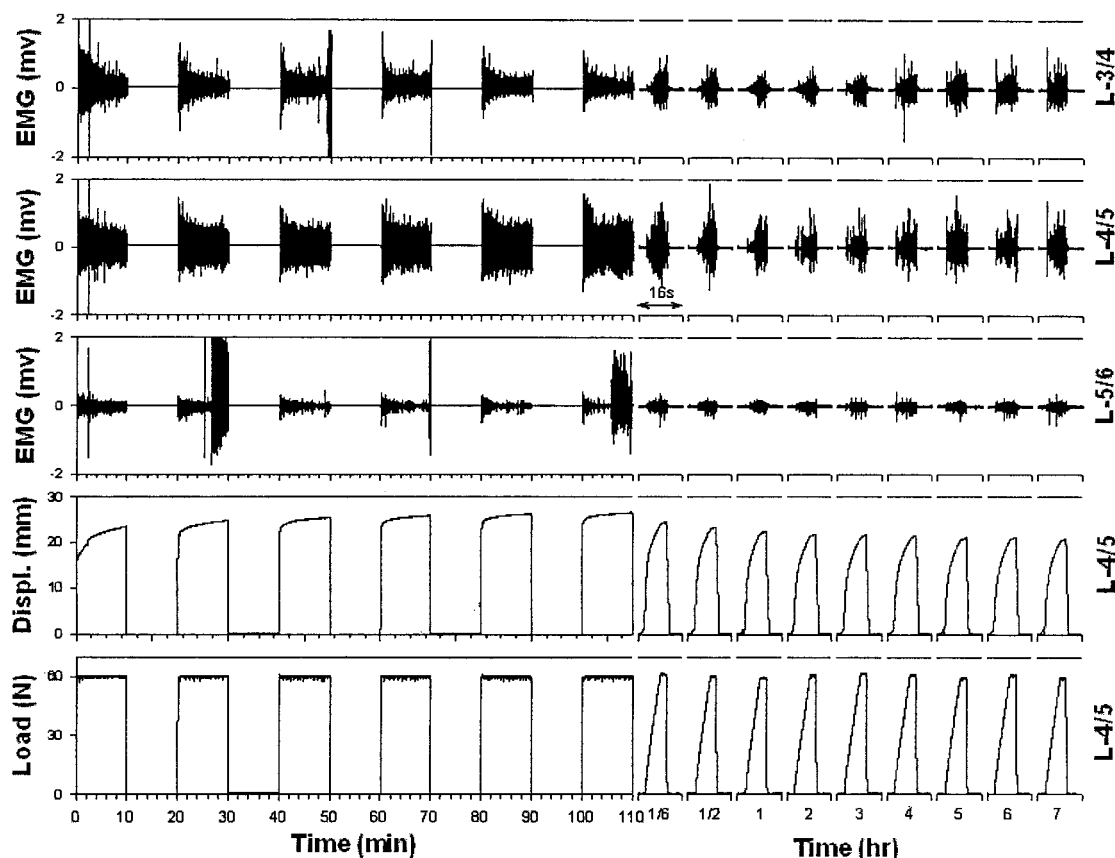


Figure 1. A typical recording of EMG from the L-3/4, L-4/5, and L-5/6 level (top three rows) as well as lumbar displacement and static load (bottom) recorded from one preparation subjected to a 60-N load. Note the large-amplitude spasms that are superimposed on the gradually decreasing EMG during different 10-min static load periods.

to 20 N followed by 2-s constant load of 20 N. The 8-s tests were recorded in 12-s windows triggered by the computer at the appropriate time. The spine remained unloaded between the specified tests (Fig. 1). The slow linear rate of increase in load over 6 s was used to avoid inflicting damage to the ligaments, which is known to occur with the exposure of viscoelastic tissues to a sudden or fast stretch.¹⁹ Similarly, the load was increased linearly to 20 N in the initial 6 s of each of the six 10-min loading periods.

The same protocol was repeated for two more load magnitudes, 40 N and 60 N, which are moderate and high, respectively, within the physiological range. The load magnitude was randomly selected for each experiment.

Analysis. We sampled 1.5-s windows of EMG, static load applied to the spine, and vertical displacement at the L-4/5 supraspinous ligament at the beginning of the loading period and every 20 s thereafter for each 10-min static loading period, as well as for the short tests in the recovery period. Each EMG sample

was full-wave rectified and integrated over the 1.5-s window, and normalized with respect to the value obtained from the first window of the first 10-min period. The normalized integrated EMG (NIEMG) of all the preparations subjected to the same load at the respective window was pooled, and the mean (and standard deviation) was calculated and plotted against time for each of the preparations.

Displacements of the respective window of all preparations subjected to the same load were pooled, and the mean (\pm SD) was calculated and plotted as displacement against time.

Model. The model considered is based on our previous work where a continuous 20-min static load was followed by a 7-h recovery period.^{3,25} In order to convert the equations to describe a series of work periods spaced by rest periods, two new time components are defined: T_w is the time period over which work (or load) was performed (or applied) by (to) the spine, and was 10 min in this study. T_R is the period of rest between any two work periods (T_w),

and was also 10 min in this study. The equation describing the NIEMG behavior during each of the work periods is rewritten as:

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

It was assumed that A and NIEMG_0 are not constant throughout the work–rest session; that is, A and NIEMG_0 are changing from one work period to the next. It was also assumed that T_1 may not be the same for all work periods.

Since this study employs only a 10-min rest, the first transient component of the recovery equation will be dominant and the steady-state component contribution as well as the delayed hyperexcitability term can be neglected. During the rest periods, therefore, the modified equation is as follows:

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

It was also assumed that the amplitudes of NIEMG_0 , and B will vary from one rest period to the next and that T_2 may vary as well.

Similarly, the equation describing the development of displacement (and indirectly creep in the viscoelastic tissues) during a series of work periods spaced by rest periods is given by:

$$\text{DISP}(t) = [D_{0n} + D_{Ln} (1 - e^{\frac{-(t - n(T_w + T_R))}{T_{n5}}})] \Big|_{n(T_w + T_R)}^{(n+1)T_w + nT_R} \quad (3)$$

where T_{n5} , D_{0n} , and D_{Ln} were assumed to be variables. The recovery of the displacement during the rest periods is described by:

$$\text{DISP}(t) = [D_{0n} + R_n + (D_{Ln} - R_n) e^{\frac{t - [(n+1)T_w + nT_R]}{T_{n6}}}] \Big|_{(n+1)T_w + nT_R}^{(n+1)(T_w + T_R)} \quad (4)$$

Again, D_0 , D_L , and R were assumed to be variable from one work–rest session to the next. T_{n6} was assumed to vary from one rest session to the next.

The long-term recovery after the six work–rest sessions was modeled by the original equation for long-term recovery.^{3,25}

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 nonlinear 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.

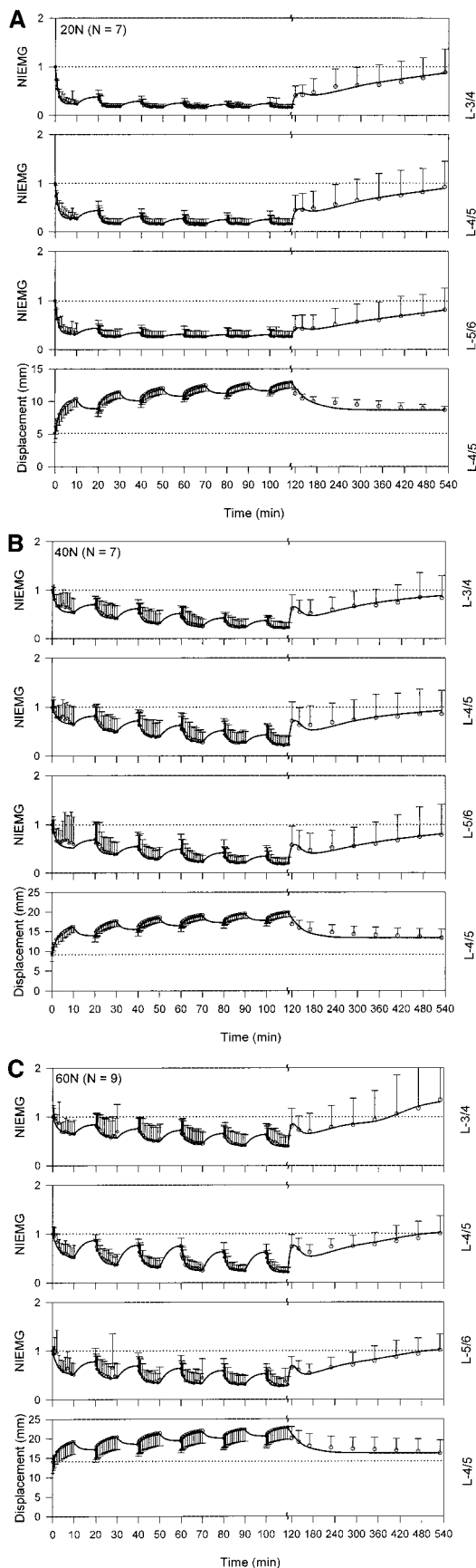
A two-way analysis of variance was performed to assess the effect of time after loading, load, and their interaction on the recovery of NIEMG at the L-3/4, L-4/5, and L-5/6 levels as well as the displacement. Significance was set at 0.05 for all statistical tests.

RESULTS

A typical recording of EMG, displacement, and load from a preparation subjected to six sessions of static flexion–rest at a load of 60 N is shown in Figure 1. The general EMG patterns exhibit an exponential decay during each session of static flexion, with partial recovery of the amplitude from the end of one flexion session to the beginning of the next. Large-amplitude spasms were observed in the flexion periods in all preparations regardless of the applied load. During the 7-h recovery period, the EMG displayed an increase in intensity after the first 10-min rest, then a decrease to the third (after 60-min rest) test and a gradual increase thereafter to the end of the recovery period. Some brief, large-amplitude spasms were observed randomly during the tests in the 7-h recovery period.

The displacement demonstrated the development of creep during the flexion periods, which partially recovered over the rest periods, leaving a large residual creep at the beginning of the next static flexion period. Overall, a large amount of creep accumulated at the end of the 2 h of static flexion–rest sessions. A gradual decrease in the displacement and creep were observed throughout the 7-h recovery period. Full recovery of the creep at the end of the 7-h rest was not observed.

Figure 2A–C displays the mean (\pm SD) of the collected data from the preparations subjected to 20-, 40-, and 60-N loads, respectively. The mean initial displacement in the preparations subjected to 20-N load was 5.1 mm and the mean creep developed during the first 10 min was 104.5%, corresponding to a mean displacement of 10.44 mm. During the following 10-min rest period it recovered to 8.871 mm, or 73.7% residual creep. In subsequent flexion–rest periods the mean residual creep accumulated, resulting in a mean cumulative creep value of 154.1% at the end of the six load–rest sessions.



The 7-h recovery period resulted in a decrease in the mean cumulative creep to 70.2%. Full recovery of the creep was not achieved in any preparation.

For the group subjected to a 40-N flexion load, the mean initial displacement at the beginning of the first 10-min session was 9.352 mm, increasing to 16.25 mm at the end of the first 10 min (mean creep of 73.77%). The first rest period resulted in a decrease of the mean creep to 48.4%. The creep accumulated over the following flexion–rest periods to a final value of 113.19%. During the recovery period, the mean creep decreased to 42.8%. Full recovery was not observed in any preparation.

The mean creep developed in the first 10-min flexion–rest period under a load of 60 N was 38.1%, which accumulated to a final mean cumulative creep of 63.5%. This recovered to a mean value of 15.9% at the end of the 7-h rest period. Full recovery was not observed in any preparation.

The mean NIEMG (normalized integrated EMG) in the L-3/4, L-4/5, and L-5/6 multifidus subjected to 20-N load decreased from 1.0 to 0.231 (76.9% decrease), 0.269 (73.1% decrease), and 0.355 (64.5% decrease), during the first 10-min flexion session and recovered to 0.37, 0.436, and 0.421 during the following 10-min rest, respectively. The following flexion–rest sessions resulted in a relatively minor decrease and recovery in the NIEMG, which was 0.164 (or 83.6% decrease), 0.159 (84.1% decrease), and 0.265 (73.5% decrease) for the three lumbar levels, respectively, at the end of the flexion–rest sessions.

During the 7-h recovery period, the NIEMG exhibited a sharp increase in the first 10 min followed by a minor decrease before a long gradual increase to 0.887, 0.923, and 0.813 at the end of the rest, in the L-3/4, L-4/5, and L-5/6 multifidus muscles, respectively. Full recovery occurred in some preparations.

For flexion loads of 40 N, the mean NIEMG decreased to 0.536, 0.645, and 0.602 in the L-3/4, L-4/5, and L-5/6 multifidus, respectively, during the first 10-min flexion session. The first 10 min of rest allowed recovery to 0.705, 0.817, and 0.695 at these levels, respectively. The following flexion–rest sessions resulted in a further gradual decrease and partial recovery, ending with a final mean NIEMG of

Figure 2. The mean (\pm SD) NIEMG of the L-3/4, L-4/5, and L-5/6 multifidi as well as the mean displacement during the six work/rest periods and the 7-h recovery are shown for loads of 20 N (A), 40 N (B), and 60 N (C). The solid lines through the data points represent the models developed to describe NIEMG and displacement during work–rest and long-term recovery.

0.227, 0.231, and 0.199 at the same levels. During the 7-h recovery period, the mean NIEMG increased in the first 10 min of rest, followed by a minor decrease. A steady gradual increase followed thereafter, ending with values of 0.841, 0.864, and 0.787 after 7 h. Full recovery of the NIEMG was observed in some preparations.

Similarly, for 60-N flexion load, reductions in NIEMG to 0.652, 0.523, and 0.511 were observed in the first flexion session, followed by recovery to 0.827, 0.862, and 0.77 at the end of the first 10-min rest period. The final mean NIEMG was 0.4, 0.217, and 0.299 at the end of the six flexion–rest sessions. During the recovery period, the mean NIEMG peaked within the first 10 min, decreased somewhat, and then gradually increased to 1.348, 1.003, and 1.007 at the end of the 7-h recovery. Several preparations exhibited individual NIEMG values well above 1.0 at the end of the 7 h of rest.

The models developed for the displacement and NIEMG data of Figure 2 are superimposed on the experimental data. For the 20-N load, A_n , T_{n1} , and NIEMG_{0n} were indeed changing from one work session to the next. For the NIEMG model in the L-3/4 level, A_n was 0.769 for the first work period and gradually decreased to 0.081, a 10-fold decrease in the six work periods. The decreases in A_n for the L-4/5 and L-5/6 levels were from 0.731 to 0.093 and from 0.684 to 0.029, respectively. Similarly, the T_{n1} of the L-3/4 muscles gradually decreased from 1.2 min in the first work session to 0.7 min in the sixth session. T_{n1} decreased from 1.3 min to 0.6 min and from 1.3 min to 0.5 min in the L-4/5 and L-5/6, respectively. The NIEMG_{0n} of the L-3/4 multifidus also gradually decreased from 0.231 to 0.164. The NIEMG_{0n} decreases for L-4/5 and L-5/6 were from 0.269 to 0.159 and from 0.316 to 0.265, respectively. During the rest periods the T_{n6} was constant at 2 min, D_{0n} gradually increased, and D_{Ln} and R_n decreased.

During the rest periods, the NIEMG displayed a constant T_{n2} , mostly at 10 min. B_n and NIEMG_{0n} were, however, gradually decreasing, indicating a gradually slower recovery.

The displacement model points out that the time constant was unchanged at 2.5 min over the six work sessions, but there was an increasing D_{0n} and decreasing D_{Ln} from session to session. Similarly, the time constant, T_{n6} , was constant at 2 min throughout rest periods but D_{0n} increased, and D_{Ln} and R_n decreased from session to session.

Similar patterns were present in the model parameters associated with loads of 40 N and 60 N. T_{n1} and A_n gradually decreased from one work session to

the next. T_{n2} was constant at 10 min in the rest periods, whereas B_n was relatively constant and NIEMG_{0n} slowly decreased. For the displacement during the six work periods, T_{n5} was nearly constant at 2.8 min, but D_{0n} and D_{Ln} were increasing and decreasing, respectively. In the rest periods, T_{n6} was constant at 2 min, D_{0n} increased, and D_{Ln} and R_n decreased.

The models developed were in good agreement with the experimental data, resulting in R^2 values of 0.9 or above, except in some cases where the spasms interrupted the smooth changes in the EMG discharge. Spasms, being random and unpredictable, could not be quantified or filtered from the experimental data.

During model development for the NIEMG in the 7-h recovery period, delayed hyperexcitability had nearly no effect on the final models for the 20- and 40-N loads. The constant, C , was very small, ranging from 0.0001 to 0.0005, and the time constants, T_d and T_d , were 300 min and 350–450 min, respectively. The only exception was the 60-N load, where C was 10 times larger for L-3/4, L-4/5, and L-5/6, at 0.004 and 0.001, respectively. T_d and T_d were also shorter, at 380 min and 250 min, respectively, indicating the earlier onset and faster development of delayed hyperexcitability. Indeed, the NIEMG data for the 60-N load showed that, at 7 h, values for the three lumbar levels exceeded 1.00 with the L-3/4 level showing the highest value of 1.348, confirming the presence and effect of hyperexcitability well within the recovery period. This is in contrast to the NIEMG values at the end of the 7-h recovery for the 20- and 40-N loads, which were below 1.0, ranging between 0.78 and 0.92. Figure 3 provides the experimental data and models for the 7-h recovery period for each of the three lumbar levels.

Table 1 shows the results of two-way analysis of variance (ANOVA). For all parameters assessed (NIEMG L-3/4, NIEMG L-4/5, NIEMG L-5/6, and displacement), there was a significant effect of time, indicating statistically significant variations in the parameters as time progressed. For NIEMG L-3/4, NIEMG L-5/6, and displacement, there was a significant effect of load, indicating significant ($P < 0.05$) variations in recovery dependent upon load; this effect achieved only a marginal effect ($P < 0.06$) for the NIEMG L-4/5. Interestingly, all parameters had a near-unity P -value for interaction, indicating that all curves were parallel to each other with respect to time.

DISCUSSION

The most important findings of this investigation were that a work–rest condition composed of six 10-min work and 10-min rest sessions resulted in a cumulative creep that did not recover over a 7-h rest period, leaving a substantial residual creep for any new work activity. The work–rest period, although generous with respect to rest (1:1 ratio), was sufficient to elicit a neuromuscular disorder consisting of spasms and muscle hyperexcitability, with a delay in recovery of normal muscle function. Importantly, the neuromuscular disorder was dependent on the load level sustained during the work period. Larger loads elicited a larger initial as well as delayed hyperexcitability of the multifidus muscles well past the 7-h rest period despite the highly favorable work–rest ratio.

As pointed out earlier, the neuromuscular disorder elicited by periods of static flexion consist of five components: decreasing EMG and superimposed spasms during flexion, and slow recovery of EMG with initial and delayed muscular hyperexcitability during the following 7 h of rest.^{25,26} It is therefore appropriate to assess the effect of the load magnitude in this investigation by comparative analysis of these five components for each of the loads.

Spasms were evident during the 10-min loading sessions regardless of the load magnitude applied. However, their frequency of occurrence, the duration of persistence, and amplitude of the spasms were very low for the 20-N load compared with the 40- and 60-N loads. It seems that strain or deformation of viscoelastic tissues results in some microdamage in the collagen fibers, irrespective of the load applied.³⁶ As shown in previous studies, such a strain–microdamage process results in reflex activation of the muscles via pain afferents, leading to the observed spasms,^{10,35} a phenomenon shown by Pedersen et al.²⁰ about 50 years ago. Since spasms occur unpredictably, a model could not be developed. Spasms also decreased the *P*-values of the models with respect to the actual data points during the 10-min loading periods. The pattern, however, was very clear and well defined by the model. Considering that two separate pathophysiological processes (creep and tissue microdamage) were presumably active simultaneously, the model managed to identify the neurological response to creep rather accurately. The presence of spasms is considered a disorder without a need for accurate quantification.

The peak of the initial hyperexcitability as measured 10 min into the recovery period was dependent on the load magnitude. From Figure 3, the

L-3/4 level peak initial hyperexcitability was 0.403, 0.621, and 0.805 for the 20-, 40-, and 60-N loads, respectively. Similarly, for the L-4/5 level, the peak was 0.442, 0.718, and 0.734, respectively, for the three loads; at L-5/6, the corresponding values were 0.427, 0.582, and 0.644. The models further confirm that larger loads resulted in larger magnitude of the initial hyperexcitability. For a load of 20 N, the constant, *B*, which governs the amplitude of the initial hyperexcitability component, ranged between 0.025 and 0.04 in the three lumbar levels; it ranged from 0.065 to 0.08 for the 40-N load and from 0.085 to 0.095 for the 60-N load. The time-constant T_2 also changed with loads, decreasing for larger loads, ranging from 16 to 18 min for 20 N, from 14 to 15 min for 40 N, and from 11 to 14 min for 60 N. Shorter time-constants showed a faster rise in the initial hyperexcitability amplitude, indicating the urgency of muscle assistance in providing stiffness to the intervertebral joints where the viscoelastic tissues are lax and have microdamage.

The delayed hyperexcitability represents the muscle's reaction to the development of an acute inflammation in the strained and microdamaged viscoelastic structures.²⁶ Since inflammation is a relatively slow metabolic process consisting of migration of neutrophils and cytokines from the vascular system into the damaged tissue, several hours are required before the inflammatory response is pronounced. It is evident from Figures 2 and 3 that a

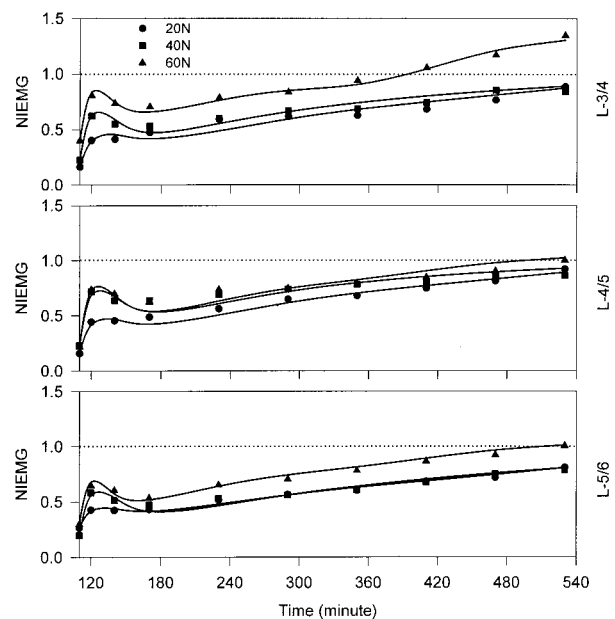


Figure 3. The mean NIEMG data and the developed models for the 7-h recovery period are shown superimposed for 20-, 40-, and 60-N loads.

Table 1. Statistical analysis using two-way ANOVA.

Parameter	Effect of time (<i>df</i> = 8)		Effect of load (<i>df</i> = 2)		Interaction (<i>df</i> = 16)	
EMG L-3/4	<i>f</i> = 2.39	<i>P</i> = 0.018	<i>f</i> = 8.63	<i>P</i> = 0.0003	<i>f</i> = 0.14	<i>P</i> = 1.0
EMG L-4/5	<i>f</i> = 2.35	<i>P</i> = 0.020	<i>f</i> = 2.82	<i>P</i> = 0.062	<i>f</i> = 0.14	<i>P</i> = 1.0
EMG L-5/6	<i>f</i> = 3.18	<i>P</i> = 0.002	<i>f</i> = 4.63	<i>P</i> = 0.011	<i>f</i> = 0.08	<i>P</i> = 1.0
Displacement	<i>f</i> = 5.03	<i>P</i> < 0.0001	<i>f</i> = 223.9	<i>P</i> < 0.0001	<i>f</i> = 0.09	<i>P</i> = 1.0

ANOVA, analysis of variance; *df*, degrees of freedom; EMG, electromyography.

delayed hyperexcitability was not present during the 7-h recovery period for loads of 20 N and 40 N. The models further confirm that the delayed hyperexcitability component was minimal or nonexistent for the low and moderate loads. By contrast, the data and associated model for the 60-N load show the presence of delayed hyperexcitability. The NIEMG data point at 7 h exceeded 1.0 at each of the three lumbar levels for the 60-N load, whereas the corresponding data points were below 1.0 for the 20- and 40-N loads. The model further confirms the presence of delayed hyperexcitability, with the constant, *C*, being 10 times larger (0.001–0.004) for the 60-N load than for the two smaller loads (0.0001–0.0005). The time constant *T*₄ was also much shorter, at 250 min, as would be expected with a relatively faster metabolic process associated with the deposit of neutrophils and cytokines in damaged viscoelastic tissues. The time delay, *T*₀, indicating the initiation of the effect of the delayed hyperexcitability was 380 min for the 60 N load in contrast to the 450 min for the two smaller loads, further confirming the pronounced effect of larger load on the neuromuscular disorder.

The statistical analysis also indicates that the NIEMG recovery curves from the L-3/4 and L-5/6 lumbar levels were significantly different (*P* < 0.05) for different loads, with increased NIEMG hyperexcitability when subjected to larger loads. For the L-4/5 lumbar level, the hyperexcitability of the multifidus muscles was larger for larger loads at a significance level of *P* < 0.062.

Overall, the spasms during the static flexion period as well as the initial and delayed muscle hyperexcitability during the 7-h recovery period were more intense when the lumbar spine was subjected to loads of larger magnitude, experimentally confirming the epidemiological data that load magnitude is indeed a compounding risk factor in the development of CLBD.

The valuable effect of frequent rest was highly pronounced in this study compared with our previous one.²⁵ In the earlier study, a 20-min static flexion followed by 7-h rest resulted in an intense low back

disorder, including spasms and initial and delayed hyperexcitability, irrespective of the load level. In the data presented here, the cumulative flexion time was three times longer (i.e., 60 min rather than 20 min), yet delayed hyperexcitability did not manifest itself for loads of 20 N and 40 N. The equally long rest periods seem to attenuate substantially or even prevent the development of delayed hyperexcitability and its associated microdamage and inflammation. Frequent rest periods, therefore, are a valuable, effective, and necessary preventive tool that should be properly used when designing work schedules consisting of periods of static lumbar flexion.

The flexion–rest schedule selected in the loading paradigm of this experiment was deliberately set at a highly favorable ratio of 1:1 (i.e., 10-min flexion followed by 10-min rest). The epidemiological data point out a possible dose–duration relationship where the duration of the static load in flexion is a risk factor. We therefore sought to minimize this effect with a shorter flexion period and equally long rest period in order to bring out clearly the effect of load magnitude and avoid compounding of two effects. The reality of the workplace, however, stipulates substantially longer work periods. Under such circumstances, the magnitude of the load applied during flexion will probably have a substantially larger effect on the intensity of the resultant CLBD.

The results of this study provide new insight into the development of the initial phase of CLBD from electrophysiological and biomechanical perspectives. Larger loads seem to elicit creep in the lumbar viscoelastic tissues, which presumably results in greater microdamage to the collagen fibers and, in turn, gives rise to intense inflammation in these tissues. The muscles are reflexly-recruited to increase the stiffness of the joints for the purposes of maintaining stability and preventing or limiting exposure of the viscoelastic tissue to any additional damage. It is evident that an overnight rest of 7 h is not sufficient to restore the creep developed in the tissues or to resolve the acute inflammation developed therein. Affected workers, therefore, will start the next work day with residual creep, microdamage,

and acute inflammation, and further compound these factors with additional static work. It is conceivable that, with long-term exposure to static flexion, the acute inflammation will progress to chronic inflammation, which is known to be associated with pain, muscle stiffness, weakness, and limited range of motion.^{13,22} Treatment of chronic inflammation with prolonged periods of rest never restores full strength of viscoelastic tissues^{32,36} or fully resolves the impact of any inflammation. Permanent disability, therefore, should be avoided with preventive measures, such as avoiding long-term exposure to static activities. Finally, sufficient rest periods spaced within static flexion periods seem to represent an effective and valuable preventive measure, attenuating the development of CLBD.

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