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Cyclic Load Magnitude is a Risk Factor for a Cumulative Lower Back Disorder

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Learning Objectives

- Summarize what previous studies have shown about the effects of static loading of the lumbar spine on regional viscoelastic tissues such as ligaments and intervertebral discs and on muscle tissue.
- Explain how, in electromyographic studies of the lumbar spine in cats, the degree of cyclical (repetitive) loading influenced the magnitude and duration of delayed muscular hyperactivity.
- Estimate the respective contributions of cyclical loading of the lumbar spine to acute and chronic inflammatory changes, and compare the effects of cyclical loading to those of static loading.

Abstract

Objective: *Epidemiological data suggest that high loads lifted by workers engaged in static and cyclic daily activities may be a risk factor for low back disorder. Our previous research provided physiological and biomechanical validation of the epidemiological data for static load conditions. The objective of this report was to provide physiological and biomechanical experimental validation to the epidemiological data in cyclic (repetitive) load conditions.* ***Methods:*** *Three groups of in vivo feline models were subjected to 3 cyclic load levels in a series of 6 periods of 10 minutes of work spaced by 10 minutes of rest followed by 7 hours of rest. Multifidus electromyography (EMG) and lumbar displacement were statistically analyzed after processing.* ***Results:*** *Delayed muscular hyperexcitability was observed only in moderate (40 N) and high (60 N) loads ($P < 0.0001$) but was absent in low (20 N) loads. The magnitude of the delayed hyperexcitability was found to be higher ($P < 0.0001$) in the high (60 N) loads compared with the moderate (40 N) loads.* ***Conclusions:*** *Exposure to moderate and high loads in cyclic (repetitive) work results in an acute neuromuscular disorder indicative of soft tissue inflammation that may become chronic with further exposure.* (J Occup Environ Med. *2007;49:375–387*)

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This work was supported by grant R01-OH-007622 from the National Institute for Occupational Safety and Health (NIOSH).

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DOI: 10.1097/JOM.0b013e318046eb0b

Cumulative trauma disorder (CTD) is common among workers exposed to intense physical activities as part of their daily occupational routine.[1] The disorder expresses itself as muscular weakness, limited range of motion, stiffness/spasms, and pain in the affected joints. CTD is unresponsive to various conventional treatments, and prolonged periods of nonexposure (eg, rest) provide only partial relief. Up to 2 years of nonexposure results in diminished pain and regain of strength and range of motion of up to only 70% of what was available previously.[1,2]

The epidemiological literature found a strong statistical correlation between static and cyclic (repetitive) work activities and CTD. Specifically, the risk factors were shown to be high loads, longer durations, and the number of repetitions of exposure to the loads.[1,3,4,5] Experimental physiological and biomechanical confirmation of the epidemiological findings were lacking. Furthermore, the etiology of CTD in terms of the affected organs (muscles, tendons, ligaments, nerves, etc) and the method of failure were unknown.

A series of projects undertaken in the last few years provided valuable new insights.[6–11] Working with the feline model, researchers have shown that a series of periods of exposing the lumbar spine to static loads spaced with rest periods (for example, six periods of 10 minutes of work with 10 minutes of rest in between) resulted in substantial elongation and laxity of the viscoelastic tissues (ligaments, discs, facet cap-

sule, etc) while triggering spasms in the multifidus muscles. Seven hours of rest after loading were not sufficient for restoring the original resting length or tension of the viscoelastic tissues. In loading paradigms that employed high loads, longer loading durations, short rests in between, and higher repetitions, a delayed multifidus hyperexcitability was observed 2 to 3 hours into the 7-hour rest period. The hyperexcitability was also associated with the development of an acute inflammation in the supraspinous ligaments as expressed by more than a 1000-fold increase in the density of the neutrophil in the ligament.[10]

Demonstrating that high loads, long work periods, short rest periods, etc, are risk factors in cyclic work is still necessary.

The new insights gained pointed out that the failed organs were the viscoelastic tissues. The new insights listed above also serve as the rationale for formulating a hypothesis for the development of CTD. We were able to formulate the hypothesis that the collagen fibers of the viscoelastic tissues develop microdamage as a direct result of exposure to the load. As the microdamage exceeds a certain threshold due to high loads, many repetitions, and/or insufficient rest, an acute inflammation is triggered, which, in turn, elicits hyperexcitability in the lumbar muscles in order to protect the soft tissues from additional damage. The acute inflammation most likely can be resolved spontaneously within 2 to 3 days, yet workers do not have this option. Returning to work with an acute inflammation and being subjected to additional exposure to tissue loading can, over time, lead to a chronic inflammation and a permanent disability.[12]

Another series of experiments with normal human subjects exposed to static and repetitive (cyclic) loading of the lumbar spine and the knee resulted in spasms and significant changes in muscular activity and synchronization, confirming the potential of an acute disorder[13–18] and the applicability of the feline findings in humans.

Overall, the outcome from the feline model and human subjects' studies provided a developing experimental infrastructure to confirm epidemiological predictions, identify possible organs, and formulate a hypothesis of their failure as well as assist in developing a model of the initial phase of the development of CTD.

Experimental physiological and biomechanical data confirming that high-load magnitudes are a risk factor for CTD in repetitive loading is still lacking and is the subject of this report. There are significant differences between cyclic and static loads. Static loading includes a single tissue-stretching action and long periods of maintaining the stretch (eg, anterior flexion action and then remaining flexed for a period). A typical example is a concrete, farm, or roofing worker who bends over and then remains in that condition for an extended period of time while performing his work. Cyclic loading, however, includes multiple tissue-stretching actions while maintaining the maximal stretch only for a brief moment. A typical example is a worker lifting a shipment of 50 boxes from the floor to a moving belt. In this condition, the posterior viscoelastic tissues stretch during the anterior flexion phase to the deepest angle needed to grasp the box and then immediately shorten as the spine extends during the lifting phase. This function is repeated 50 times until all the boxes are loaded, requiring the repeated stretching of the posterior viscoelastic tissues as opposed to a single stretch in the static example above.

We hypothesize that application of high-magnitude cyclic loads to the lumbar spine will elicit a delayed hyperexcitability after 7 hours of rest, whereas light loads applied for the same duration will not. It is expected that the new information resulting from this study will complement our knowledge gained so far for static loads in confirming epidemiological data, in understanding the risk factors for the development of CTD, and in developing new strategies for prevention.

Materials and Methods

Preparation. Nineteen cats (3.95 ± 1.15 kg) were anesthetized intravenously (IV) with alphachloralose (60 mg/kg) after a preanesthetic dose of xylazine (intramuscular) or isoflurane (gas) and atropine (IV) in a protocol approved by the Institutional Animal Care and Use Committee. An incision was made from the thoracic to the sacral level and then retracted laterally to expose the dorsolumbar fascia. A stainless-steel s-shaped hook was then applied around the supraspinous ligament between L4 and L5. The preparation was then placed on a rigid stainless-steel frame with external fixtures that isolated the lumbar spine from the thoracic and sacral levels. A saline-soaked gauze pad was placed over the exposed area to prevent the tissue from drying. Three groups were used at the cyclic frequency of 0.25 Hz, one subjected to a 20-N cyclic load (n = 6), another at 40 N (n = 7), and the last group at 60 N (n = 6).

Instrumentation. With the specimen positioned prone on the stainless-steel frame, external fixtures were placed on the posterior processes of L1 and L7 to isolate the specimen. The external fixtures were placed on the ends of the lumbar vertebrae in order to prevent interaction with the thoracic, sacral, and pelvic sections of the spine. Experimentation was limited to the lumbar vertebrae.

Three pairs of EMG fine-wire electrodes were inserted 5 mm to 6 mm to the right of the midline into the multifidus muscles at L3–L4, L4–L5, and L5– L6, respectively. A ground electrode was placed in the gluteus muscle. Each pair of electrodes 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 6Hz–500Hz. The EMG response was constantly monitored on oscilloscopes and recorded to a computer at a sampling rate of 1000 Hz.

A stainless-steel s-shaped hook was inserted around the supraspinous ligament between L4 and L5 and connected to the vertical actuator of the 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. Vertical displacement of the actuator and the load cell output were also sampled into the computer along with the EMG data.

Protocol. A tensile vertical load of 20 N was applied onto the stainless-steel hook between L4 and L5 at a frequency of 0.25 Hz for a period of 10 minutes. The 20-N force was determined in a previous paper to be at the low end of the physiological range, just above the threshold for action potential.[19] Next, the load was withdrawn, and the preparation was subjected to a resting period of 10 minutes. Six such cyclic loading-rest periods were applied for a total of 2 hours.

During the 7-hour rest/recovery period following the cyclic loading/rest period, 8-second tests were performed to assess vertical displacement along with residual creep and EMG recovery. Tests were applied after 10, 20, and 30 minutes of rest and at each hour subsequently. Each 8-second test consisted of a single cycle to 20 N peak. The 8-second tests were triggered by the computer at set intervals. Vertical tension was released between the recovery tests.

The same protocol was also used for cyclic loading at 40 N and 60 N for comparison with 20 N.

Analysis. Cyclic loading, vertical displacement, and EMG response were recorded in 1.5-second increments. After the first 10-minute loading cycle, samples were recorded every 20 seconds during each 10-minute cyclic loading session and for each subsequent test during the recovery period.

Each EMG sample was integrated during the respective window and normalized with respect to the value of the first window of the 10-minute loading period in each data set. The normalized integrated EMG (NIEMG) of each specimen was classified into its respective load. The mean (±standard deviation [SD]) was plotted as three different sets: NIEMG, load, and displacement versus time.

Model. The model being utilized is from previous work based on 10-minute static loading and 10-minute rest periods for a 2-hour period and then a 7-hour recovery period.[6,7,9,11,20] The static loading experiments were used as a comparative study with cyclic loading. In this study, the model equations are analyzed based on cyclic loading behavior in which the equations represent two separate entities—one being the cyclic loading period, and the other based on the recovery period. The model for the loading session of NIEMG can be described by the following equation:

$$NIEMG(t) = Ae^{-t/T_1} + NIEMG_0, \quad (1)$$

where $NIEMG_0$ is the steady-state amplitude, A is the amplitude of the exponential, t is the time and T_1 is the time constant. Both t and T_1 assist in defining the rate of decay.

The model for long-term recovery is also based on exponential decay. It is described by the following equation:

$$NIEMG(t) = tBe^{-t/T_2} + E(1 - e^{-t/T_3}) + C(t - T_d)e^{-(t - T_d)/T_4} + NIEMG_0, \quad (2)$$

where B, C, and E describe the amplitude of each term. The first term, tBe^{-t/T_2}, describes the initial transient hyperexcitability. It reaches a peak within the first 10 minutes of recovery and then decays within 1 hour. Steady-state recovery can be defined by $E(1 - e^{-t/T_3})$. This term slowly rises exponentially during the recovery period to the initiation of delayed hyperexcitability. The time delay (T_d), describes the initiation of delayed hyperexcitability.

In order to describe the relative work/rest sessions, we added two variables to the equation. Equation 1 is rewritten as equation 3 to account for the spacing of the work/rest sessions. The variables are defined as T_w being the period of time in which the cyclic loading was applied (10 minutes), and T_r is the rest period in between periods of work (10 minutes).

$$NIEMG(t) = A_n e^{\frac{-[t - n(T_w + T_r)]}{T_{n1}}\Big|_{n(T_w + T_r)}^{(n+1)T_w + nT_r}} + NIEMG_{0n} \quad (3)$$

The amplitude A_n and time constant T_{n1} are variable throughout the experiment. $NIEMG_{0n}$ is the residual value from the previous cycle during the work-rest session. Both variables are allowed to change from one work session to the next.

To account for the 10-minute resting session, only the transient component in the equation for recovery is used. The steady state and hyperexcitability terms are neglected, since their time constants are very large relative to the transient component. We used the following equation during the rest session:

$$NIEMG(t) = (t - [(n + 1)T_w + nT_r]) \times B_n e^{\frac{t - [(n+1)T_w + nT_r]}{T_{n2}}\Big|_{(n+1)(T_w + nT_r)}^{(n+1)(T_w + T_r)}} + NIEMG_{0n} \quad (4)$$

The amplitude (B_n), time constant (T_{n2}), and $NIEMG_{0n}$ are also variable during the resting periods in the 2-hour work-rest session.

In a similar manner, an equation describing the residual displacement

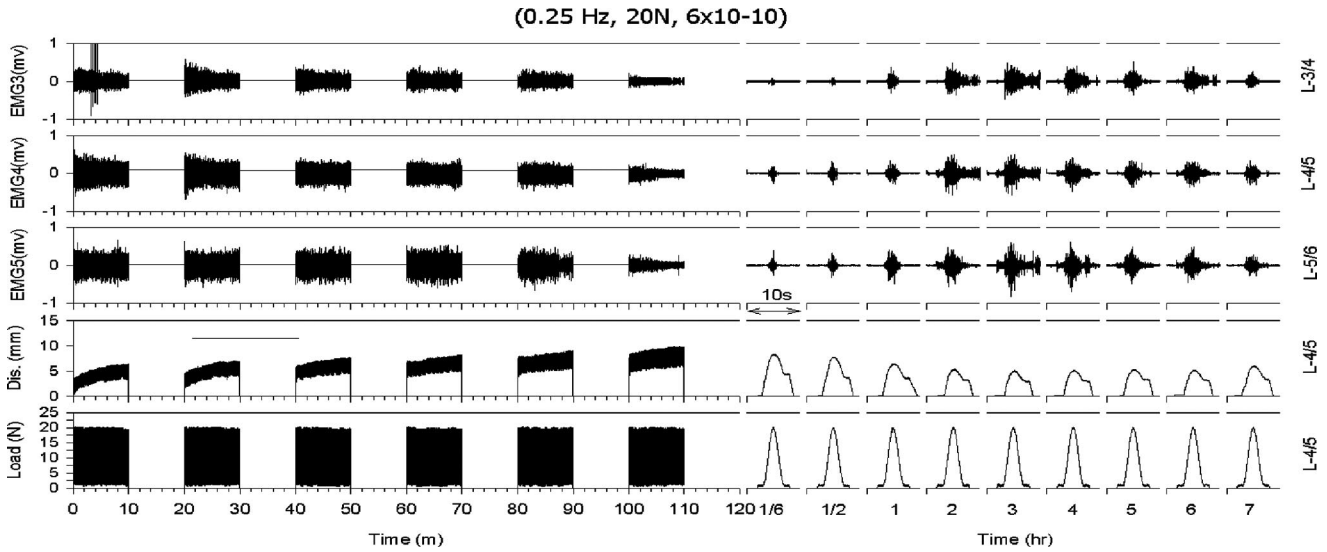

Fig. 1. A typical recording of EMG from L3–L4, L4–L5, and L5–L6 from one specimen subjected to six sessions of periodic cyclic loading (20 N) with a 1:1 work/rest ratio and 7-hour recovery period. Note the large-amplitude spasms in each loading session after initial hyperexcitability.

(viscoelastic creep) is defined throughout the work periods as:

$$\delta(t) = \left[\delta_{0n} + \delta_{Ln}\left(1 - e^{\frac{[t - n(T_w + T_r)]}{T_{n5}}}\right)\right]\Big|_{n(T_w + T_r)}^{(n+1)T_w + nT_r} \quad (5)$$

Tn_5, δ_{0n} and δ_{Ln} are variables from one work-rest session to the next. Residual recovery from viscoelastic displacement is expressed in the following equation:

$$\delta(t) = \left[\delta_{0n} + R_n + (\delta_{Ln} - R_n) \times e^{\frac{t - [(n+1)T_w + nT_r]}{T_{n6}}}\right]\Big|_{(n+1)(T_w + nT_r)}^{(n+1)T_w + T_r} \quad (6)$$

Variables from the residual recovery equation include Tn_6, R_n, δ_{0n}, and δ_{Ln}. The T_{n6} is a variable factor between rest sessions.

The recovery period after six work-rest periods was modeled using the original equations for long-term recovery.

Best-fit models were generated after the calculation of the mean and standard deviation using the Marquardt-Levenberg nonlinear regression algorithm. In some cases, convergence of the algorithm had failed. Therefore, in order to optimize the regression, sequential recursive iteration was used to determine the initial and final values.

In order to evaluate the effects of loading on recovery time and displacement, we completed a two-way analysis of variance (ANOVA). Statistical significance was set at 0.05 for all tests.

Results

A typical recording of EMG data from an experimental specimen subjected to 20-N and 6 cyclic load-rest sessions is shown in Fig. 1. The patterns in the EMG suggest exponential decay during the cyclic work sessions, with partial recovery in between sessions. Large-amplitude spasms appeared randomly as the EMG response decreased. Multifidus spasms occurred throughout all classifications of load regardless of the applied load magnitude. At the end of the 2-hour cyclic work period, creep had accumulated substantially and did not recuperate after the 7-hour recovery period.

Data was separated into the three loading classifications for comparison: 20 N, 40 N, and 60 N at 6 sessions of 10-minute 1:1 work/rest ratios (for a total of 2 hours). Figure 2A–C displays the mean ± SD data for the three load categories. The data exhibited exponential rise and decay of displacement denoting the creep and its recovery, respectively.

The group subjected to the 20-N cyclic load (Fig. 2A) had an initial mean displacement of 3.50 mm, with an increase to 7.14 mm at the end of the first 10-minute loading period. The associated mean creep was 104%, and the first rest period exhibited a decrease in mean creep to 57.1% of the initial mean displacement. Creep that accumulated over the 2-hour cyclic work/rest session resulted in a final value of 177.4%. At the end of the 7-hour recovery period, the mean creep decreased to 65.1% of the initial mean displacement.

For the group subjected to the 40-N cyclic load (Fig. 2B) the initial mean displacement was 7.28 mm, with an increase to 11.99 mm (65% mean creep) at the end of the first 10-minute loading period. A decrease in mean creep to 34.1% of the initial mean displacement was observed after the first rest period. Creep accumulated over the periods of cyclic work/rest to 116.1% of the

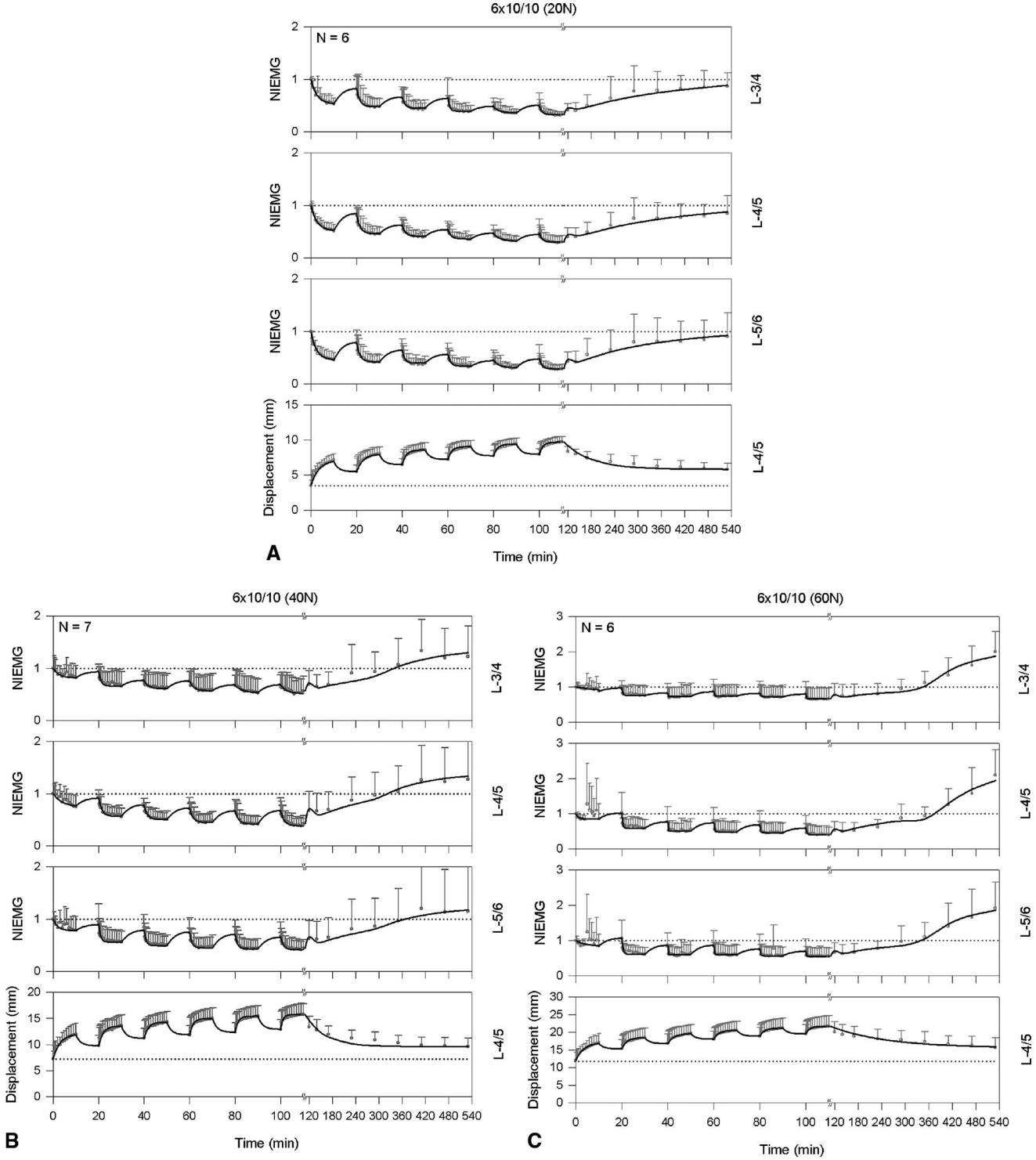

Fig. 2. The mean ± SD of the NIEMG collected at L3–L4, L4–L5, and L5–L6 along with mean displacement. The exponential model is represented by a superimposed nonlinear fit line. A linear increase during the recovery period with a higher value at the end (compared with the beginning) of the experiment indicates acute inflammation and neuromuscular disorder. The data displays loads at (A) 20 N, (B) 40 N, and (C) 60 N.

initial mean displacement. At the end of the recovery period, the mean creep decreased to 31.7%.

For the 60-N load (Fig. 2C), there was a 42% mean creep at the end of the first work period, and the first rest period exhibited a decrease in mean creep to 27.1% of the initial mean displacement. Creep accumulated over the cyclic work/rest sessions resulted to 80%. At the end of the recovery period, the mean creep decreased to 29.4% of the initial mean displacement.

In all of the preparations, none of the specimens exhibited full recov-

TABLE 1

A. Model Parameters (6 × 10-10, 20 N)

Work Period: $NIEMG(t) = \Sigma\ (A_n e^{-t/Tn1} + NIEMG_{ssn})$

Vertebral Level	n	A_n	T_{n1} (min)	$NIEMG_{ssn}$	r^2
L3–L4	0	0.4626	2.7	0.5257	0.9565
	1	0.3519	1.4	0.4770	0.9536
	2	0.1979	1.3	0.4491	0.9336
	3	0.2576	0.7	0.3922	0.8950
	4	0.1184	2.4	0.3585	0.8633
	5	0.1667	1.2	0.3260	0.9445
L4–L5	0	0.4767	2.7	0.5113	0.9938
	1	0.3561	1.6	0.4571	0.9209
	2	0.2016	1.5	0.4086	0.9650
	3	0.1721	1.4	0.3594	0.9314
	4	0.1411	2.8	0.3168	0.8863
	5	0.1689	1.2	0.3018	0.9653
L5–L6	0	0.5363	2.5	0.4537	0.9314
	1	0.3658	1.5	0.4181	0.9204
	2	0.2424	1.1	0.3952	0.9193
	3	0.2219	1.1	0.3389	0.8786
	4	0.1380	2.4	0.3089	0.8721
	5	0.1965	1.2	0.2862	0.9528

Rest Period: $NIEMG(t) = \Sigma\ (tB_n e^{-t/Tn2} + NIEMG_{ssn})$

Vertebral Level	n	$NIEMG_{ssn}$	B_n	T_{n2}
L3–L4	0	0.5257	0.08	10
	1	0.4770	0.05	10
	2	0.4491	0.05	10
	3	0.3922	0.025	10
	4	0.3585	0.04	10
L4–L5	0	0.5113	0.09	10
	1	0.4571	0.045	10
	2	0.4086	0.035	10
	3	0.3594	0.03	10
	4	0.3168	0.035	10
L5–L6	0	0.4537	0.09	10
	1	0.4181	0.06	10
	2	0.3952	0.045	10
	3	0.3309	0.03	10
	4	0.3089	0.045	10

Recovery: $NIEMG(t) = E(1 - e^{-t/T3}) + tBe^{-t/T2} + C(t - Td)e^{-(t - Td)/T4} + NIEMG_{ss}$

Vertebral Level	E	T_3 (min)	B	T_2 (min)	C	T_4 (min)	T_d (min)	$NIEMG_{ss}$	r^2
L3–L4	0.6740	250	0.03	9	0.0002	400	460	0.3260	0.9568
L4–L5	0.6982	250	0.03	10	0.0001	400	460	0.3018	0.9503
L5–L6	0.7138	200	0.05	8	0.0001	450	410	0.2862	0.9489

B. Displacement Model Parameters (6 × 10-10, 20 N)

Work Period: $Disp(t) = \Sigma\ (D_{0n} + D_{Ln}(1 - e^{-t/Tn5}))$

n	D_{0n} (mm)	D_{Ln} (mm)	T_{n5} (min)	r^2
0	3.5008	3.4334	3.5	0.9478
1	5.4976	2.4375	2.9	0.9400
2	6.4902	2.0887	1.7	0.9032
3	7.2341	1.8290	1.7	0.8686
4	7.7385	1.6806	1.7	0.8433
5	7.9524	1.7586	1.6	0.8875

Rest Period: $Disp(t) = \Sigma\ (D_{0n} + R_n + (D_{Ln} - R_n)e^{-t/Tn6})$

n	D_{0n} (mm)	D_{Ln} (mm)	R_n (mm)	T_{n6} (min)
0	3.5008	3.4334	1.9968	2
1	5.4976	2.4375	0.9926	2
2	6.4902	2.0887	0.7439	2
3	7.2341	1.8290	0.5044	2
4	7.7385	1.6806	0.2139	2

(Continued)

TABLE 1
(Continued)
Recovery: $Disp(t) = D_O + R + (D_L\_R)e^{-t/T6}$

D_0 (mm)	D_L (mm)	R (mm)	T_6 (min)	r^2
3.5008	6.2136	2.3165	70	0.9057

NIEMG indicates normalized integrated electromyography (EMG).

ery of creep at the end of 7 hours of rest.

Mean NIEMG at 20-N load displayed decreasing values to 0.526, 0.511, and 0.454 after the first 10-minute cyclic work session in the L3–L4, L4–L5, and L5–L6 multifidus muscles, respectively. The first 10 minutes of rest displayed recovery to 0.829, 0.814, and 0.784 for the respective levels. The succession of work/rest sessions resulted in a further decrease in NIEMG to the final values of 0.408, 0.399, and 0.425, respectively. An increase in NIEMG was observed after the last work/rest period followed by a slight decrease. Final values after the 7-hour recovery period were 0.861, 0.843, and 0.897 for the respective multifidus positions. Full recovery was not seen in any of the preparations.

The results for the 40-N load showed that after the first 10-minute cyclic loading session, the mean NIEMG values decreased to 0.815, 0.743, and 0.779 in the L3–L4, L4–L5, and L5–L6 multifidus muscles, respectively. Partial recovery was seen after the first resting session to 0.939, 0.915, and 0.907 at the respective levels. The succession of work/rest sessions resulted in a further decrease in NIEMG to the final values of 0.695, 0.677, and 0.627. An increase in NIEMG was observed after the last work/rest period followed by a slight decrease. A gradual increase followed throughout the recovery period with resulting mean NIEMG values of 1.222, 1.268, and 1.149 at the end of the 7-hour rest session. Full recovery was seen in four of the seven preparations (unity).

For the 60-N load, a decrease in mean NIEMG values to 0.876, 0.853, and 0.842 after the first 10-minute cyclic loading session was seen. Recovery to 0.949, 1.017, and 1.060 was seen after the first 10-minute resting session. The succession of work/rest sessions resulted in a further decrease in mean NIEMG to the final values of 0.775, 0.520, and 0.655. After the cyclic work session, NIEMG values produced a peak in the first 10 minutes of the 7-hour recovery session, followed by a slight decrease and then an exponential increase to the end. Mean NIEMG values at the end of the 7-hour recovery period were 1.994, 2.091, and 1.914 for the respective multifidus positions. All specimens displayed NIEMG values of more than 1.0 after the 7-hour recovery period.

The best-fit models were superimposed on the experimental data in Figs. 2A–C. For the 20-N loading, it can be observed that A_n, T_{n1}, and $NIEMG_{0n}$ generally decreased during each work session. A_n and T_{n1} only had a slight increase during the fifth work period. During the resting sessions, the displacement model values displayed that T_{n6} was constant, and δ_{Ln} and R_n decreased while δ_{0n} increased. NIEMG modeling displayed that T_{n2} was constant during the resting period while B_n and $NIEMG_{0n}$ gradually decreased, indicating slow recovery. Respective values can be seen in Table 1.

Superimposed models for the 40-N and 60-N loads were similar to the 20-N load magnitude when comparing the work/rest sessions. The model parameters for the work sessions had no distinct pattern in the values of T_{n1} and A_n in between work sessions. Values of T_{n1} were highest at the initiation of each work session, as seen in Tables 2 and 3.

Recovery models of mean NIEMG displayed C values with a 10-fold difference in each respective load. Values of B, E, and T_2 were found to be inversely correlated with load. Similarities were found for 40 N and 60 N in T_4 and T_d with low values ranging from 280 to 365 minutes, whereas at 20 N the range was significantly higher from 400 to 460 minutes.

In order to verify the accuracy of the best-fit models from which R^2 values were derived, with some exceptions of random spasms causing a disturbance in the EMG, we obtained R^2 values of >0.9 for the best-fit models to the data. Unfortunately, random spasms cannot be excluded or filtered from the data set, while their presence decreases the R^2 value to some extent.

Overall displacement data and statistical analysis (Table 4) displayed evidence of the residual effects of creep due to cyclic loading. The ANOVA was utilized to verify the association between time and load and NIEMG. Values for the F statistic for both the independent variables of load and time displayed values above the critical values (>3.06, >2.00 load and time, respectively). Therefore, the magnitude of the independent variables of time and load contributed to the prediction of the corresponding NIEMG/displacement values. Interaction statistics were found to have no correlating factors (<1.72). To further prove the existence of the association between the time/load and resulting NIEMG/displacement, P values for each correlation displayed values near zero.

Discussion

The major findings of this investigation consist of the fact that for the same work schedule, moderate (40

TABLE 2

A. Model Parameters (6 × 10-10, 40 N)

Work Period: $NIEMG(t) = \Sigma\ (A_n e^{-t/Tn1} + NIEMG_{ssn})$

Vertebral Level	n	A_n	T_{n1} (min)	$NIEMG_{ssn}$	r^2
L3–L4	0	0.181	2.5	0.819	0.623
	1	0.281	1.0	0.658	0.788
	2	0.141	2.0	0.608	0.690
	3	0.180	1.8	0.570	0.882
	4	0.133	3.5	0.534	0.905
	5	0.162	2.8	0.516	0.808
L4–L5	0	0.236	4.0	0.764	0.799
	1	0.351	1.5	0.564	0.934
	2	0.255	1.5	0.504	0.948
	3	0.261	1.5	0.449	0.955
	4	0.251	1.5	0.418	0.951
	5	0.284	1.6	0.384	0.950
L5–L6	0	0.219	2.0	0.781	0.410
	1	0.347	0.8	0.561	0.922
	2	0.286	0.8	0.493	0.942
	3	0.288	1.0	0.448	0.917
	4	0.270	1.0	0.430	0.976
	5	0.250	1.2	0.414	0.923

Rest Period

$NIEMG(t) = \Sigma\ (tB_n e^{-t/Tn2} + NIEMG_{ssn})$

Vertebral Level	n	$NIEMG_{ssn}$	B_n	T_{n2}
L3–L4	0	0.819	0.03	10
	1	0.658	0.03	10
	2	0.609	0.04	10
	3	0.570	0.03	10
	4	0.534	0.04	10
L4–L5	0	0.764	0.04	10
	1	0.564	0.06	10
	2	0.503	0.06	10
	3	0.449	0.06	10
	4	0.418	0.07	10
L5–L6	0	0.781	0.03	10
	1	0.561	0.06	10
	2	0.493	0.07	10
	3	0.448	0.07	10
	4	0.430	0.06	10

Recovery: $NIEMG(t) = E(1 - e^{-t/T3}) + tBe^{-t/T2} + C(t - Td)e^{-(t - Td)/T4} + NIEMG_{ss}$

Vertebral Level	E	T_3 (min)	B	T_2 (min)	C	T_4 (min)	T_d (min)	$NIEMG_{ss}$	r^2
L3–L4	0.440	220	0.07	6	0.003	340	275	0.560	0.891
L4–L5	0.586	120	0.087	8	0.003	330	280	0.415	0.958
L5–L6	0.562	180	0.08	7	0.002	320	280	0.438	0.942

B. Displacement Model Parameters (6 × 10-10, 40 N)

Work Period: $Disp(t) = \Sigma\ (D_{0n} + D_{Ln}(1 - e^{-t/Tn5}))$

n	D_{0n} (mm)	D_{Ln} (mm)	T_{n5} (min)	r^2
0	7.283	4.539	3.0	0.947
1	9.756	3.752	2.0	0.840
2	11.167	3.087	1.8	0.808
3	11.841	3.120	1.5	0.829
4	12.337	3.081	1.3	0.824
5	12.904	2.824	1.0	0.858

Rest Period: $Disp(t) = \Sigma\ (D_{0n} + R_n + (D_{Ln} - R_n)e^{-t/Tn6})$

n	D_{0n} (mm)	D_{Ln} (mm)	R_n (mm)	T_{n6} (min)
0	7.283	4.636	2.473	2
1	9.756	3.752	1.411	2
2	11.167	3.087	0.674	2
3	11.841	3.120	0.495	2
4	12.337	3.081	0.567	2

(Continued)

TABLE 2
(Continued)
Recovery: $Disp(t) = D_o + R + (D_L\_R)e^{-t/T6}$

D_0 (mm)	D_L (mm)	R (mm)	T_6 (min)	r^2
7.283	8.445	2.311	50	0.867

NIEMG indicates normalized integrated electromyography (EMG).

N) and high (60 N) loads applied to the lumbar spine elicit a delayed muscular hyperexcitability, whereas mild (20 N) loads did not. Delayed hyperexcitability was shown to be associated with the development of an acute inflammation in the supraspinous ligament.[10] The moderate and high loads, therefore, elicited sufficient microdamage in the collagen fibers of the viscoelastic tissues to trigger an acute inflammatory response that, if exposed to further work over time, may result in a chronic inflammation (or cumulative trauma disorder) and the long-term disability associated with it.

In a previous report assessing the effect of load magnitude for a similar work/rest schedule but for static loads, it was found that only the 60-N load elicited a mild delayed hyperexcitability.[6] It seems that cyclic loading is more challenging for the viscoelastic tissues than a static load. In comparison with the two loading modes, several factors delineate the differences. In static loads, the ligaments (for example) are subjected to a single stretch and then remain loaded throughout the work period. Cyclic loading, however, presents many repetitions of tissue stretching within the same working period but only a very brief period of being subjected to the peak load (during the peak of the sinusoid). The stretching phase as well as the long duration of being subjected to the load could be challenging events for the tissue. In consideration of the two factors, it is evident that the multiple stretch phases resulted in a more pronounced hyperexcitability and at lower loads, despite the fact that the full load was sustained only for a very brief moment. One can, therefore, conclude that exposure to the stretching action and its repetition within a given period is more damaging to the viscoelastic tissues as they decrease its ability to sustain high loads without developing an acute inflammation. In fact, another report utilizing a similar protocol but increasing the number of static repetitions from six sessions to nine loading sessions of 10-minute loads elicited an inflammatory response.[7] Repetition of the stretching action of the viscoelastic tissues emerges as a primary risk factor for creating microdamage in the collagen fibers. Many repetitions result in a decrease in the load magnitude that the tissue can sustain without triggering an acute response.

The creep developed in the lumbar spine at the end of the six loading sessions was 139%, 83%, and 66% for 20 N, 40 N, and 60 N, respectively. Logically, we could assume that heavier loads should result in larger final creep at the end of the six work periods, yet decreasing creep with load was present. Two factors were responsible for the observation. Larger loads resulted in larger reflexive EMG from the multifidi.[19,21] Larger peak-to-peak EMG as well as its frequency content as represented by the IEMG indicates stronger muscular contraction and its associated force. Larger forces seen in the higher loads increased the stiffness of the lumbar spine and decreased its displacement and associated observed creep. The true final creep in the viscoelastic tissues was, therefore, masked by the increased stiffness of the lumbar spine. The stronger muscular contractions associated with higher loads explains the lower creep values observed in high loads.

The second factor that masked the creep are the increased magnitudes of the multifidus spasms observed in the preparations subjected to higher loads. The increased magnitude and frequency of the spasms in the high-load groups constituted further increase in lumbar stiffness and masking of the creep developing in the viscoelastic tissues.

Overall, the decreasing creep with increasing load after the six loading periods is explained well and justified. This phenomenon, however, points to an important neurological feature: the ligamento-muscular reflex provides the lumbar spine with a mechanism in which increased musculature activity compensates for the laxity developing in the lumbar viscoelastic tissues. This compensatory mechanism serves well to preserve lumbar stability and simultaneously protect the soft tissues from further exposure to excessive damage.

At the end of the 7 hours of rest that followed the six loading periods, the creep recovered to 108%, 55%, and 44% for the 20-N, 40-N, and 60-N loads, respectively. Two important features are noticeable. The first consist of the fact that 7 hours of rest are not sufficient to allow full recovery of the creep developed in 60 minutes of cumulative work, regardless of the load magnitude. The second feature consists of the lower residual creep associated with increasing load magnitude.

The absence of complete recovery of the creep developed in the viscoelastic tissues is not as surprising. Previous reports investigating this issue demonstrated that a rest period equal or longer to the loading period yields only a partial recovery.[22–24] In fact, the majority of the creep indeed

TABLE 3

A. Model Parameters (6 × 10-10, 60 N)

Work Period: $NIEMG(t) = \Sigma\ (A_n e^{-t/Tn1} + NIEMG_{ssn})$

Vertebral Level	n	A_n	T_{n1} (min)	$NIEMG_{ssn}$	r^2
L3–L4	0	0.0783	10	0.8761	0.1706
	1	0.1936	0.43	0.7555	0.6799
	2	0.0756	0.01	0.7347	0.3324
	3	0.1284	0.25	0.7454	0.8187
	4	0.1050	0.52	0.7209	0.9104
	5	0.1446	0.22	0.6649	0.9429
L4–L5	0	0.1468	1	0.8532	−0.7120
	1	0.4339	0.37	0.5834	0.8154
	2	0.2619	0.16	0.5033	0.8570
	3	0.2640	0.35	0.4817	0.9003
	4	0.2119	0.32	0.4596	0.9496
	5	0.1899	0.42	0.4030	0.9076
L5–L6	0	0.1477	4	0.8424	−0.4754
	1	0.4537	0.51	0.6067	0.7936
	2	0.2609	0.14	0.6002	0.8523
	3	0.2647	0.25	0.5920	0.9507
	4	0.1884	0.31	0.5648	0.1766
	5	0.1491	0.25	0.5500	0.9565

Rest Period: $NIEMG(t) = \Sigma\ (tB_n e^{-t/Tn2} + NIEMG_{ssn})$

Vertebral Level	n	$NIEMG_{ssn}$	B_n	T_{n2}
L3–L4	0	0.8761	0.025	10
	1	0.7555	0.02	10
	2	0.7347	0.035	10
	3	0.7454	0.02	10
	4	0.7209	0.02	10
L4–L5	0	0.8532	0.045	10
	1	0.5834	0.05	10
	2	0.5033	0.065	10
	3	0.4817	0.055	10
	4	0.4596	0.035	10
L5–L6	0	0.8424	0.06	10
	1	0.6067	0.07	10
	2	0.6002	0.07	10
	3	0.5920	0.045	10
	4	0.5648	0.035	10

Recovery: $NIEMG(t) = E(1 - e^{-t/T3}) + tBe^{-t/T2} + C(t - Td)e^{-(t - Td)/T4} + NIEMG_{ss}$

Vertebral Level	E	T_3 (min)	B	T_2 (min)	C	T_4 (min)	T_d (min)	$NIEMG_{ss}$	r^2
L3–L4	0.3351	200	0.07	5	0.009	300	335	0.6649	0.9674
L4–L5	0.5970	200	0.09	5	0.010	325	365	0.4030	0.9781
L5–L6	0.4500	160	0.095	5	0.009	285	332	0.5500	0.9841

B. Displacement Model Parameters (6 × 10–10, 60 N)

Work Period: $Disp(t) = \Sigma\ (D_{0n} + D_{Ln}(1 - e^{-t/Tn5}))$

n	D_{0n} (mm)	D_{Ln} (mm)	T_{n5} (min)	r^2
0	12.0386	4.7393	3.5	0.9692
1	15.2985	3.1366	2.2	0.8788
2	16.7792	2.7748	2.7	0.8970
3	18.0457	2.4740	2.2	0.8987
4	18.8702	2.2908	2.1	0.8960
5	19.5251	2.1398	1.6	0.8921

Rest Period: $Disp(t) = \Sigma\ (D_{0n} + R_n + (D_{Ln} - R_n)e^{-t/Tn6})$

n	D_{0n} (mm)	D_{Ln} (mm)	R_n (mm)	T_{n6} (min)
0	12.0386	4.7393	3.2599	2
1	15.2985	3.1366	1.4807	2
2	16.7792	2.7748	1.2665	2
3	18.0457	2.4740	0.8245	2
4	18.8702	2.2908	0.6549	2

(Continued)

TABLE 3
(Continued)
Recovery: $Disp(t) = D_o + R + (D_L\_R)e^{-t/T6}$

D_0 (mm)	D_L (mm)	R (mm)	T_6 (min)	r^2
12.0386	9.6304	3.5365	140	0.8618

NIEMG indicates normalized integrated electromyography (EMG).

TABLE 4
Two-Way ANOVA

Parameter	Effect of Time (df = 8)		Effect of Load (df = 2)		Interaction (df = 16)	
	F	*P*	*F*	*P*	*F*	*P*
EMG L3–L4	10.85	<0.0001	32.53	<0.0001	1.18	0.30
EMG L4–L5	10.20	<0.0001	15.67	<0.0001	1.54	0.09
EMG L5–L6	4.77	<0.0001	5.31	<0.0001	1.22	0.26
Displacement	7.94	<0.0001	455.15	<0.0001	0.20	1.00

ANOVA indicates analysis of variance.

recovers in the first hour of rest, yet the last 10% to 20% of the creep takes up to 24 hours to recover.[24] Our model confirms that observation. This issue puts importance and emphasis on the value of appropriately chosen durations of rest for workers and athletes exposed to intense periods of physical activities. The objective of such rest periods are not only to overcome muscle fatigue but to primarily restore the resting length and operating properties of ligaments, tendons, discs, and capsules.

The lower residual creep associated with increasing load magnitudes leads back to muscular activity. High-load magnitudes exhibited delayed hyper excitability during the last few hours of the 7-hour recovery. The delayed hyperexcitability was of larger magnitude for the larger load (see Fig. 3). Therefore, the larger muscular activity was expressed by higher stiffness of the lumbar spine, masking further the true creep in the viscoelastic tissues.

From the discussion above, it can be a reasonable conclusion that the measured creep does not truly represent the microdamage and the associated laxity in the ligaments, discs, facet capsule, etc, since it is masked by muscular activity. The true elongation and laxity of the soft tissue could not be measured directly, since the increased muscular activity shortened the distance between the adjacent vertebrae in an attempt to prevent the soft tissue exposure to further stretch. In essence, the muscles took over the function of providing tension between the vertebrae after the ligaments sustain a certain level of damage. This leads to the assumption that the direct relationship between the creep and laxity to the neuromuscular disorder developed by higher loads cannot be established by the protocol employed and requires different approaches.

The statistical analysis confirms that the NIEMG curves for the three loads during the 7-hour recovery are functions of time ($P < 0.0001$) and different from each other ($P < 0.001$). This indicates that the recovery curve for the 20-N load is different from the 40-N and 60-N curves, as well as differences between the 40-N and 60-N curves. Essentially, the statistics confirm that the 60-N load elicited a delayed hyperexcitability, which is significantly larger in magnitude than that of the curve for the 40-N load.

The larger hyperexcitability observed in the NIEMG recovery curve of the 60-N load points out that the inflammation developed in the viscoelastic tissues was more intense and developed at a faster rate after the loading ceased. Since the presence of inflammation requires the infiltration of neutrophils and cytokines from the circulatory system, it is a time-dependent process. The appearance of hyperexcitability could be seen 2 to 3 hours after the 7 hours of initiation of recovery, and it did not reach a peak by the seventh hour (see Fig. 3). Most likely, the concentration of inflammatory agents continued past the seventh hour, increasing the intensity of the disorder.

Further confirmation of the increased hyperexcitability with increasing load magnitude is presented by the model developed for the recovery period. The constant C of equation 2 represents the peak of the hyperexcitability if given sufficient time to mature. For the group subjected to the 20-N load, C was insignificant at 0.0001, and it increased to 0.003 and to 0.0095 for the 40-N and 60-N loads, respectively. The increases represent 30-fold and 95-fold increases in maximal amplitude of the delayed NIEMG component.

Additional validation of the delayed hyperexcitability is given by the time constants T_4 and T_d. For the 20-N load, T_4 was 400 minutes and T_d was 460 minutes, indicating a need for a very long time if and when

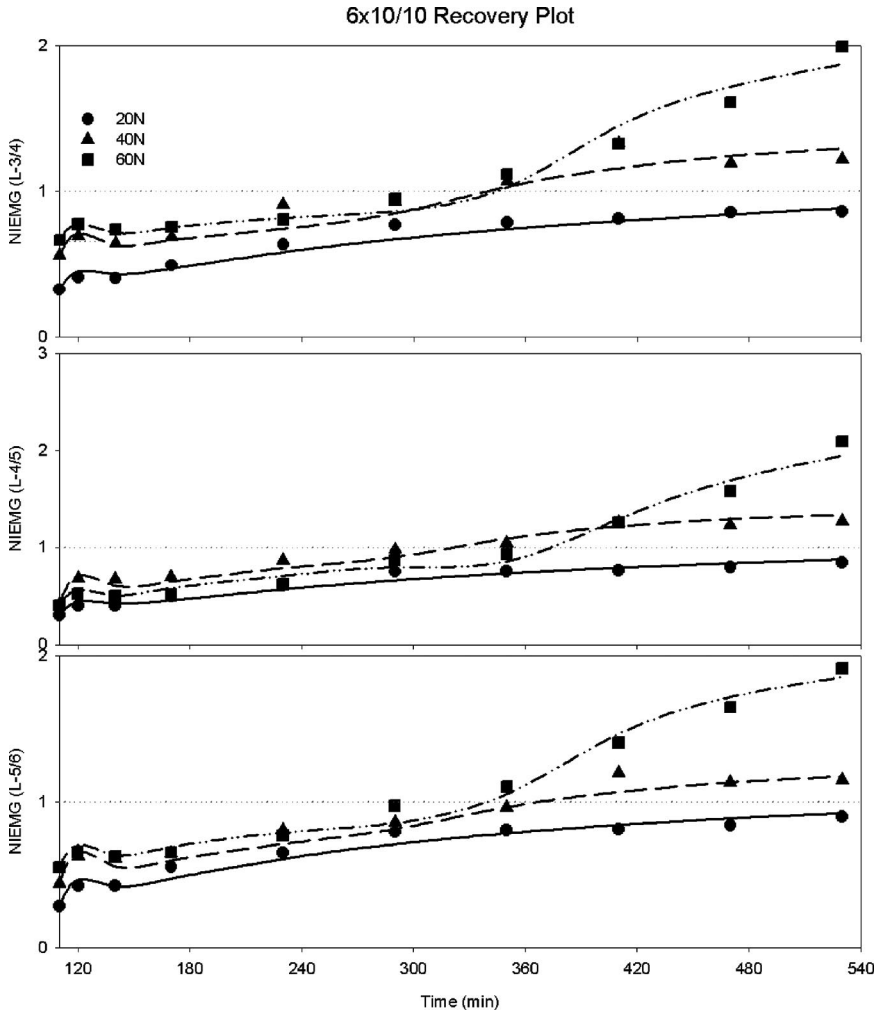

Fig. 3. Superimposed nonlinear models for the mean NIEMG 7-hour recovery components are shown for 20-N, 40-N, and 60-N loads, respectively.

the delayed hyperexcitability develops. Conversely, the T_4 and T_d values for the group subjected to 40 N were 330 minutes and 280 minutes, respectively. For the group subjected to the 60-N load, the time constants T_4 and T_d were 325 minutes and 365 minutes, respectively. A significant acceleration of the delayed development of hyperexcitability is evident when the load increased from 20 N to 40 N, whereas only minor changes in timing occurred when the load increased from 40 N to 60 N. If both amplitude C and time constants (T_4 and T_d) are considered together, one can conclude that once sufficiently high loads are applied to trigger an inflammation and a neuromuscular disorder (eg, delayed hyperexcitability), the model predicts the increase in the intensity and duration of the disorder. Additional increases in load yield only significant increase in the intensity of the hyperexcitability but not in the duration necessary for it to develop. This may translate to larger amounts of neutrophils and cytokines infiltrating into the affected tissue over the same or longer period, resulting in a more intense acute inflammation.

Spasms were observed during the loading period as well as during the short loading tests of the 7-hour recovery period. Clinically, spasms indicate the occurrence of tissue damage.[25] Indeed, Woo et al[2] observed microdamage in the collagen fibers of ligaments subjected to repeated stretch within the physiological limits. The group subjected to the large load exhibited more intense and frequent spasms, indicating that larger microdamage is present. Unfortunately, the unpredictable occurrence of the spasms in timing and intensity could not be quantified on the background of the expected reflexive EMG.[19,21,26] This scenario, in turn, prevented the formation of any relationships between the spasms, the creep developed, and the resulting disorder. Nevertheless, the spasms indicate that microdamage occurred in the low- and high-magnitude load groups. It seems that the level of microdamage must exceed a certain threshold in order to trigger an inflammatory response as seen in the high-load magnitude groups.

Finally, the results of this investigation were derived from data collected from the feline model. The validity of such data depends on demonstration that such processes indeed occur in humans. A series of projects addressed this issue by demonstrating spasms, functional neuromuscular changes and development of laxity in the knee and lumbar spine of normal humans subjected to cyclic and static loading.[13–18] The similarity of the responses in normal humans provides confidence in the validity of the results presented here. A recently available report also confirms the biomechanical similarity of the lumbar spine between bipeds and quadrupeds.[27] Differences in size are evident between humans and cats, and that will impact the absolute load magnitudes, number of repetitions, work and rest durations, etc, as well as the time constants in our model equation. The anatomy, physiology, and neurological responses of the human and feline are similar and so are the collagen structures of the ligaments, disc, facet capsule, and dorsolumbar fascia. Overall, it is reasonable to accept that with scaling modifications, the conceptual findings of this study are relevant to humans.

Conclusions

The data emerging from this investigation allow the following conclusions:

1. High magnitudes of cyclic loads applied to the lumbar spine can trigger a delayed hyperexcitability, a neuromuscular disorder indicative of an acute inflammation.
2. The intensity of the neuromuscular disorder increases with the magnitude of the load applied to the lumbar spine.
3. Cyclic loading seems to be more challenging to the viscoelastic tissues than static loading, as cyclic loads trigger a neuromuscular disorder at lower load magnitudes than that in static loading.

An experimental validation of the epidemiological data is provided for the classification of high-load magnitudes in repetitive occupational activities as a risk factor for the development of a cumulative trauma disorder. High and moderate cyclic loads lifted over a period of time were shown to induce an acute neuromuscular disorder that can further evolve over weeks or months of exposure into a CTD. Workers engaged in such activities should be subjected to limited time of exposure, work rotation, increased rest periods, and/or similar procedures in order to prevent the development of CTD.

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