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

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Workers engaged in occupational activities requiring prolonged static and cyclic activities report up to ten times more musculoskeletal disorders than the general population.[40] Such occupational disorders, specifically low back disorders (LBDs), are a costly burden on society. Repetitive (cyclic) lumbar flexion (such as in loading and unloading boxes) causes the development of creep (e.g., elongation) of the various lumbar viscoelastic tissues (e.g., ligaments, disks, and joint capsules).[1,4,13,18,21] The ligaments, when creep has developed, are longer and lax, whereas the disks lose fluid and have a deformed collagenous shell.[1] The creep in these viscoelastic tissues may introduce laxity in the intervertebral joint and the possibility of excessive motion and injury. Yet, the overall stiffness of the intervertebral joint is dependent mostly on the forces developed by various lumbar muscles.[19,21,24] Normal and balanced muscle function is, therefore, paramount for the stability and safety of the lumbar spine.[35]

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

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

Abbreviations: CTD, cumulative trauma disorder; EMG, electromyogram; LBD, low back disorder; NIEMG, normalized integrated EMG
Key words: creep; cyclic; electromyography (EMG); disorder; ligaments; muscles; reflex; spine
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flexion under constant load as opposed to constant displacement.[8] The neurological responses to cyclic lumbar flexion under constant peak load are unknown. It is also not known whether different peak loads have an impact on the neurological response of the lumbar musculature.

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

METHODS

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

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

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

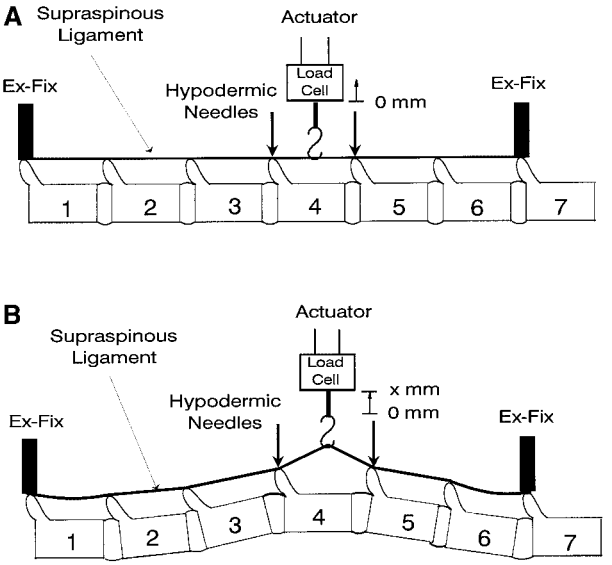

FIGURE 1. Schematic representation of the experimental arrangement showing the lumbar spine at rest **(A)**, and during peak flexion **(B)**.

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

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

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

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

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

The displacements of the respective window of all preparations subjected to the same peak load were also pooled and presented as mean displacement (±SD) vs. time. Analysis of variance with repeated measures was applied to the displacement data to determine whether changes in displacement over time and with respect to the three load levels (20 N, 40 N, and 60 N) were statistically significant.

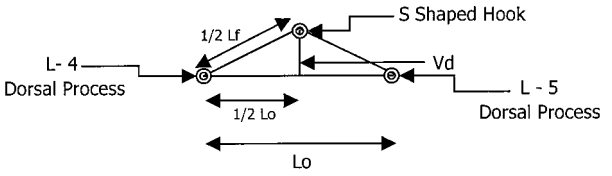

FIGURE 2. Schematic representation of the supraspinous ligament loaded with a 1-N test load before and after 20-min cyclic loading, and at the end of the recovery (rest) period, to determine the creep that developed in the ligament due to cyclic loading and the residual creep after recovery.

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

$$Lf = 2\sqrt{(\tfrac{1}{2}\, Lo^2) + Vd^2} \qquad (1)$$

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

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

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

For the NIEMG:

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

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

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

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

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

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

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

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

For the NIEMG, the model format was:

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

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

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

The second and third terms, therefore, are transient features that first increase and then reverse (decrease) over time to finally arrive to near zero as the effect of hyperexcitability diminishes with rest. Furthermore, the third term, which represents the delayed hyperexcitability, becomes effective only after $t \geq T_d$; that is, the effect of this term is null until recovery time exceeds T_d. Overall, the model provides a unique prediction of the NIEMG at any given time during a rest period following a cyclic loading period.

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

RESULTS

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

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

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

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

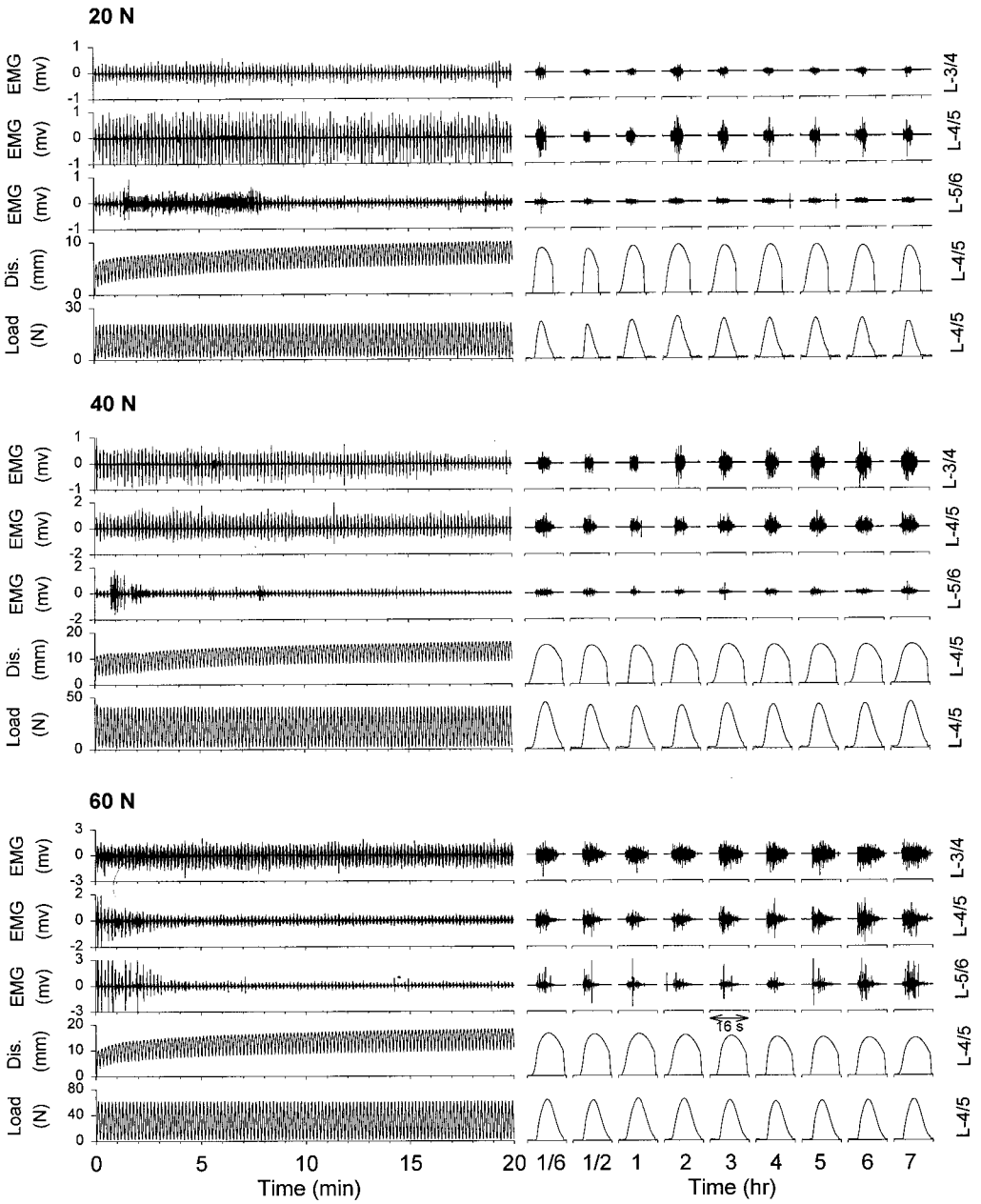

FIGURE 3. Typical EMG recordings from the L-3/4, L-4/5, and L-5/6 motion segments and the displacement and load associated with cyclic loading with 20-N (top), 40-N (middle), and 60-N (bottom) loads. The test cycles during the recovery period are shown on an expanded time scale after the 20-min marker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

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

The mean displacement associated with the first cycle with peak 40-N load was 11.99 mm followed by an exponential-like increase to the end of the 20 min, culminating with a peak mean displacement of 16.6 mm, a 38.4% increase due to creep development in the viscoelastic tissues. During the recovery period, the displacement decreased in an exponential fashion, reaching a mean value of 14.04 mm at the end of 7 h, which corresponding to 17.1% residual displacement (or 17.1% larger than the initial displacement of 11.99 mm). Full recovery of the displacement was not

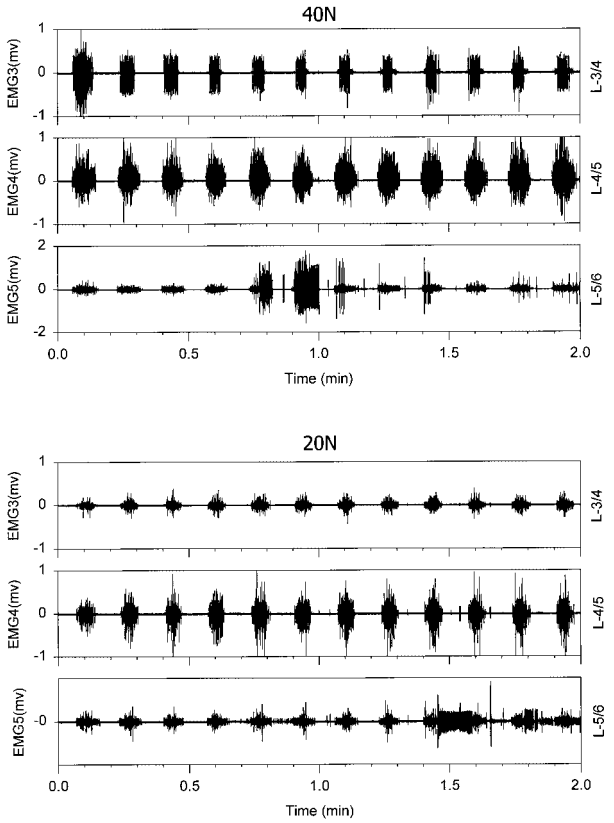

FIGURE 4. Expanded time scale of the first 2 min of a typical EMG recording from Figure 4 for the 20-N (bottom) and 40-N (top) loads showing spasms superimposed on the reflex EMG activity elicited by the lumbar flexion. In the bottom panel, spasms are evident as the outbursts after the 1.5-min marker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

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

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

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

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

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

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

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

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

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

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

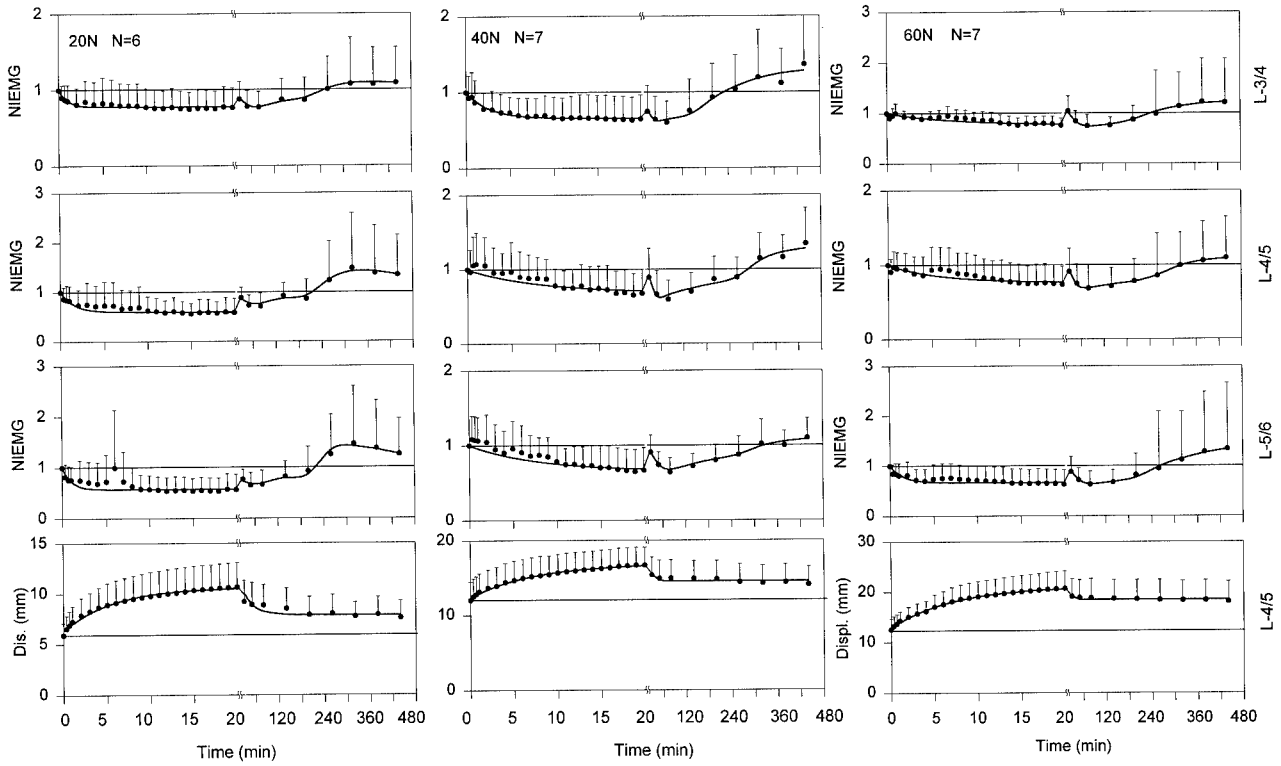

FIGURE 5. The mean and standard deviations of the pooled data from all preparations subjected to 20-N (left), 40-N (middle), and 60-N (right) loads. The models developed for the data are shown superimposed on the data.

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

Control Group. The EMG data recorded from the control group, which was not loaded at all, remained at the baseline level throughout the 20-min period when loading occurred in the experimental groups, plus a 7-h rest period. This confirmed that the spasms and EMG changes recorded from the experimental groups were directly related to the load applied and not to any other artifactual factor associated with the protocol.

Displacement Model

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

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

Table 1. Vertical displacement model during cyclic loading.

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

Table 2. Vertical displacement model during recovery.

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

Table 3. NIEMG models during cyclic loading.

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

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

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

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

For the recovery period, the proposed model is given in eq. (6), and the constants determined by the analysis are given in Table 4. A very good fit to the physiological data was obtained as the r^2 values ranged from 0.889 to 0.982. Table 4 confirms that the different peak loads applied to the respective experimental groups resulted in different responses. The time constant T_4, which governs the recovery rate of reflex EMG activity during rest, indicates that higher loads required longer rest time for the reflex EMG to recover to its initial level observed at the beginning of 20-min cyclic flexion.

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

DISCUSSION

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

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

Table 4. NIEMG model during recovery.

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

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

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

The model describing the decreasing EMG during the 20 min of load-control cycling flexion demonstrated that most phasic activity diminishes within the first 5–10 min, after which further decrease is minimal. It should be noted that the mean NIEMG values in Figure 5 were not influenced by the exponentially increasing creep alone, as the effect of the spasms was also present. This causes several data points to be placed outside the predicted model, and in turn to also artificially decrease the r^2 value. However, the model represents only one of the two components present in the data—that is, the phasic decrease in EMG due to the phasic transient response of the fast and slow mechanoreceptors. The spasm component is not represented in the model. It should also be noted that compounding the experimental data from all the preparations subjected to the same load attenuates the effect of the spasms to some degree, but does not eliminate it completely. Therefore, although the r^2 values resulting from the model fit are reasonable, ranging from 0.707 to 0.977 (except the L-5/6 level at 20 N, which yielded $r^2 = 0.364$), this value could have been drastically improved if it had been possible to isolate and remove the effect of the spasms.

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

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

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

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

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

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

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

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